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# Monitoring, Identification, and Intervention for Metabolic Disorders in Veterans with Psychotic Disorders

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# MONITORING, IDENTIFICATION, AND INTERVENTION FOR METABOLIC DISORDERS IN VETERANS WITH PSYCHOTIC DISORDERS

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**ABSTRACT**

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In light of growing evidence that certain antipsychotics may cause potentially life-threatening metabolic side-effects, the purpose of this study was to determine how regularly mental health clinicians (MHCs) currently monitor and manage metabolic abnormalities in overweight and obese patients with psychotic disorders. We hypothesized that MHCs monitor, identify and intervene for metabolic abnormalities in their patients at significantly lower rates than primary care physicians (PCCs), and that such rates may jeopardize patient health.

We performed a one-year cross-sectional medical record review of primary care and mental health routine outpatient visit notes from the West Haven Campus of the Veteran Affairs Connecticut Healthcare System. We reviewed the records of a cohort of 123 veterans who met the following inclusion criteria: (1) primary diagnosis of schizophrenia or schizoaffective disorder; (2) at least one routine mental health visit at the West Haven VA facility between July 1, 2005 and June 30, 2006; and (3) overweight or obese, as determined by a body mass index (BMI) $\geq$ 25. We excluded all deaths.

The 123 subjects were predominantly white and male (56% and 93%, respectively) with an average body mass index (BMI) of 32.4 (SD=5.4). 97% of subjects were taking an antipsychotic of some sort, and 85% were taking a second-generation antipsychotic.

Zero diagnoses of metabolic syndrome and zero waist-size measurements were documented by PCCs or MHCs. The following differences in documentation were found between PCCs and MHCs, respectively: weight (85% vs. 11%; $p<0.001$); BMI (48% vs. 0%; $p<0.001$); identified weight as an issue (45% vs. 28%; $p<0.005$); identified the link between antipsychotics and weight issues (10% vs. 12%; not significant); made diet and exercise recommendations (42% vs. 19%; $p<0.001$); ordered a weight-management referral (21% vs. 3%; $p<0.001$); ordered or considered ordering a change of antipsychotic medication or dose due to weight-related issues (6% vs. 3%; not significant). PCCs ordered laboratory tests at much higher rates than MHCs, including blood glucose, thyroid stimulating hormone, urinalysis, lipid panel, and hemoglobin A1C (differences were large and significant).

We concluded that MHCs monitor, identify and intervene for metabolic abnormalities in their patients at significantly lower rates than PCCs, and that such rates are unacceptably low. The problem is one of a systemic failure in quality control and may pose a danger to patients. We advocate a rapid organizational response and systemic changes at the local and national level to improve quality.

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# INTRODUCTION

## Historical Background: Metabolic Derangement, Psychotic Disorders, and Antipsychotics

Well before the advent of antipsychotics there was a string of clinicians who had associated psychotic disorders with metabolic abnormalities. In 1904 Kraeplin described an association between weight gain and the regression of dementia praecox symptoms (1, 2). In1926, Kasanin described an association between schizophrenia and elevated blood glucose (3). Kooy, writing in 1919, associated psychotic disorders with abnormal levels of blood glucose (4). In 1947 Kryspin-Exner observed patients gaining weight as soon as or before symptoms improved (2, 5).

Even in the antipsychotic era, some investigators have postulated that there are potentially drug-independent associations between schizophrenia and metabolic dysfunction (6). Furthermore, Brown et al. and others have shown that determining the causes of metabolic dysfunction in psychotic patients is complicated by the unhealthy lifestyle that is observed in many such patients, a finding that makes it more difficult to delineate drug-induced from disease or behavior-induced metabolic dysfunction in psychotic patients (7, 8).

With the introduction of chlorpromazine into clinical practice by Smith-Kline and French in 1952 and the subsequent explosion of antipsychotic discovery and use, antipsychotics emerged as a potential cause of metabolic abnormalities in psychotically disordered patients (9). In 1954 Dobkin et al. found that normal volunteers responded to

injected chlorpromazine with increased blood glucose levels (10). Then in 1960 Klett and Caffey associated weight gain with the clinical efficacy of phenothiazine derivatives (2, 11). Singh et al. found similar associations in a study published in 1970 (2, 12).

The first atypical antipsychotic, clozapine, became commercially available in Europe in 1971 (13). By the mid-1990's several atypical antipsychotics had been developed and were being prescribed by physicians (13). Atypical, or second-generation, antipsychotics were originally greeted with much fanfare because they caused fewer extrapyramidal side-effects than first-generation antipsychotics. Also, they were hailed as more effective at relieving the negative symptoms of schizophrenia. Clozapine, in particular, was considered more clinically effective than any of the first-generation antipsychotics, despite its dangerous side-effect profile. As the use of atypical antipsychotics grew rapidly, investigators began to notice patterns of side-effects related to metabolism. Studies suggested an association between the atypical anti-psyschotic medications and weight gain, metabolic syndrome, and even diabetes. All of this literature will be discussed extensively in subsequent sections of this thesis.

Recently, as investigators and physicians have become more aware of the metabolic side effects of antipsychotics, and as concern has grown about the negative health consequences of overweight and obesity, academics have begun to examine the health services question of how doctors and other healthcare providers monitor and manage the metabolic status of patients with psychotic disorders. In light of this context, it has become questionable whether the common *de facto* divide between psychiatry and the rest of medicine is truly in the best interest of patients. At present, it is understood in the medical profession that many psychiatrists in the outpatient setting do not regularly

conduct physical exams or laboratory tests, and that they do not communicate with any regularity with internists or surgeons.

**Epidemiology and Health Effects of Overweight and Obesity**

Based on the 1999-2000 National Health and Nutrition Examination Survey (NHANES), over 60% of the United States population is overweight and over 25% are obese (14). These conditions are associated with an array of co-morbidities, particular cardiovascular disease, hypertension and diabetes (14). Obesity-associated direct costs among US adults may exceed 5% of all US healthcare expenditures (15). In the veteran population the figures are worse than in the general population: over two in three are overweight or obese, and more than one in three is classified as obese (14). A study by Mokdad et al. found that weight-related factors such as poor diet and physical inactivity followed tobacco as the leading actual causes of death in the United States in the year 2000, and that they may soon become the greatest actual causes of death (16). The Institute of Medicine found that weight loss as low as 5% of total body weight was associated a meaningful reduction of morbidity and mortality (17, 18). One would then suspect that a similar gain in weight might be associated with the converse (17). Guidelines for categorizing weight were set forth by a study performed by the National Institutes of Health (NIH) in 1998 (19, 20). It was found that health risks increase as patients moved from normal weight through the higher weight categories (17, 21).

Although considerable evidence has pointed to the adverse health effects of overweight and obesity, a provocative recent study by Flegal et al. in 2007 suggested that

relative to a normoweight population, overweight was not associated with cause-specific death from cancer or cardiovascular disease (22). Additionally, overweight was actually associated with a decrease in non-cancer, non-cardiovascular deaths (22). Obesity was associated with increased cause-specific mortality from cardiovascular disease and certain obesity-related cancers but it was not associated with mortality from non-cancer, non-cardiovascular causes (22). When combined, overweight and obesity were associated with increased cause-specific deaths from diabetes and kidney disease (22). This study raises the question of what the real implications for overweight are relative to normoweight, given that prior to this study one might have assumed that overweight was a broadly negative condition. The results even suggest that for certain categories of disease overweight may be protective. Flegal's study does appear, however, to lend further support to the claim that obesity leads to adverse health outcomes, particularly adverse cardiovascular outcomes. There also appears to be a concerning association between overweight and obesity, as a combined category, and diabetes and kidney disease. Even though overweight appears to be more benign than obesity, one must be concerned that the development of overweight in any given patient may ultimately lead to obesity.

Unfortunately, reversing overweight and obesity is difficult, though surgical, behavioral and pharmacological approaches to treatment do exist. Due to the risks posed by obesity, in particular, and the difficult of treatment, factors that contribute to the development of the condition must be mitigated by physicians whenever possible, particularly when such factors are related to medical treatment itself.

**Antipsychotics and Weight**

Due to the co-morbidities associated with overweight and obesity, it is concerning that there is strong evidence showing that certain antipsychotic medications cause weight gain – evidence that we will return to in detail at a later point. The high prevalence of antipsychotic treatment in populations of patients with psychotic disorders makes this group of patients particularly at risk for developing overweight and obesity. The psychotic population is also at risk simply because it is part of the general US population, where overweight and obesity are increasing at alarming rates (14). In addition, patients with psychotic disorders often have impairments in self-care that place them at risk for unhealthy lifestyles and hence weight gain (7, 8). Indeed, patients with serious mental illness have been shown to be at increased risk of developing obesity (6, 23-25), and obesity has been associated with excess deaths from cardiovascular disease (22). Also, schizophrenia is associated with significantly elevated rates of cardiovascular disease relative to the general population (26, 27), and this risk could be magnified by co-morbid metabolic dysfunction.

Treatment-associated weight gain concerning not only because of it may increase medical morbidity and mortality, but also because it may be a factor in reducing psychiatric medication compliance (17, 28-30)**,** although this association has been disputed by some studies (31). Weight gain may have a negative impact on the self-image and social status of psychotic patients (30). Indeed, weight gain in patients with schizophrenia has been shown to have a negative impact on quality of life (17, 30).

First-generation antipsychotics have been linked to weight gain, although in general the literature shows less concern about these drugs relative to the second-generation antipsychotics. A 1999 meta-analysis by Allison et al. used a random effects model to understand the impact of various antipsychotics on weight (17). The investigators found that haloperidol, chlorpromazine and thioridazine/mezoridazine were all associated with statistically significant weight gain (17). Fluphenazine, molindone and placebo were not associated with weight gain, although non-pharmacologic control was associated with a weight gain similar to haloperidol (17). The mean estimated statistically significant weight gain for first-generation antipsychotics at 10 weeks ranged from approximately 1.08 kg for haloperidol to 3.19 kg for thioridazine/mesoridazine (17). These results are of course limited by the quality of studies as well as issues such as dosing. Interestingly, the weight effect of the first-generation antipsychotics does not appear related to chemical structure or potency (2). Some studies have suggested that molindone and diphenylbutylpiperidine pimozide may actually induce weight loss of several kilograms (17, 32-34). Interestingly, early studies suggested that successful treatment with chlorpromazine was associated with weight gain, whereas weight loss was associated with worsening symptoms (35). This finding lines up with the early observations by Kraeplin about weight-associated symptom improvement and deterioration in schizophrenics (1).

Second-generation antipsychotics have been closely associated with weight gain. Clozapine is one of the best studied atypicals, and there is a well documented association between its use and weight gain. Allison et al.'s meta-analysis from 1999 showed a statistically significant mean gain of 4.45 kg associated with clozapine treatment at 10

weeks (17). Published in 2000, Henderson et al.'s five-year prospective naturalistic study of 82 patients taking clozapine found a mean weight gain of 11.6 kg over four years, and weight gain did not level off until month 46 (36). Olanzapine has been very clearly linked to weight gain as well (17). Risperidone appears to cause less weight gain than olanzapine, but the gains are still significant (17). Although quetiapine is a newer drug, there is some evidence that it also causes significant weight gain (17, 37). Ziprasidone appears to be the one atypical antipsychotic with limited impact on weight (17). Allison's meta-analysis showed no significant association between ziprasidone and weight gain (17). This offers clinicians a pharmacologic alternative to be considered if their patients gain significant weight on a non-ziprasidone atypical. It should be reiterated that according to the same meta-analysis, placebo was associated with weight loss. Although this relationship did not reach statistical significance, it does suggest that the weight gain associated with atypical antipsychotics is in fact drug-related.

**Antipsychotics and Diabetes**

Diabetes is a disease characterized by abnormally elevated blood glucose levels. The disease is an emerging epidemic in the United States (38). From 2000-2007 the incidence of diabetes in the United States increased by 54% (39). According to the Center for Disease Control, in 2004 15.2 million Americans had diabetes (www.cdc.gov). Particularly worrisome is the growing prevalence and incidence of type 2 diabetes, a disease of insulin-resistance, which is often associated with obesity and is contributing to much of the overall growth in the diabetes disease burden. Some have estimated that 90%

of type 2 diabetes can be attributed to excess weight (40). Diabetes affects almost every organ system and is associated with numerous serious health risks, including coronary artery disease, stroke, peripheral neuropathy, peripheral vascular disease, retinopathy, kidney disease, and gastroparesis. Furthermore, diabetics are at further risk of complications when in a hospital setting or after surgery (41-43).

Investigators have noted an association between schizophrenia and diabetes. This association was shown to exist before the widespread use of atypical antipsychotics (44, 45). Nevertheless, in recent years antipsychotics – particularly atypical antipsychotics – have been blamed for the rising incidence of diabetes in psychotic patients (46, 47). In a recent study by Srihari et al. that examined the prevalence and management of type 2 diabetes in patients receiving antipsychotic medications, diabetes was two-and-a-half times as prevalent in the study population than in the general population (48). The authors found that 71% of the 494 patients were taking one or more atypical antipsychotics (48). In a naturalistic study of clozapine-naïve patients, it was found that over a five-year period of treatment with clozapine, 36.6% of patients developed diabetes (36). Interestingly, no significant risk of diabetes was attributed to weight gain, use of valproate or clozapine dosing (36). In a large epidemiologic study of 56,849 schizophrenic patients taking antipsychotics over the span of 1-2 years, Leslie and Roesnheck suggested that the attributable risks of diabetes associated with atypical antipsychotics was small – ranging from 0.05% for risperidone to 2.03% for clozapine (49). Interestingly, the attributable risk for quetiapine and risperidone was not significantly different from that for conventional antipsychotics (49). Clozapine and olanzapine had the highest risk (49). Yet in a cross-sectional study of 38,632 schizphrenic

patients on antipsychotics it was found that patients taking atypical antipsychotics were 9% more likely to have diabetes than those who received typical antipsychotics (50). The association of atypical antipsychotics and increased prevalence of diabetes was even stronger in patients younger than 40 (50).

A paper by Dixon et al. suggested that patients who had schizophrenia and diabetes had better outcomes, as measured by HbA1c, than patients with no severe mental illness (51). This suggests although psychotic disorders and the drugs used to treat them predispose patients to developing diabetes, such patients are not necessarily unable to manage their diabetes. If that is the case beyond this study sample, it raises the importance of monitoring for the development of diabetes and then intervening – such interventions may be as effective or more effective in schizophrenic patients as in non-schizophrenic patients. Notably, patients taking olanzapine had higher HbA1c levels than patients taking other antipsychotics (51).

Gianfrancesco et al. examined the odds of developing type 2 diabetes in diabetes-naïve patients who were treated with various antipsychotics using data from 2.5 million patients cared for by managed care and insurance companies(52). The study period was 12 months, and patients reporting diabetes up to 8 months prior to the study were excluded (52). Olanzapine, clozapine and certain first generation antipsychotics increased the risk of developing diabetes significantly, whereas risperidone did not increase risk relative to untreated patients (52). The finding about risperidone aligns with Sernyak's study (50). In support of Gianfrancesco et al.'s findings, in 2004 Citrome et al. found that exposure to multiple second-generation antipsychotics or clozapine or quetiapine significantly increased the risk of treatment-emergent diabetes mellitus (53).

Regarding treatment, Klein et al. published an interesting article suggesting that metformin is an effective intervention for weight gain, decreased insulin sensitivity, and abnormal glucose metabolism related to atypical antipsychotics (54). The limitation of this study is that it was conducted in children and adolescents; nevertheless, it may be translatable to adult patients and should be studied in that population. In fact, a recent study from China, conducted by Wu et al., investigated the value of interventions in 128 schizophrenic patients using atypical antipsychotics. They found that after a 12-week period, patients who received dietary education and partially supervised exercise, metformin, or both, had significant weight loss and reduced insulin resistance relative to drug placebo (55).

**Antipsychotics and Blood Glucose Levels**

A growing body of literature has suggested that antipsychotic use may lead to changes in blood glucose levels (without necessarily leading to diabetic levels) (56-60). Lindenmayer et al. published a prospective randomized double blind trial in 2003 with 157 patients with schizoaffective or schizophrenic disorder to look at the association between antipsychotics and blood glucose level (61). The study was conducted over 14 weeks (61). Baseline blood glucose levels were taken before the patients were started on therapy, and included a 6-week fixed dose period an 8-week variable dose period (61). They found that clozapine, olanzapine, and haloperidol were associated with elevated levels of fasting plasma glucose levels (61). Risperidone was not associated with significant changes in blood glucose (61). Most of the elevations of plasma glucose did

not lead to levels associated with the diagnosis of diabetes, although approximately 14% of patients did develop diabetic-levels of blood glucose during the course of the study (61). However, given Wilson et al.'s finding (below) that glucose intolerance may more labile in patients receiving atypical antipsychotics, one wonders whether the point fasting blood glucose levels used in this study could be misleading (62).

A particularly concerning result was noted in a 2002 study by Wilson et al., which examined data from 126 patients treated with atypical antipsychotics. The investigators found 11 cases of new-onset, acute, and severe glucose intolerance after treatment with clozapine, olanzapine or quetiapine (62). Most worrisome, of these 11 patients, 5 developed diabetic ketoacidosis (62). Interestingly, glucose metabolism was labile in all of the cases, and 2 of the patients had resolution of glucose intolerance despite continued treatment with antipsychotics. The authors pointed out that labile glucose intolerance is typically suggestive of type 1 diabetogenesis (62). They also made the interesting point that many of the symptoms of diabetes overlap with those of antipsychotic medications, such as dry mouth, blurry vision, hyperphagia and polyuria, thereby blunting the typical triggers for patient alarm (62). Therefore, physicians should always consider diabetes in their differential diagnosis of these common side effects of antipsychotics.

In 2002 Newcomer et al. studied blood glucose levels in 48 schizophrenic patients on various antipsychotics relative to 31 healthy controls matched for adiposity and age (2, 58). First generation antipsychotics were associated with very small increases in blood glucose levels after glucose challenge, while the second generation antipsychotics risperidone, clozpaine and olanzapine were associated with significant increases (58).

**Antipsychotics and Metabolic Syndrome**

Syndrome X, later renamed metabolic syndrome, was first described in 1988 by Gerald Reaven to describe a cluster of risk factors that included hypertension, glucose intolerance, high triglycerides, and low high-density lipoprotein (HDL) cholesterol (63). Metabolic syndrome has been defined in numerous ways by various organizations over the years, but one of the more commonly accepted definitions was published by the International Diabetes Federation (IDF) in April of 2005 (64). The IDF defined metabolic syndrome by focusing on central obesity. Their definition required a person to have central adiposity defined on the basis of waist circumference, and two or more of the following four factors: elevated concentration of triglycerides, reduced contentration of HDL cholesterol, elevated blood pressure and dysglycemia (64). According to this definition, and using US data on adults from the National Health and Nutrition Examination Survey data from 1999-2002, Ford calculated that nearly 40% of the US population has metabolic syndrome (64). Using the National Cholesterol Education Program (NCEP) definition, he calculated the prevalence to be 35% (64).

The metabolic syndrome has been associated with elevated cardiovascular mortality and morbidity, elevated all-cause mortality, and risk of diabetes (63, 65). Using the NCEP definition of metabolic syndrome, the relative risk is 1.27 for all-cause mortality, 1.65 for cardiovascular disease, and 2.99 for diabetes (65). The population-attributable fraction for the metabolic syndrome was 6-7% for all-cause mortality, 12-17% for cardiovascular disease, and 30-52% for diabetes (65).

Lamberti et al. published a cross-sectional study on the prevalence of metabolic syndrome in 93 schizophrenic outpatients receiving clozapine relative to 2,701 comparison subjects (66). The prevalence of metabolic syndrome was significantly higher in patients receiving clozapine (53.8%) than among the comparison group (20.7%) (66). Within the clozapine population, associations were found with age, BMI, and duration of treatment with clozapine(66). The potentially high rate of metabolic syndrome in psychotic patients is concerning given that schizophrenia is already associated with increased cardiovascular mortality (26, 27).

**Putative Mechanisms for Antipsychotic-Induced Metabolic Abnormalities and Molecular Assessment of Metabolic Risk in Patients with Psychotic Disorders**

Researchers have offered various explanations for the cause of weight gain and metabolic disturbance in psychotic patients. None of the evidence is very strong, so most hypotheses remain rather speculative. Hypotheses have included ideas related to changes in basal metabolic rate, altered levels of baseline physical activity, alterations in appetite and satiety, insulin resistance and impaired cellular glucose metabolism.

Mouse models of visceral obesity and certain human studies have suggested an association between visceral fat and metabolic syndrome, leading some researchers to believe that the accumulation of visceral fat during antipsychotic use may underlie certain metabolic consequences of antipsychotic use (67). Mice overexpressing 11-beta hydroxysteroid dehydrogenase develop visceral fat deposition and other metabolic abnormalities, suggesting a possible pathomechanism for antipsychotics (67). Visceral fat

also correlates with insulin resistance, suggesting a possible pathological relationships where antipsychotics may be involved (68). The hormone resistin may also play a role in the development of type 2 diabetes (69). Zhang et al. conducted an imaging study in a sample of 46 patients with first-break psychosis (antipsychotic-naïve) to determine whether abdominal fat deposition increased after 10 weeks of exposure to an antipsychotic, primarily risperidone or chlorpromazine (70). It was concluded that, relative to healthy controls without exposure to antipsychotics, the subjects exposed to antipsychotics had substantitally increased desposition of subcutaneous and intra-abdominal fat (70). In these same patients, levels of leptin, fasting lipids, and non-fasting glucose were found to be elevated (70). Zhang et al.'s findings may reflect one of the reasons that waist circumference has been found to be such an effective tool for determining who is at risk for metabolic syndrome (71).

Certain genes have been associated with the development of metabolic syndrome, and interaction between these genes and antipsychotics may result in metabolic aberrations in patients (2). Genes putatively involved in the pathways that determine the effects of antipsychotics on metabolism include those encoding leptin, the leptin receptor, the melanocortin 4 receptor, pro-opiomelanocortin, prohormone convertases, B-adrenergic receptors in adipose tissue, fatty acid binding protein, lipases, mitochondrial proteins, and TNF-alpha and glycogen synthase (2).

A monozygotic twin study by Theisen et al. lends credence to the hypothesis that genetics play an important role in the development of metabolic abnormalities in patients taking antipsychotics. Theisen's group found that both twins gained weight after starting

first-generation antipsychotics and later clozapine, for a total of 38 kg and 40 kg, respectively, over a 2.5 year period (72).

Zhang et al. found a functional polymorphism -2548G/A in the promoter region of the leptin gene that was associated with significantly increased weight gain in antipsychotic-naïve patients exposed to antipsychotics (73).

A prospective study by Basile et al. involving 80 patients treated for schizophrenia with clozapine found that weight gain at 6 weeks was correlated with polymorphisms in 9 genes encoding seratonin, histamine, adrenergic receptors, cytochrome p450 or TNF-alpha (74).

The relationship between weight gain, certain atypical antipsychotics, and activation of the TNF-alpha system have suggested that the TNF-alpha system may underlie the relationship between antipsychotics and weight gain (2, 75, 76). TNF-alpha and soluble TNF receptor levels are increased in obese subjects (2). Clozapine, olanzapine, amitrypitiline and mirtazapine clearly activate the TNF-alpha system (2, 77-79). Drugs that did not cause weight gain, such as haloperidol, paroxetine and venlavaxine did not influence the TNF-alpha system (2, 78). TNF-alpha system activation does not appear to be the result of weight gain, because it occurs during the first week of treatment (2). This has led some researchers to suggest that TNF system activation might be used as a marker to predict weight gain on an individual basis, enabling early alterations in drug choice or dosing (2).

**Detection and Monitoring of Metabolic Disorders in Psychotic Patients**

The growing evidence of an association between psychotic disorders, antipsychotic drugs, and metabolic abnormalities raises the important question of how to best monitor for metabolic disorders in a population with psychotic disorders. In fact, there are many questions that must be answered with regard to screening in this population, including both those that must be answered by any healthcare screening program and those that must be answered in this particular context. For one, there is the issue of tests. Which tests are superior in terms of sensitivity and specificity? How should the tradeoff between sensitivity and specificity be managed, that is, which is more valuable for a particular test? What thresholds should be set for positive and negative results? How simple should it be to implement the test? What should prompt a clinician to order a test, that is, what positive predictive value ought to be required? Then there is the question of what interventions should be provided upon obtaining an abnormal result. Should interventions be biological, social, psychological, or some combination of the above? Should they be conducted by consults or by the physician ordering the test? Finally, related closely to the prior question, there is a health systems question, with challenges for providers, payors, and patients. Who should monitor metabolic data in the population of chronically mentally ill? Should it be PCCs, MHCs, or someone else? What responsibility do psychiatrists have to monitor metabolic data in a patient for whom they prescribe a drug that has possible metabolic side effects? If touching and examining patients interferes with certain forms of psychotherapy, should such psychotherapists abstain from prescribing drugs that require the use of a physical exam to monitor side effects? These are very real questions that arise in the context of monitoring metabolic disorders in patients with psychotic disorders, and they offer vivid examples of how

historical divisions within psychiatry, between psychiatrist-as-physician and psychiatrist-as-psychotherapist, continue to play out today.

There is evidence in the medicine literature that physicians do not adequately detect and monitor metabolic abnormalities in the general population. A group at Harvard studied 55,000 physician visits from 1995 to 1996 and found that only 8.6% of physicians reported obesity in their patients, despite a national a prevalence of 22.7%, according to the Third National Health and Nutrition Examination Surveys (NHANTES), 1988-1994 (80). In 2005 Ruser et al. conducted a cross-sectional study of 424 patients cared for by medicine residents in the Yale Internal Medicine Residency Programs and produced evidence that internal medicine residents markedly under-recognize and under-treat overweight and obesity (81). The problem of under-recognition exists not only in the case of weight, but also in the case of diabetes and other metabolic disorders. For example, according to the 1999-2002 NHANTES, 30.1% of diabetes in the general population was undiagnosed (48).

There is also inadequate identification and management of overweight and obesity in the mentally ill population. Evidence suggests that schizophrenia patients are under-treated for hypertension, dyslipidemia and diabetes (82). Nasrallah et al. used data from the Clinical Antipsychotic Trials of Intervention Effectiveness (CATIE) to evaluate rates of non-treatment in schizophrenic patients (82). They found that patients were not treated at the following rates: 30.2% for diabetes, 62.4% for hypertension, and 88.0% for dyslipidemia (82). Yet in a study by Srihari et al. at a Community Mental Health Center, which looked at patients on antipsychotic medications, 13.9% of diabetics in the schizophrenic population had previously undetected diabetes, which was in contrast to a

surprisingly higher 30.1% of diabetics who were undiagnosed in the general population (48). Additionally, a study by Dickerson et al. suggested that patients with schizophrenia and affective disorders were more likely to report receiving some general medical services in the past year than the general population, including having visited a general medical doctor and having a complete physical exam (83).

A study very relevant to this thesis was published in 2005 by Buckley et al. (84). The investigators collected responses from psychiatrists to a ten-question survey about clinical practices with regard to metabolic side effects from second-generation antipsychotics. There were 1,534 targets and 258 responses. Although 86% of respondents reported altering their prescribing behavior due to the side effect profile of second-generation antipsychotics, a full 41.7% of respondents stated that they had difficulty obtaining or were unable to obtain resources for determining waist circumference (84). Also, 23.3% of respondents stated they had difficulty obtaining or were unable to obtain resources for determining fasting blood glucose (84). Given that there is strong evidence that waist circumference and fasting blood glucose are two of the most effective tests for monitoring metabolic health risks, the data in Buckley et al.'s study is concerning (71, 85-87). The authors also examined baseline testing. They defined "frequently testing at baseline" as conducting a test more than 60% of the time before initiating therapy (84). The following percentage of psychiatrists said that they ordered the following tests "frequently" prior to initiating antipsychotic therapy: 35% glucose, 27% lipids, and 6% waist circumference (84). Less than 25% of psychiatrists obtained a blood pressure measurement "frequently" prior to initiating antipsychotic therapy (84). For each of the following tests, over 40% of psychiatrists reported that,

after starting a second generation antipsychotic, they did not routinely obtain the following tests: waist circumference, glucose, blood pressure, or lipid profile (84). Less than 25% of psychiatrists took a monthly weight (84). These results suggest that, in the context of prescribing second-generation antipsychotics, routine monitoring of metabolic metrics has not become a norm in psychiatry – in contrast to all recommendations from expert panels to date (85-87). It appears, according to Buckley et al., that "although clinicians are aware of the emergent side effect profile of second-generation antipsychotics … the impact of recent guidelines upon actual practice is, at best, modest. This is an evolving standard of care" (84).

A paper by Motsinger et al. reviewed pharmacy data from a community health center over a 6-month period for patients prescribed atypical antipsychotics (88). They found that 13% of patients prescribed atypical antipsychotics had fasting blood sugar levels and 30% had lipid panels measured during the six month study period (88). Psychiatrists ordered tests at the lowest rates, and physicians trained in primary care plus psychiatry ordered such tests at the highest rates (88).

In terms of cost effective screening for metabolic syndrome, Straker et al. produced evidence suggesting that abdominal obesity was most sensitive, at 92%, while fasting glucose was most specific, at 95.2%, for identifying the presence of metabolic syndrome (85). Combining abdominal obesity and elevated fasting blood glucose had 100% sensitivity (85). They concluded that measuring abdominal obesity via weight circumference and fasting blood glucose was a simple and cost-effective means of screening for metabolic syndrome (85).

A group in Canada, Ardern et al., determined that waist circumference (WC) was an effective tool for detecting metabolic syndrome in women with elevated BMI, but not in men (89). Janssen et al. found that WC cutoffs helped to identify increased health risk within all weight categories, including normal (71). The health risks that they reviewed included hypertension, diabetes, dyslipidemia and the metabolic syndrome (71).

Marder et al. published recommendations for physical health monitoring of patients with schizophrenia (86). The paper was a direct result of a conference at Mount Sinai School of Medicine in New York that had focused on the topic (86, 87). The authors suggested (1) clinics be capable of weighing patients; (2) patients should be encouraged to track their own weight; (3) BMI monitoring should be supplemented by waist circumference recording; (4) patients should be weighed at every visit for the first 6 months of treatment or after a medication change (86). The authors also believed that a gain of 1 unit BMI, or a waist circumference of 35 inches for women and of 40 inches for men, indicates the need for an intervention (86). In addition, the authors determined that a BMI over 25 should be cause for considering the relative risks of weight gain posed by different antipsychotics (86). They emphasized that clinicians should make an effort to recognize weight gain early to prevent weight gain and obesity, as reversal of these phenomena is extremely difficult (86).

With regard to diabetes monitoring, the Marder/Mount Sinai group had further recommendations. They suggested obtaining an initial fasting plasma glucose level before starting any new antipsychotic, with HbA1c as a secondary option (86). They recommended further testing of fasting glucose or HbA1c every 4 months if patients possess significant risk factors for diabetes (based on family history, BMI and waist

circumference) or if the patient gains weight during antipsychotic treatment (86). Providers should inform and ask patients about diabetes symptoms such as polydipsia, polyuria and weight change (86).

The Marder/Mount Sinai group also had recommendations regarding lipid monitoring (86). They suggested that all patients with schizophrenia should have a lipid panel performed, including total cholesterol, LDL, HDL, and triglycerides (86). If LDL is normal, it should be re-tested every 2 years, but if it is above 130 mg/dl, it should be tested every 6 months (86). Mental health practitioners should identify patients who fulfill the criteria for metabolic syndrome and should consider all patients with schizophrenia at risk for coronary artery disease (86).

An article in the British literature recommends an even more aggressive initial evaluation and early monitoring of metabolic metrics in schizophrenic patients. For example, they suggest obtaining monthly weights (90).

A 2006 review by Cohn et al. (91) points out that there is a dearth of literature about the cost-effectiveness of metabolic monitoring of patients taking antipsychotics (91). Also, Cohn et al. suggested that although psychiatrists do not have the obligation of becoming experts at the diagnosis and treatment of disorders such as obesity, hypertension, heart disease and diabetes, they do carry responsibility for delegating responsibility to qualified experts when appropriate (91).

Using a randomized controlled trial method, Druss et al. looked at organizational structures and the effectiveness of novel structures relative to standards (92). They contrasted outcomes in an integrated care system at the Veterans Affairs medical system, where psychiatric services were integrated with medical services, with the status quo. By

integrating care, they found psychiatric patients were more likely to visit a primary care physician and had a greater mean number of visits to a primary care clinician (92). They also determined that patients had significantly improved general health as measured by the 36-item Short-Form Health Survey (92). Although general health improved, there was no improvement in mental health (92). Importantly for healthcare administrators, the cost of the two systems was equivalent (92). This system is in fact that one at which the data for this thesis was collected.

## STATEMENT OF PURPOSE

We are at an interesting point in the history of psychiatry in which crucial questions are emerging about the role and responsibility of psychiatrists and other mental health clinicians (MHCs) in monitoring, reporting, and managing the medical illness of their patients, as well as the medical side-effects of prescribed psychiatric medications. The emergence of powerful biological treatments for psychiatric disorders, along with an increased awareness by clinicians of the medical and biological dimensions of psychiatric illness, has led to a gradual closing of the historical schism that existed between psychiatry and the rest of medicine since the beginning of the psychoanalytic era. This thesis aims to contribute productively to the discussion about the role MHCs currently play in monitoring a potentially life threatening medical side-effect of second generation antipsychotic medications. We ask this key question:  How do MHCs and primary care clinicians (PCCs) currently monitor, report and manage the metabolic status of patients

with psychotic disorders? In answering that question, we will attempt to answer a second key question: should the status quo change and if so, how?

The scientific means for answering our first key question is to put forth a hypothesis and then test it with empirical data. Our null hypothesis is that there is no difference in the rates of monitoring, identifying and intervening for metabolic abnormalities by MHCs and PCCs at the Veteran's Affairs facilities in West Haven, Connecticut. Should we reject the null hypothesis, we secondarily hypothesize that MHCs monitor, report and intervene for metabolic abnormalities in their patients at rates that are significantly lower than those of PCCs, and that such rates are too low. The results of our hypothesis testing will enable us to put forth an answer to our second key question, which was whether the status quo should change and if so, how.

## MATERIALS AND METHODS

### Study Overview

We performed a cross-sectional medical record review of overweight and obese patients with psychotic disorders at the Mental Hygiene Clinic at the West Haven Campus of the Veterans Affairs Connecticut Healthcare System (VACHS). The study was approved by the VACHS Human Investigations Committee. As data was extracted from medical records, it was de-identified in order to protect the privacy of subjects.

### Inclusion Criteria

We searched the VACHS electronic medical record database for patients who met the following inclusion criteria: (1) primary diagnosis of schizophrenia or schizoaffective disorder (from DSM-IV, International Classification of Disease codes 295.10, 295.30, 295.60, 295.70, and 295.80); (2) at least one routine mental health visit at the West Haven VA facility (VA stop codes 502, 552 and 576) between July 1, 2005 and June 30, 2006; and (3) overweight or obese, as determined by a body mass index (BMI) $\geq$ 25. We excluded all deaths. After identifying subjects who met all of our selection criteria, we had a remaining cohort of 123 subjects.

**Data Extraction Methods**

We parsed the electronic medical records of subjects in the cohort and extracted relevant data from July 1, 2005 through June 30, 2006. Data was first recorded on standardized paper sheets (see Appendix) and then entered into Microsoft Excel spreadsheets. For each subject, we examined general patient background data, primary care clinician (PCC) visit notes, and mental health clinician (MHC) visit notes. We only examined data from routine outpatient visits, ignoring emergency room visits and hospitalizations.

Two psychiatrists, one primary care physician, and one medical student extracted data from the medical records. In order to develop a standardized process for extracting and recording data, and so as to decrease inter-rater variability, all raters reviewed the same charts for four weeks and compared results on a weekly basis. Changes to the data

extraction form and discussions about the process improved the subsequent standardization of data collection.

We extracted data regarding age, sex, race/ethnicity, weight, BMI, alcohol and tobacco use, co-morbidities, antipsychotic treatment, number of PCC visits, and number of MHC visits. Co-morbidities extracted from the past medical history section of notes included diabetes type 1, diabetes type 2, hypertension, hypercholesterolemia, dyslipidemia, osteoarthritis or degenerative joint disease, obstructive sleep apnea, hyperthyroidism, metabolic syndrome, and coronary artery disease. We examined clinician notes for documentation of the following information: waist size, weight, height, and BMI. We recorded identifications of weight in the problem list, history of present illness, or assessment and plan of clinician notes. We recorded whether clinicians identified a linkage between weight and antipsychotics and whether they recommended weight loss as a therapy for a diagnosis other than obesity. We also recorded whether clinicians documented ordering any of the following laboratory tests in their notes: blood glucose, thyroid stimulating hormone (TSH), urinalysis, lipid panel, or hemoglobin A1C.

Finally, we recorded instances when interventions related to weight management were documented in clinician notes. We determined whether a weight-related referral was made for a nutrition/dietician, physical therapist, behavioral modification clinic, social worker, endocrinologist, surgeon or other consultant. We recorded whether the clinician made dietary or exercise recommendations. We also determined whether pharmacotherapy for weight loss was considered or ordered, and whether a change in antipsychotic medication or dose considered or ordered as a result of weight issues.

**Statistical Methods**

After collecting all the data, we analyzed it in Microsoft Excel. For continuous variables such as weight, we calculated descriptive statistics such as mean, median, mode, range, minimum, maximum, and standard deviation. For discrete variables characterized by either a positive or negative value, we calculated sample proportions. For any given variable there were two samples, one from primary care and one from mental health, so it was necessary to compare the sample proportions from these two groups in order to determine whether the clinician groups acted differently. Towards that end, we calculated 95% confidence intervals for the difference in sample proportions (see equation below). (All confidence intervals reported in this thesis can be assumed to be 95% confidence intervals for the difference in two proportions.) If the confidence interval did not cross zero, the difference was deemed statistically significant. We also calculated a p-value for the difference in sample proportions by setting the lower bound of the confidence interval equal to zero, determining the subsequent z-score, and thereby finding the p-value.

$$CI = \hat{\pi}_1 - \hat{\pi}_2 \pm Z_{\alpha/2}\sqrt{\frac{\hat{\pi}_1(1-\hat{\pi}_1)}{n_1} + \frac{\hat{\pi}_2(1-\hat{\pi}_2)}{n_2}}$$

## RESULTS

(Note: All tables are displayed in the text body but have also been consolidated in the Appendix.)

As displayed in **Table 1**, we found that in our population of 123 subjects, a majority of subjects were white (56%). African-Americans (21%) and Not Documented (20%) comprised significant minorities. Only 2% of subjects were Hispanic. As shown in **Table 2**, our subjects were predominantly male (93%), and the remaining only 7% were documented as female.

**Table 1: Subject Race/Ethnicity (N=123)**

| | Percent |
|---|---|
| Not Documented | 20% |
| White | 56% |
| Hispanic | 2% |
| African-American | 21% |
| Other | 1% |

**Table 2: Subject Gender (N=123)**

| | Percent |
|---|---|
| Male | 93% |
| Female | 7% |

Statistics regarding the age, weight, BMI and number of routine office visits by subjects are presented in **Table 3**. The mean age of subjects was 54 years (SD=8 years). Subjects were overweight or obese: the mean weight was 218 pounds (SD=25 pounds) and the mean BMI was 32.4 (SD=5.4). Subjects visited mental health providers more often than primary care providers. The mean number of PCC office visits was 3.4 visits (SD=4.0 visits); the mode was lower at 2.0 visits. The mean number of MHC visits was 15.6 (SD= 11.1 visits); the mode was lower at 12 visits.

**Table 3: Age, Weight, BMI, and Number of Routine Office Visits (N=123)**

| | | | | Number of Routine Visits | |
|---|---|---|---|---|---|
| | AGE (years) | WEIGHT (pounds) | BMI | Primary Care | Mental Health |
| **Mean** | 54 | 218 | 32.4 | 3.4 | 15.6 |
| **Median** | 54 | 215 | 31.3 | 2.0 | 12.0 |
| **Mode** | 54 | 233 | 31.7 | 2.0 | 12.0 |
| **Minimum** | 31 | 137 | 25.0 | 1.0 | 1.0 |
| **Maximum** | 85 | 312 | 50.5 | 41.0 | 59.0 |
| **Range** | 54 | 175 | 25.5 | 40.0 | 58.0 |
| **Standard Deviation** | 8 | 35 | 5.4 | 4.0 | 11.1 |

**Table 4** displays data on alcohol and tobacco use, as well as the co-morbidities that were extracted from the past medical history data found in clinician notes. In our sample, 51% of subjects were tobacco smokers. The most prevalent co-morbidities were the following: type 2 diabetes (23%); hypertension (57%); hypercholesterolemia (30%);

and dyslipidemia (40%). 10% of subjects carried diagnoses of diabetes, hypertension and dyslipidemia, thus meeting the criteria for metabolic syndrome – and yet there was not a single diagnosis of metabolic syndrome in the entire sample. The prevalence of coronary artery disease was 9%.

**Table 4: Subject Co-morbidities (N=123)**

| | Percent |
|---|---|
| Diabetes Type I | 1% |
| Diabetes Type II | 23% |
| Hypertension | 57% |
| Hypercholesterolemia | 30% |
| Dyslipidemia | 40% |
| Osteoarthritis; Degenerative Joint Disease | 19% |
| Obstructive Sleep Apnea | 10% |
| Hypothyroidism | 5% |
| Metabolic Syndrome | 0% |
| Coronary Artery Disease | 9% |
| Any Smoking (current) | 51% |
| Any Alcohol Use (current) | 21% |
| High Alcohol Use (current) (women >7 drinks per week; men >14 drinks per week) | 9% |

**Table 5** shows data about the rates of antipsychotic treatment in the sample population, as determined from clinician notes. We found that 37% of subjects were prescribed a first-generation antipsychotic, 85% were prescribed a second-generation

antipsychotic, 97% were prescribed at least one antipsychotic of any type, and 25% were prescribed both a first-generation and a second-generation antipsychotic. The following percentage of subjects were prescribed particular second-generation antipsychotics: 10% aripiprazole (Abilify); 11% clozapine (Clozaril); 22% olanzapine (Zyprexa); 24% quetiapine (Seroquel); 29% risperidone (Risperdal); and 8% ziprasidone (Geodon).

**Table 5: Rates of Antipsychotic Therapy (N=123)**

| | Percent |
|---|---|
| Prescribed First Generation Antipsychotic | 37% |
| Prescribed Second Generation Antipsychotic | 85% |
| Prescribed at Least One Antipsychotic of Any Type | 97% |
| Prescribed Both a First and Second Generation Antipsychotic | 25% |
| Aripiprazole (Abilify) | 10% |
| Clozapine (Clozaril) | 11% |
| Olanzapine (Zyprexa) | 22% |
| Quetiapine (Seroquel) | 24% |
| Risperidone (Risperdal) | 29% |
| Ziprasidone (Geodon) | 8% |

**Table 6** shows the rates at which PCCs and MHCs documented weight-related biometrics. In the sample population, not a single subject had their waist-size documented by a PCC or MHC. Waist-size is a fundamental biometric used for assessing risks associated with metabolic syndrome. Far more subjects had their weight documented by PCCs than by MHCs (85% vs. 11%; CI 0.66-0.82; $p<0.001$). The same was true for

documentation of BMI (48% vs. 0%; CI 0.39-0.57; p<0.001). It is notable that MHCs also failed to document BMI in any subjects.

**Table 6: Documentation of Biometrics in Visit Notes (N=123)**

| | | | 95% CI | | |
|---|---|---|---|---|---|
| | Primary Care | Mental Health | Lower Bound | Upper Bound | P-value |
| Waist size | 0% | 0% | - | - | - |
| Weight | 85% | 11% | 0.66 | 0.82 | <0.001 |
| Height | 59% | 2% | 0.48 | 0.66 | <0.001 |
| BMI | 48% | 0% | 0.39 | 0.57 | <0.001 |

**Table 7** displays the rates at which PCCs and MHCs identified weight as an issue in their notes. PCCs identified the issue of weight more often than MHCs (45% vs. 28%; CI 0.04-0.28; p<0.005). PCCs were also far more likely than MHCs to mention weight loss as a therapy for a diagnosis other than obesity (28% vs. 4%; CI 0.15-0.32; p<0.001). PCCs and MHCs rarely identified the link between antipsychotics and weight issues in their notes (10% vs. 12%; not a statistically significant difference).

**Table 7: Identification of Weight Issues Documented in Visit Notes (N=123)**

| | | | 95% CI | | |
|---|---|---|---|---|---|
| | Primary Care | Mental Health | Lower Bound | Upper Bound | P-value |
| Identified weight as an issue in problem list | 28% | 13% | 0.06 | 0.25 | <0.005 |
| Identified weight as an issue in history | 26% | 15% | 0.01 | 0.21 | <0.015 |
| Identified weight as an issue in assessment and plan | 39% | 17% | 0.11 | 0.33 | <0.001 |
| Identified weight as an issue in at least one of the following: problem list; history; assessment and plan | 45% | 28% | 0.04 | 0.28 | <0.005 |
| Identified link between weight and antipsychotics | 10% | 12% | -0.10 | 0.05 | 0.730 |
| Identified weight loss as a therapy for a diagnosis other than obesity | 28% | 4% | 0.15 | 0.32 | <0.001 |

As shown in **Table 8**, PCCs ordered laboratory tests that can identify metabolic abnormalities far more often than MHCs did. For every test, including blood glucose, thyroid stimulating hormone, urinalysis, lipid panel, and hemoglobin A1C, there was a large and statistically significant difference between the proportion of subjects that were

ordered tests by PCCs versus by MHCs. For example, PCCs ordered a blood glucose for 50% of subjects and MHCs only did so for 4% of subjects (CI 0.36-0.55; p<0.001).

**Table 8: Tests Ordered in Visit Notes (N=123)**

| | | | 95% CI | | |
|---|---|---|---|---|---|
| | Primary Care | Mental Health | Lower Bound | Upper Bound | P-value |
| Blood glucose | 50% | 4% | 0.36 | 0.55 | <0.001 |
| TSH | 15% | 4% | 0.04 | 0.19 | <0.005 |
| Urinalysis | 14% | 0% | 0.08 | 0.20 | <0.001 |
| Lipid panel | 71% | 2% | 0.61 | 0.77 | <0.001 |
| Hemoglobin A1C | 25% | 2% | 0.16 | 0.32 | <0.001 |

**Table 9** shows the weight-management interventions that were documented by PCCs and MHCs. PCCs were more likely than MHCs to make dietary and exercise recommendations to subjects (42% vs. 19%; CI 0.12-0.35; p<0.001) and they were also more likely to order a weight-management referral (21% vs. 3%; CI 0.10-0.26; p<0.001). Although MHCs were more likely than PCCs to order or to consider ordering a change of antipsychotic medication or dose due to weight-related issues (6% vs. 3%), the difference was not statistically significant. The low rates at which clinicians considered altering antipsychotic prescriptions for weight-related reasons is echoed by the low rates at which they linked weight issues to antipsychotic treatment in their notes (see Table 7).

**Table 9: Interventions For Weight Loss Documented in Visit Notes (N=123)**

| | | | 95% CI | | |
|---|---|---|---|---|---|
| | Primary Care | Mental Health | Lower Bound | Upper Bound | P-value |
| Made dietary recommendations to patient | 57% | 25% | 0.20 | 0.43 | <0.001 |
| Made exercise recommendations to patient | 46% | 22% | 0.12 | 0.35 | <0.001 |
| Made exercise and dietary recommendations to patient | 42% | 19% | 0.12 | 0.35 | <0.001 |
| Ordered at least one referral for weight management | 21% | 3% | 0.10 | 0.26 | <0.001 |
| Considered or ordered pharmacotherapy for weight loss | 1% | 0% | -0.01 | 0.02 | 0.158 |
| Considered or ordered change of antipsychotic medication due to weight | 2% | 4% | -0.06 | 0.03 | 0.764 |
| Considered or ordered change of antipsychotic dose due to weight | 2% | 5% | -0.07 | 0.02 | 0.846 |
| Considered or ordered change of antipsychotic medication and/or dose due to weight | 3% | 6% | -0.08 | 0.03 | 0.823 |

## DISCUSSION

In order to better understand how mental health clinicians (MHCs) and primary care clinicians (PCCs) identify, monitor and intervene for metabolic disorders in patients with psychotic disorders, we studied the medical records of 123 overweight or obese veterans (BMI ≥ 25) with psychotic disorders at the Veteran Affairs outpatient facilities in West Haven, Connecticut during a one year period from July 1, 2005 to June 30, 2006. Our subjects were predominantly white and male (56% and 93%, respectively), with an average age of 54 years (SD=8 years). The subjects had an average weight of 218 pounds (SD=35 pounds) and an average body mass index (BMI) of 32.4 (SD=5.4). We found that 97% of subjects were taking an antipsychotic of some sort, and 85% were taking a second-generation antipsychotic – the category most frequently associated with metabolic side effects. The most frequently prescribed second-generation antipsychotics were risperidone (29%), quetiapine (24%), and olanzapine (22%), all of which have been associated with metabolic side-effects in the medical literature – particularly risperidone and olanzapine (see Introduction for details)**.** In general, we found that, despite far more average annual visits to MHCs than PCCs (15.6 visits vs. 3.4 visits), PCCs were much more likely than MHCs to identify, monitor and treat a variety of weight and metabolic parameters in our study population over the course of a year.

Our results have led us to reject the null hypothesis that there is no difference in the rates of monitoring, identifying and intervening for metabolic disorders by MHCs and PCCs at the Veteran's Affairs facilities in West Haven, Connecticut. We also accept our secondary hypothesis, which is that MHCs monitor, report and manage metabolic

abnormalities in their patients at rates that are significantly lower than those of PCCs. We will discuss the evidence leading to these conclusions in the subsequent sections of this Discussion.

**Documentation of Biometrics by Clinicians**

MHCs documented weight-related biometrics at extremely low rates, both absolutely and relative to PCCs. During the one-year study period, MHCs documented weight measurements for only 11% of subjects, whereas PCCs documented weight measurements for 85% of subjects (CI 0.66-0.82; $p<0.001$). Similarly, MHCs documented BMI for 0% of subjects, whereas PCCs documented BMI for 48% of subjects (CI 0.39-0.57; $p<0.001$). Our findings are concerning because they suggest that PCCs were seven to eight times more attentive to measurements of weight than MHCs, despite the fact that 97% of subjects in the study were prescribed antipsychotics by MHCs. A possible explanation for the disparity between clinician groups is that MHCs might expect PCCs to monitor the weight-related side effects of the antipsychotics that MHCs prescribe. Also, MHCs may not have the resources to perform weight monitoring, such as scales and support staff. That being said, even if MHCs do rely upon PCCs to perform all metabolic monitoring, such an expectation should be explicit and the results from primary care consultations and follow-up should be documented clearly in mental health visit notes.

**Diagnosis and Monitoring of Metabolic Syndrome by Clinicians**

It is concerning that, although our subjects carried significant rates of diagnoses that are associated with metabolic syndrome, including 23% type 2 diabetes, 55% hypertension, and 38% dyslipidemia —10% of subjects carried all three diagnoses—, there was not a single clinician that identified a subject as having metabolic syndrome. It seems highly unlikely that, in the setting of such co-morbidities, there was truly a 0% prevalence of metabolic syndrome in the cohort. Moreover, given that the literature suggests that the general prevalence of metabolic syndrome in the United States may be as high as 40%, clinicians in our study were likely significantly under-diagnosing metabolic syndrome (64). We also determined that zero subjects were measured for a waist-circumference by a PCC or MHC during the study period. This too is alarming, given that the literature has deemed waist circumference to be one of the most valuable metrics for diagnosing metabolic syndrome risk and for assessing associated health risks (see Introduction for details) (71, 85). Our results indicate that the level of clinician awareness about metabolic syndrome, and the rate at which it is diagnosed and followed by PCCs and MHCs, may be grossly inadequate. Given the morbidity associated with metabolic syndrome, and the baseline cardiovascular risks associated with schizophrenia (26, 27), our findings suggests that subjects in our study may face significantly greater health risks if the quality of monitoring is not improved.

**Identification and Interpretation of Weight Issues by Clinicians**

We examined the rate at which clinicians listed weight as an issue in certain key sections of the visit note, including the problem list, the past medical history, and the assessment and plan. When we evaluated how often clinicians identified weight as an issue in at least one of these sections of the visit note, the difference between groups was significant: PCCs identified weight as an issue in 45% of subjects, whereas MHCs identified it in only 28% of subjects (CI 0.11-0.33; $p<0.005$). Thus weight does not appear to be a standard issue addressed by clinicians in patients who are overweight or obese.

We also found that both MHCs and PCCs infrequently identified the link between antipsychotics and weight gain in their notes (10% vs. 12%; not a statistically significant difference). Given the extensive literature linking antipsychotics to weight gain, which we discussed in the Introduction, the fact that all of the subjects in our study were overweight or obese, and the fact that almost all subjects were taking an antipsychotic, clinicians should have remarked on this linkage more frequently in their notes. The failure to identify the link between antipsychotics and weight was echoed by the low rates at which PCCs and MHCs ordered or considered ordering adjustments to antipsychotic prescriptions due to metabolic side-effects (3% and 6%, respectively; not a statistically significant difference). It is surprising that, despite MHCs' expertise in psychopharmacology and their role as the primary prescribers of antipsychotics in our study sample, they did not significantly outperform PCCs on criteria of linkage or prescription change.

Interestingly, PCCs were also much more likely than MHCs to recommend weight loss for a diagnosis other than obesity (28% versus 4%; CI 0.15-0.32; $p<0.001$).

This reveals that, at least as understood by their documenting habits, PCCs displayed a greater awareness of the health benefits offered by weight loss beyond merely loss of weight (e.g. lower triglycerides and lower blood pressure). Given that PCCs are tasked with monitoring and treating a much wider range of medical conditions than MHCs (e.g. diabetes and heart disease), it makes sense that they would be more attuned to, and concerned about, how weight issues interact with non-psychiatric disease processes. At the same time, given that the schizophrenic psychiatric population faces increased risks for several co-morbid medical diseases and conditions (26, 27, 93), MHCs should be attentive to those risks and how they are monitored**.**

**Laboratory Testing and Intervention by Clinicians**

On almost every dimension of laboratory testing or intervention that we studied in clinician notes, PCCs were more interventionalist than MHCs. According to their notes, PCCs ordered all of the following tests at higher rates than MHCs: glucose (ordered for 50% versus 4% of subjects; CI 0.36-0.55; $p<0.001$); thyroid stimulating hormone (15% versus 4%; CI 0.04-0.19; $p<0.005$); urinalysis (14% versus 0%; CI 0.08-0.20; $p<0.001$), lipid panel (71% versus 2%; CI 0.61-0.77; $p<0.001$); and hemoglobin A1c (25% versus 2%; CI 0.16-0.32; $p<0.001$). In terms of interventions, PCCs documented ordering at least one referral for weight or metabolic-related concerns for 21% of subjects, whereas MHCs only did so for 3% of subjects (CI 0.10-0.26; $p<0.001$). In terms of counseling, PCCs counseled 42% of subjects on both diet and exercise, while MHCs only did so for

19% of subjects (CI 0.12-0.35; p<0.001). Additionally, only one PCC – and not a single MHC – mentioned pharmacotherapy for weight loss in their note.

As mentioned earlier, rates of recommending a change of antipsychotic drug or dose were very low among both PCCs and MHCs (3% vs. 6%; not a statistically significant difference). This was surprising, given that the literature has documented that different antipsychotics carry different metabolic risk profiles, and hence patients who experience metabolic side-effects from one antipsychotic may benefit from a change to another drug or a lower dose (see Introduction for details). Our data suggests that MHCs may be focusing primarily on the management of psychiatric symptoms without devoting sufficient attention to the management of drug side-effects.

**Study Limitations**

Since our study focused exclusively on veterans who receive their care within a single healthcare system, it is difficult to extrapolate the results to other settings, such as private practice psychiatry, prisons, or other state-funded medical centers. Also, one of the primary care clinics at the West Haven VA, where we conducted our study, is in the same building as a mental health clinic and focuses specifically on patients with severe mental illness. This sort of integration and communication between primary care and mental health is not standard in the nation. One would suspect that this model actually enhances both PCC and MHC awareness about the metabolic side-effects of antipsychotics, and thus that more disjoined healthcare delivery systems would perform even worse.

It is worth noting that the PCCs and MHCs who cared for the subjects in our study had varying levels of professional education and expertise. For example, PCCs were predominantly attending physicians, resident physicians, and advanced practice registered nurses (APRNs), whereas MHCs included a broad mixture of attending physicians, resident physicians, APRNs, registered nurses (RNs), psychologists, and social workers (although 93% had contact with an attending physician, resident physician, or APRN during the study period). It is not clear how this diversity of caregivers affected rates of identification, monitoring and treatment in the PCC and MHC groups.

Our study is also limited by the fact that we primarily gathered clinical data from clinician notes, rather than from laboratory transactions or directly observed behavior. The focus on documentation could have led to a biased representation of actual clinician behaviors. First, if the culture and attitudes that inform MHCs' documentation habits differ significantly from those of PCCs, then the two groups could have systematic differences in documentation outputs. For example, it might be the case that PCCs write-up their blood glucose orders in notes more regularly than MHCs, even though the two groups might actually request blood glucose orders at identical rates. Likewise, MHCs and PCCs might identify weight as an issue and counsel patients on diet and exercise at equal rates, but they might document such findings and actions differently. If these types of systematic documentation differences exist between groups, then our study would reveal more about communication and the documentation of behaviors than it would about the behaviors themselves. Future studies could actually evaluate documentation

differences by comparing documentation in notes to laboratory orders or observed behaviors.

Aside from systematic inter-group documentation biases, there may also be general documentation issues affecting our study. It is likely that clinicians only document a limited sample of the behaviors that they actually perform, and similarly, that they only document a limited sample of the clinical knowledge that they generate. Thus documentation rates may appear much lower than one would expect. A good example of this might be coronary artery disease: if clinicians did not document the condition, then we did not see it in the past medical history, and so the rates of coronary artery disease may have appeared low – even if clinicians were aware of and treating the condition.

Another documentation issue that may have impacted our study results is that PCCs may have issued the orders that we examined for reasons unrelated to antipsychotics or metabolic issues (e.g. ordering a urinalysis to assess urinary tract infection symptoms). This activity may have muddled our comparisons between PCCs and MHCs on the basis of tests ordered, because MHCs are responsible for identifying and treating a more limited panel of non-psychiatric diseases than PCCs.

We used three physicians and one medical student to analyze the medical records and this raises the possibility that there was inter-rater variability. In addition, there may have been intra-rater variability, considering the large amount of note material that we were evaluating. For example, there is a certain degree of subjectivity in determining whether a clinician has definitively "identified a link between weight and antipsychotics as an issue" in his or her note, and a rater's analysis may vary for a given rater or between raters depending on the material and context in any given note. To counteract bias, all of

the raters gathered on a regular basis to compare approaches and to discuss ambiguous cases. Furthermore, during the first four weeks of analysis the group met weekly to discuss a set of comparison cases thereby helping to standardize data collection methods.

Our study looked at collective rates of identification, monitoring and treatment across a one-year period, yet this may have masked the disparities that exist on a per-note basis. For example, "identified weight as an issue" was marked a positive "yes" if it occurred at least once in a clinician note during the one-year period. So there could have been many positives among either many or few notes during the year, or a single positive among either many or few notes. In all of these cases the outcome would be the same: a positive "yes." This phenomenon of ambiguity could also occur in the case of negatives. If the data had been examined on a per-note basis, we may have found either an amplification or diminution of differences between PCCs and MHCs. Our suspicion is that, since the average number of MHC visits in our study was much higher than the number of PCC visits (15.6 visits vs. 3.4 visits), the differences would in fact be amplified, lending further evidence to support our finding that, relative to MHCs, PCCs more aggressively identify, monitor and treat metabolic dysfunction in patients with psychotic disorders.

**General Implications of Study Results**

MHCs often prescribe antipsychotics that cause metabolic side-effects to patients with psychotic disorders, and yet our study has shown that they infrequently monitor such side-effects. These results bring objective evidence to support the more subjective results

that were reported in Buckley et al.'s survey of physicians, from which the researchers concluded, "Although clinicians are aware of the emergent side-effect profile of second-generation antipsychotics … the impact of recent guidelines upon actual practice is, at best, modest. This is an evolving standard of care" (84). Our results also show that patients suffering from psychosis interact more frequently with MHCs than PCCs, and in the general community many psychotic patients probably do not regularly see PCCs. This creates a situation that allows patients who develop side-effects to fall systematically through the cracks of the "monitoring system" (if such a system could accurately be said to exist). It seems that MHCs should be charged with the primary responsibility to monitor the side-effects of the drugs they prescribe. Just as MHCs are professionally responsible for monitoring for agranulocytopenia in patients who take clozapine, MHCs should also be responsible for monitoring for metabolic side-effects in patients who take antipsychotics, particularly second-generation antipsychotics. Indeed, MHCs should be identifying and monitoring patients with metabolic disorders for a variety of reasons: (1) MHCs prescribe the antipsychotics that cause metabolic side-effects; (2) MHCs see their patients more often than PCCs; (3) patients without access to PCCs may still access MHCs; (4) MHCs often understand patients' biopsychosocial context better other clinicians; and (5) MHCs often have very strong relationships and interpersonal leverage with their patients. Relative to the Mount Sinai Conference guidelines regarding monitoring for the metabolic side-effects of antipsychotics, which we discussed in detail in the Introduction, the MHC system in our study, and perhaps nationally too, is underperforming (86, 87). Rather than describing this situation as an "evolving standard of care," in the words of Buckley et al., we believe it might be more accurately described

as a “dangerous failure in quality control.” Indeed, undetected metabolic side-effects from atypical antipsychotics can be life-threatening. This story is yet another example of the serious quality control problems that are rampant in American healthcare. Quality control systems in American healthcare are abysmal when compared to those of industries such as the airline industry or of companies such as Toyota.

After MHCs identify patients with develop metabolic side-effects, the subsequent step is either to manage the issue themselves or to obtain a consultation from a specialist who can do so. Our study suggests that neither active management of side-effects nor the consultation of specialists is happening adequately today.

There is no value in merely pointing fingers or allocating blame for what is without question a system-wide failure of quality control. Instead, efforts must be made by leadership at both the local and national level to introduce systemic reforms that will ensure that all patients will be appropriately monitored and treated for the metabolic side-effects of antipsychotics.

Briefly, we might gain some insight on today’s quality-control problem by speculating about its historical origins. It is not unreasonable to suspect that today’s problem began with the historical separation of psychiatry and the rest of medicine. In the past, both the lack of biological treatments for psychiatric disorders and the dominance of psychoanalytic practice, in which the patient’s body was rarely if ever touched, led the daily routines of psychiatry to diverge from the rest of medicine. Metaphorically, the stethoscope was put aside and replaced by the couch. But since the advent and proliferation of biological treatments, such as antipsychotics, those routines have needed to change and become reacquainted with the rest of medicine. Nevertheless, it seems as

though the historical residue indeed persists, leaving us with a psychiatric approach to patient care, and a system of mental health, that continues to focus insufficient attention on the body relative to the mind.

**Options for a Local Response to Study Results**

If the implications of our findings are interpreted narrowly, so as to apply only locally, then we should examine what our findings mean for the West Haven VA. Clearly the metabolic parameters of patients on second-generation antipsychotics are not being adequately followed by MHCs at the West Haven VA, and this is systemic quality failure. Thus system reform is critical. Six solution frameworks can be used to quickly address the quality failure: (1) establish quality goals and objectives; (2) appoint leadership; (3) educate; (4) allocate resources; (5) facilitate dialogue; and perhaps most importantly, (6) standardize work processes.

Within each of these six frameworks, there are some very direct steps that the West Haven VA could take to have rapid impact. Quality improvement could be declared a goal, supported by explicit and measurable objectives. A leader could be appointed to coordinate and monitor the organization's drive toward quality improvement. This leader should be familiar with the tools and concepts of quality management (94-98). In terms of education, sending a letter to all MHCs regarding the findings of this study, along with a copy the Mount Sinai guidelines, would be one way to initiate an awareness campaign. Our findings and the Mount Sinai guidelines could be presented at a mental health staff meeting or mental health grand rounds. Also, the Mount Sinai guidelines could be posted

in the mental health clinics as a reminder to staff about the importance of monitoring for metabolic disorders, and information about the weight and diabetes management referral resources that are available to clinicians at the VA could be consolidated and disseminated to providers. In terms of resource allocation, leadership would need to first determine what resources are already in place, followed by the deployment of budgetary resources and personnel to address gaps (e.g. lack of scales, measuring tapes, or technical staff). Regarding facilitating a dialogue, gatherings could be organized to bring together clinicians from different areas of the West Haven VA provider community in order to discuss ways to improve the management of metabolic disorders within the network. Finally, in order to standardize processes, management at the West Haven VA could put in place a clinical reminder that "pops up" in the electronic medical record of overweight or obese patients who take antipsychotics. This would remind MHCs to evaluate the weight and other metabolic parameters of such patients. By standardizing the work process in this way, variance in clinician behavior would drop and quality of care would subsequently improve.

Another issue to address is that of process standards for documentation and notes within the electronic medical records, which is a more general problem throughout the VA healthcare system. Anecdotally, those familiar with the VA system will often comment on the problem of clinicians copying and pasting notes, and the problem of casually written notes that possess inadequate relevant information. The concern is that there is too little relevant information and too much irrelevant information in the system – the signal to noise ratio is low. We encountered this problem repeatedly during our study. Poor documentation ultimately obfuscates critical information and systematically impairs

communication, thereby adversely affecting the quality of patient care throughout the system. Policies, incentives and better workflow designs need to be implemented at the VA to resolve the problem, perhaps on a hospital or even nation-wide basis.

**Options for a National Response to Study Results**

If the implications of our findings are interpreted more broadly, that is if we consider them as potentially representative of a phenomenon occurring on a national level rather than simply at the level of the West Haven VA, then we must address a vast and complex problem. In fact, the situation may be much worse for patients on a national level than it was in our study. For one, the West Haven VA is affiliated with the Yale academic medical network, so one might assume that the clinicians there are more up-to-date on the current standards of care than clinicians at non-academic sites around nation. Also, the VA system offers relatively integrated and well-coordinated care, especially at the West Haven VA, where there is a primary care clinic devoted exclusively to patients with severe mental illnesses. But outside of the VA, mental healthcare in the US is often isolated from the rest of medicine. This makes the consultation process a challenge and may limit patient access to primary care physicians who monitor metabolic parameters. For example, community nephrologists, cardiologists, and renal transplant surgeons communicate regularly, but community psychiatrists are often outside of such quotidian communication and referral loops. Our study raises the question of whether this status quo should change. Indeed, psychiatrists are physicians first and psychiatrists second, working within the medical model of care, and so more effort should be brought to

integrate them with the greater medical community. Otherwise patients, like those in our study, will suffer from the effects of a poorly coordinated system.

How can we address the poor quality of metabolic monitoring and treatment on a national level? To begin, research such as this study must continue so that we can better understand how the current system of care operates and thus how it should change. Subsequently, leaders in mental health must accordingly formulate recommendations and guidelines to address the concerns raised by such research. The Mount Sinai guidelines are a good example of how this can be done. An absolutely critical step, and perhaps also the most challenging one, is to mobilize professional change. An effort is required by academic leaders, continuing medical education groups, and professional organizations – particularly the American Psychiatric Association – to drive awareness and implementation of guidelines. The effort and collaboration of non-psychiatric professional groups, such as the American Medical Association and American College of Physicians, is also needed. These groups must assist in bringing mental health care closer to the rest of medicine so that the problems of obesity and diabetes can be tackled in a coordinated fashion (among other issues). It is particularly important to link-up primary care clinicians and internal medicine specialists who focus on metabolic disorders with networks of psychiatrists and other MHCs.

In addition to leveraging the intrinsic incentives of patient care and professionalism, extrinsic financial incentives, tied to measurements, can be put in place to drive the adoption of guidelines and recommendations. Thus lobbying at various governmental levels, including Congress and the Center for Medicare and Medicaid Services, should press forward to ensure appropriate reimbursement and parity for mental

health services. As performance measurements and performance-based incentives become more mainstream, and if they are effective at improving outcomes, one would hope that these tools would also be used to drive the adoption of guidelines and recommendations in the area of metabolic monitoring – and that appropriate reimbursement would be tied to such activities. Nevertheless, performance measurements alone, even in the absence of financial incentives, could drive change simply by drawing attention to the importance of metabolic monitoring. Yet to implement performance measurements on a large scale would require extensive improvements in information technology and communication throughout the fragmented mental health system, a scenario that appears unlikely in the near future in the absence of serious national healthcare reform.

It will also be important for the concepts of quality control and of total quality management to finally be absorbed and applied in a serious manner in American healthcare. Medicine has remained far too insular and has a tremendous amount to learn from other fields such as business operations management, manufacturing, and military systems control. It will be necessary for leaders within medicine to acquaint themselves with the concepts of quality management (94-98), and to retain experts from other fields such as business and manufacturing in order to improve the quality of healthcare delivery systems.

Setting aside broader issues of professional change and national healthcare reforms, there does exist one very direct solution that could rapidly change clinician behavior nationally, and it is based on what we have learned from clozapine regulation. The manufacturers of clozapine require psychiatrists to submit the results of a blood cell

count before the drug can be dispensed. A similar requirement to report biometrics (e.g. BMI and waist-size), in order for a pharmacy to dispense an antipsychotic, would rapidly alter psychiatric practice.

## Future Research Directions

Beyond the content of this study, there is much work to be done. It would be useful to better understand how rates of documentation relate to actual behaviors, for example by comparing documentation of laboratory orders to laboratory transactions. It would also be interesting to determine if there are systemic differences between the documentation behavior of MHCs and PCCs, which one might suspect could be due to differences in professional culture, such as the high priority that MHCs may place on privacy relative to PCCs as a result of their training.

It is also critical to study whether the adoption of monitoring guidelines, such as those from Mount Sinai, actually improve outcomes (e.g. minimizes weight gains or decreases diabetes conversion rates). In order to drive individual and organizational change in the era of evidence-based medicine, proof that certain behaviors are linked to improved outcomes is critical. Then, if it is found to be true that metabolic monitoring by MHCs does indeed improve outcomes in areas such as weight gain and diabetes, we need in turn to better understand how to change clinician behavior such that guidelines will be adopted rapidly. An understanding of how to drive systemic change in clinical behavior might be found in the analysis of historical attempts to do so, or through cross-pollination by examining the literature on how other service industries improve quality through total

quality management systems such as six-sigma (94-98). In addition to incorporating guidelines into practice and measuring their impact, such guidelines should be regularly updated to incorporate new tools as they emerge (e.g. promising genetic and metabolic tests, such as TNF-alpha, which were discussed in the Introduction).

Once a metabolic problem is recognized, the next step is intervention, and so researchers must also continue to create new interventions – and to verify the value of those that are used in current practice. Weight gain that results from antipsychotic treatment may require interventions that differ from those that are used for other types of weight gain. For example, one might ask: what are the relative benefits of changing antipsychotic dose, changing antipsychotic drug, consulting a nutritionist, or starting pharmacotherapy (e.g. metformin) for weight-gain? These and similar questions would be valuable to answer. Indeed, some researchers have already started to do so, a subject that we reviewed in the Introduction.

**Conclusion**

In this cross-sectional study of 123 overweight or obese US veterans with psychotic disorders, 97% of whom were taking an antipsychotic, we determined with statistical significance that mental health clinicians identify, monitor and intervene for metabolic disorders, such as weight gain and hyperglycemia, at much lower rates than primary care physicians, and such rates are too low. We suspect that this is a problem of systemic, not individual, omission. Our findings are disconcerting given the growing evidence that the antipsychotics prescribed by mental health clinicians – particularly

second-generation antipsychotics – are linked to the development of metabolic dysfunction. In order to address this system-wide pattern of clinician behavior, which may be leading to serious adverse consequences for patients, we believe that organizational and operational systems must be changed, and that quality control systems must be instituted. Changes to the status quo ought to occur as soon as possible on both a local and national level so as to prevent harm to patients. Otherwise patients may continue to develop higher rates of preventable weight gain, diabetes, and metabolic syndrome – along with the life-threatening co-morbidities that are associated with such conditions.

# APPENDIX

## TABLES

**Table 1: Subject Race/Ethnicity (N=123)**

| | Percent |
|---|---|
| Not Documented | 20% |
| White | 56% |
| Hispanic | 2% |
| African-American | 21% |
| Other | 1% |

**Table 2: Subject Gender (N=123)**

| | Percent |
|---|---|
| Male | 93% |
| Female | 7% |

**Table 3: Age, Weight, BMI, and Number of Routine Office Visits (N=123)**

| | | | | Number of Routine Visits | |
|---|---|---|---|---|---|
| | AGE (years) | WEIGHT (pounds) | BMI | Primary Care | Mental Health |
| **Mean** | 54 | 218 | 32.4 | 3.4 | 15.6 |
| **Median** | 54 | 215 | 31.3 | 2.0 | 12.0 |
| **Mode** | 54 | 233 | 31.7 | 2.0 | 12.0 |
| **Minimum** | 31 | 137 | 25.0 | 1.0 | 1.0 |
| **Maximum** | 85 | 312 | 50.5 | 41.0 | 59.0 |
| **Range** | 54 | 175 | 25.5 | 40.0 | 58.0 |
| **Standard Deviation** | 8 | 35 | 5.4 | 4.0 | 11.1 |

**Table 4: Subject Co-morbidities (N=123)**

| | Percent |
|---|---|
| Diabetes Type I | 1% |
| Diabetes Type II | 23% |
| Hypertension | 57% |
| Hypercholesterolemia | 30% |
| Dyslipidemia | 40% |
| Osteoarthritis; Degenerative Joint Disease | 19% |
| Obstructive Sleep Apnea | 10% |
| Hypothyroidism | 5% |
| Metabolic Syndrome | 0% |
| Coronary Artery Disease | 9% |
| Any Smoking (current) | 51% |
| Any Alcohol Use (current) | 21% |
| High Alcohol Use (current) (women >7 drinks per week; men >14 drinks per week) | 9% |

**Table 5: Rates of Antipsychotic Therapy (N=123)**

| | Percent |
|---|---|
| Prescribed First Generation Antipsychotic | 37% |
| Prescribed Second Generation Antipsychotic | 85% |
| Prescribed at Least One Antipsychotic of Any Type | 97% |
| Prescribed Both a First and Second Generation Antipsychotic | 25% |
| Aripiprazole (Abilify) | 10% |
| Clozapine (Clozaril) | 11% |
| Olanzapine (Zyprexa) | 22% |
| Quetiapine (Seroquel) | 24% |
| Risperidone (Risperdal) | 29% |
| Ziprasidone (Geodon) | 8% |

**Table 6: Documentation of Biometrics in Visit Notes (N=123)**

| | | | 95% CI | | |
|---|---|---|---|---|---|
| | Primary Care | Mental Health | Lower Bound | Upper Bound | P-value |
| Waist size | 0% | 0% | - | - | - |
| Weight | 85% | 11% | 0.66 | 0.82 | <0.001 |
| Height | 59% | 2% | 0.48 | 0.66 | <0.001 |
| BMI | 48% | 0% | 0.39 | 0.57 | <0.001 |

**Table 7: Identification of Weight Issues Documented in Visit Notes (N=123)**

| | | | 95% CI | | |
|---|---|---|---|---|---|
| | Primary Care | Mental Health | Lower Bound | Upper Bound | P-value |
| Identified weight as an issue in problem list | 28% | 13% | 0.06 | 0.25 | <0.005 |
| Identified weight as an issue in history | 26% | 15% | 0.01 | 0.21 | <0.015 |
| Identified weight as an issue in assessment and plan | 39% | 17% | 0.11 | 0.33 | <0.001 |
| Identified weight as an issue in at least one of the following: problem list; history; assessment and plan | 45% | 28% | 0.04 | 0.28 | <0.005 |
| Identified link between weight and antipsychotics | 10% | 12% | -0.10 | 0.05 | 0.730 |
| Identified weight loss as a therapy for a diagnosis other than obesity | 28% | 4% | 0.15 | 0.32 | <0.001 |

**Table 8: Tests Ordered in Visit Notes (N=123)**

| | | | 95% CI | | |
|---|---|---|---|---|---|
| | Primary Care | Mental Health | Lower Bound | Upper Bound | P-value |
| Blood glucose | 50% | 4% | 0.36 | 0.55 | <0.001 |
| TSH | 15% | 4% | 0.04 | 0.19 | <0.005 |
| Urinalysis | 14% | 0% | 0.08 | 0.20 | <0.001 |
| Lipid panel | 71% | 2% | 0.61 | 0.77 | <0.001 |
| Hemoglobin A1C | 25% | 2% | 0.16 | 0.32 | <0.001 |

**Table 9: Interventions For Weight Loss Documented in Visit Notes (N=123)**

| | | | 95% CI | | |
|---|---|---|---|---|---|
| | Primary Care | Mental Health | Lower Bound | Upper Bound | P-value |
| Made dietary recommendations to patient | 57% | 25% | 0.20 | 0.43 | <0.001 |
| Made exercise recommendations to patient | 46% | 22% | 0.12 | 0.35 | <0.001 |
| Made exercise and dietary recommendations to patient | 42% | 19% | 0.12 | 0.35 | <0.001 |
| Ordered at least one referral for weight management | 21% | 3% | 0.10 | 0.26 | <0.001 |
| Considered or ordered pharmacotherapy for weight loss | 1% | 0% | -0.01 | 0.02 | 0.158 |
| Considered or ordered change of antipsychotic medication due to weight | 2% | 4% | -0.06 | 0.03 | 0.764 |
| Considered or ordered change of antipsychotic dose due to weight | 2% | 5% | -0.07 | 0.02 | 0.846 |
| Considered or ordered change of antipsychotic medication and/or dose due to weight | 3% | 6% | -0.08 | 0.03 | 0.823 |

**DATA ENTRY FORMS**

ID Numl

**BMI Survey- Data Extraction Tool**

**Date:** (mm/dd/yy) ☐☐/☐☐/☐☐

**Data Collector:** ☐CR ☐MR ☐RR ☐Other _____

**Site:** ☐VA ☐CMHC

**Patient Data:**

**DOB:** (mm/dd/yy) ☐☐/☐☐/☐☐

**Race/ Ethnicity:**
- ☐ **ND (ND= not documented)**
- ☐ **White**
- ☐ **Hispanic**
- ☐ **African-American**
- ☐ **Other:** _____

**Patient Gender:** ☐ **Male** ☐ **Female**

***First measurement between 7/1/05- 7/1/06*

**Weight in lbs:** ☐☐☐

**Height in inches:** ☐☐☐

**Calculate BMI=**(wt in lbs x 703/ht in inches$^2$) ☐☐.☐

**BMI Survey- Data Extraction Tool**

****Data from visits between 7/1/05-7/1/06*

**Number of Routine PC Visits:** _____

**Number of Routine MH Visits:** _____ *(**include CSP, MHICM, and CTI notes)*

**Location:** ☐ **West Haven** ☐ **Newington** ☐ **CBOC**

**Mental Health Provider:**

**Status:** ☐ Trainee ☐ Attending ☐ APRN ☐ RN ☐ PhD ☐ SW

**Gender:** ☐ Male ☐ Female

**Patient Medical History:**

☐ Diabetes Type 1
☐ Diabetes Type 2
☐ Hypertension
☐ Hypercholesterolemia
☐ Dyslipidemia
☐ Osteoarthritis/ DJD
☐ Gall bladder dz/ cholecystectomy
☐ Polycystic Ovary Disease
☐ Obstructive Sleep Apnea
☐ Hypothyroidism
☐ Metabolic Syndrome
☐ Coronary Artery Disease
☐ Breast CA
☐ Colon CA
☐ Prostate CA

**Social History:**

☐ Current smoker
☐ Current alcohol drinker
☐ Etoh consumption exceeds 7 per week for women/ 14 per week for men

**Antipsychotic Medication**

☐ 1st Generation
☐ 2nd Generation

☐ Aripiprazole (Abilify) ☐ Clozapine (Clozaril) ☐ Olanzapine (Zyprexa)
☐ Quetiapine (Seroquel) ☐ Risperidone (Risperdal) ☐ Ziprasidone (Geodone)

ID Number_____

**BMI Survey- Data Extraction Tool**

**Primary Care:**

**Visit Data:**

Waist size (in inches) documented: ☐ Y ☐ N  If yes, waist size: ☐☐

Weight documented: ☐ Y ☐ N

Height documented: ☐ Y ☐ N

BMI documented: ☐ Y ☐ N

Weight listed on problem list in chart? ☐ Y ☐ N

Weight listed in Past Medical History? ☐ Y ☐ N

Weight listed on Assessment/Plan? ☐ Y ☐ N

Weight loss recommended as therapy for diagnosis other than obesity? ☐ Y ☐ N

Weight linked to antipsychotics? ☐ Y ☐ N

**Primary Care Action Plan included:**

Testing:

Fasting glucose: ☐ Y ☐ N

Thyroid hormone: ☐ Y ☐ N

HbA: ☐ Y ☐ N

Fasting lipids: ☐ Y ☐ N

UACR: ☐ Y ☐ N

Other:

Referral to nutrition/dietician: ☐ Y ☐ N

Referral to PT: ☐ Y ☐ N

Referral to Counseling/ Beh Mod/ MOVE ☐ Y ☐ N

Referral to Social Work? ☐ Y ☐ N

Referral to endocrinologist? ☐ Y ☐ N

Referral to surgery? ☐ Y ☐ N

Other weight related referrals: ☐ Y ☐ N

If yes, please describe:_____

Dietary Recommendations: ☐ Y ☐ N

Exercise Recommendations: ☐ Y ☐ N

Pharmacotherapy: ☐ Y ☐ N

If yes, what agent?_____

Pt. refuses/ not interested: ☐ Y ☐ N

Change of antipsychotic med.? ☐ Y ☐ N

Change dose of antipsychotic med.? ☐ Y ☐ N

ID Number_____

**BMI Survey- Data Extraction Tool**

## Psychiatry Visit(s):

**Visit Data:**

| | | |
|---|---|---|
| Waist size (in inches) documented: | ☐ Y ☐ N | If yes, waist size: ☐☐ |
| Weight documented: | ☐ Y ☐ N | |
| Height documented: | ☐ Y ☐ N | |
| BMI documented: | ☐ Y ☐ N | |
| Weight listed on problem list in chart? | ☐ Y ☐ N | |
| Weight listed in Past Medical History? | ☐ Y ☐ N | |
| Weight listed on Assessment/Plan? | ☐ Y ☐ N | |
| Weight loss recommended as therapy for diagnosis other than obesity? | ☐ Y ☐ N | |
| Weight linked to antipsychotics? | ☐ Y ☐ N | |

**Primary Care Action Plan included:**

Testing:

| | | | |
|---|---|---|---|
| Fasting glucose: | ☐ Y ☐ N | Referral to endocrinologist? | ☐ Y ☐ N |
| Thyroid hormone: | ☐ Y ☐ N | Referral to surgery? | ☐ Y ☐ N |
| UA: | ☐ Y ☐ N | Other weight related referrals: | ☐ Y ☐ N |
| Fasting lipids: | ☐ Y ☐ N | If yes, please describe:_____ | |
| HbAC: | ☐ Y ☐ N | Dietary Recommendations: | ☐ Y ☐ N |
| Other:_____ | | Exercise Recommendations: | ☐ Y ☐ N |
| Referral to nutrition/dietician: | ☐ Y ☐ N | Pharmacotherapy: | ☐ Y ☐ N |
| Referral to PT: | ☐ Y ☐ N | If yes, what agent?_____ | |
| Referral to Counseling/ Beh Mod/ MOVE | ☐ Y ☐ N | Pt. refuses/ not interested: | ☐ Y ☐ N |
| Referral to Social Work? | ☐ Y ☐ N | Change of antipsychotic med.? | ☐ Y ☐ N |
| | | Change dose of antipsychotic med.? | ☐ Y ☐ N |

ID Number_____

**)utcomes DATA:**

***** First Lab and last Lab within study period of 7/1/05-7/1/06***

| | | | |
|---|---|---|---|
| **nitial Wt:** | ☐☐☐ | **Final Wt:** | ☐☐☐ (Cover Sheet) |
| **;P:** | ☐☐☐/☐☐☐ | **BP:** | ☐☐☐/☐☐☐ (Cover S |
| **lgA1C:** | ☐☐.☐ | **HgA1C:** | ☐☐.☐ (Cumm. Labs) |
| **.DL:** | ☐☐☐ | **LDL:** | ☐☐☐ (Cumm. Labs) |
| **lDL:** | ☐☐☐ | **HDL:** | ☐☐☐ (Cumm. Labs) |
| **riglycerides:** | ☐☐☐☐ | **Triglycerides:** | ☐☐☐☐(Cumm. Labs) |
| **holesterol:** | ☐☐☐ | **Cholesterol:** | ☐☐☐ (Cumm. Labs) |

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