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Relationship between Insulin Resistance and C-Reactive Protein in a Patient Population Treated with Second Generation Antipsychotic Medications

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Abstract

C-reactive protein (CRP) is an inflammatory marker associated with obesity, insulin resistance, and cardiovascular disease. A recent study found CRP to be higher in individuals treated with certain antipsychotic medications such as olanzapine; however, it is unclear whether this is associated directly with drug intake or indirectly with drug-associated weight gain and insulin resistance. The objective of this study was to explore potential predictors of CRP including insulin resistance, components of the metabolic syndrome, psychiatric diagnosis, and antipsychotic medication in patients treated with antipsychotics. Sixty-four outpatients without diabetes being treated with a single second generation antipsychotic medication had direct measurements of insulin resistance at the end of a 180-minute infusion of glucose, insulin and octreotide (insulin suppression test) as well as components of the metabolic syndrome. Insulin resistance was the strongest predictor of CRP ($r=0.52$, $p<0.001$). When adjusted for insulin resistance, there was no significant relationship between CRP and any of the components of the metabolic syndrome criteria, specific drug treatment or psychiatric diagnoses. In conclusion, insulin resistance is strongly associated with CRP levels and likely contributes to previous associations between CRP and certain antipsychotic treatments.

Keywords

CRP; insulin resistance; second generation antipsychotics; olanzapine; metabolic syndrome; obesity

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Introduction

Treatment with second generation antipsychotic (SGA) medications is associated with development of metabolic complications, such as type 2 diabetes (Sernyak *et al*, 2002; Lamberti *et al*, 2004; Smith *et al*, 2008). As SGA treatment is also associated with weight gain, it has been difficult to delineate effects secondary to weight gain from direct drug effects. For example, olanzapine treatment may have both weight-dependent (Sowell *et al*, 2002; Sacher *et al*, 2008; Newcomer *et al*, 2009) and weight-independent (Newcomer *et al*, 2002; Henderson *et al*, 2005; Henderson *et al*, 2006; Kim *et al*, 2009) associations with insulin resistance. Olanzapine treatment has also been associated with increase in circulating markers of inflammation, including C-reactive protein (CRP), in the Clinical Antipsychotic Trials of Intervention Effectiveness (CATIE) (Meyer *et al*, 2009). Low-grade inflammation may underlie many disease processes including type 2 diabetes (Ndumele *et al*, 2006) and may provide a novel mechanism for SGA-associated metabolic complications. On the other hand, CRP can also increase with obesity-associated insulin resistance (McLaughlin *et al*, 2002; Abbasi *et al*, 2008) and may only be indirectly increased with SGA treatment.

To the best of our knowledge, we are not aware of any study that has evaluated the relationship between CRP and a direct measure of insulin resistance in a population of patients treated with SGAs. In CATIE, CRP was associated with meeting greater numbers of the metabolic syndrome criteria which includes five components associated with insulin resistance (Meyer *et al*, 2009). However, insulin resistance was not directly measured.

The purpose of this study, therefore, was to assess the relationship between CRP and insulin resistance as measured directly by the insulin suppression test in a population of patients treated with a single SGA. We also explored other variables associated with CRP such as components of the metabolic syndrome criteria, psychiatric diagnoses, and SGA treatment. We hypothesized that insulin resistance would be the major predictor of CRP.

Methods

Subjects

Subjects included 64 outpatients (36 male, 28 females) who were taking a single SGA for at least 3 months. A subset of the study population was recently included in a study evaluating the relationship between body mass index and insulin resistance in patients on SGA (Kim *et al*, 2009). SGAs included olanzapine (n=19), aripiprazole (n=19), risperidone (n=16), ziprasidone (n=6), clozapine (n=3) and quetiapine (n=1). Patients had to be on a stable antipsychotic regimen for three months preceding the study. The majority of the patients were taking SGAs for either schizophrenia/schizoaffective disorder (n=30) or bipolar disorder (n=27). Other diagnoses included depression (1), anxiety (3), posttraumatic stress disorder (2), and obsessive-compulsive disorder (1). No one was treated with a first generation antipsychotic medication. Individuals were in good general health without diabetes (fasting glucose <7mmol/L), anemia, or serious heart, liver or kidney disease.

The study was approved by the institutional review boards of Stanford University and the Palo Alto Veteran Administration Research and Development Committee. Subjects were

recruited through outpatient psychiatric clinics associated with Stanford University and Palo Alto Veteran Administration and through local providers and advertisements. Informed consent was obtained from all subjects, and the study was conducted in accordance with the Declaration of Helsinki.

Insulin Resistance

Insulin resistance was estimated by a modification (Pei *et al*, 1994) of the original insulin suppression test (Shen *et al*, 1970) which has a high correlation ($r = 0.93$) with the euglycemic clamp technique (Greenfield *et al*, 1981). After an overnight fast, an intravenous catheter was placed in each of the subject's arms. One arm was used for the administration of a 180-minute infusion of octreotide ($0.27 \text{ mcg/m}^2/\text{min}$), insulin ($32 \text{ mU/m}^2/\text{min}$) and glucose ($267 \text{ mg/m}^2/\text{min}$); the other arm was used for collecting blood samples. Octreotide is used to inhibit endogenous insulin production. Blood was drawn at baseline and at 10-minute intervals from 150 to 180 minutes of the infusion to determine the steady-state plasma glucose (SSPG) and insulin concentrations. Because steady-state insulin concentrations are similar between subjects, the SSPG concentration provides a direct measure of the ability of insulin to mediate disposal of an infused glucose load; therefore, the higher the SSPG concentration, the more insulin resistant the individual.

Metabolic Syndrome Criteria

The criteria for metabolic syndrome were derived from the recommendations of the National Cholesterol Education Program's Adult Treatment Panel III report (Grundy *et al*, 2005). To qualify for the metabolic syndrome, individuals needed to have 3 or more of the following 5 criteria: 1) waist circumference, $\geq 102 \text{ cm}$ in males and $\geq 88 \text{ cm}$ in females; 2) blood pressure $\geq 130/85 \text{ mmHg}$ or on an antihypertensive medication; 3) triglycerides $\geq 1.695 \text{ mmol/L}$; 4) high density lipoprotein cholesterol (HDL-C) concentration $< 1.036 \text{ mmol/L}$ in men and $< 1.295 \text{ mmol/L}$ in women; and 5) glucose $\geq 5.6 \text{ mmol/L}$.

Glucose concentrations were measured by the oxidase method (Beckman Analyzer 2, Brea, CA). Lipids were measured by the core laboratory at Stanford University Medical Center. Highly sensitive CRP was measured by Atherotech (Birmingham, AL) using latex-enhanced turbidimetric *in vitro* immunoassay with a lower level of detection of 0.05 mg/L .

Statistical Analysis

All statistical analysis was performed using SPSS (version 16 for Windows; SPSS, Chicago, IL). Descriptive data are presented as median and interquartile range unless otherwise specified. Potential predictors of CRP were evaluated using a univariate and multivariate analysis with SSPG in the model. The following variables were not normally distributed and log-transformed prior to analysis: triglyceride, HDL-C, SSPG, and CRP.

Results

Table 1 represents the demographic and metabolic characteristics of the study population. Two-thirds were obese ($\text{BMI} \geq 30 \text{ kg/m}^2$). Half (52%) met criteria for metabolic syndrome

with majority meeting criteria for high waist circumference (81%) and low HDL-C concentration (72%).

Univariate analyses were performed to evaluate the relationship between CRP and SSPG concentration and components of the metabolic syndrome criteria individually and by number of criteria met (Table 2). The strongest univariate predictor of CRP was SSPG concentration followed by waist circumference. Triglyceride concentration and number of metabolic syndrome criteria were weaker predictors of CRP. When SSPG concentration was included as a covariate, none of the other variables remained significantly associated with CRP ($p = 0.10$).

We also evaluated the relationship between CRP and psychiatric diagnoses or specific drug treatment in an ANCOVA adjusted for SSPG concentration. There was no significant difference in CRP levels between individuals with or without schizophrenia (natural log of CRP, mean $\pm$ SEM, 0.76 ± 0.18 vs. 0.81 ± 0.17 , $p=0.83$) or with or without bipolar disorder (0.75 ± 0.19 vs. 0.82 ± 0.16 , $p=0.78$). For drug treatment, we compared only individuals treated with olanzapine, risperidone, and aripiprazole as these drug groups had the most number of individuals. There was no significant difference in CRP levels among the three groups (olanzapine, 0.83 ± 0.23 ; risperidone, 0.93 ± 0.25 ; and aripiprazole 0.36 ± 0.22 , $p=0.18$).

As metabolic syndrome criteria are often used as a surrogate for insulin resistance, we evaluated the relationship between number of criteria met and SSPG concentration in Figure 1. Although there was a significant linear association between number of metabolic syndrome criteria and SSPG ($r=0.55$, $p<0.001$), there was also a tremendous degree of overlap in SSPG value in patients meeting from between 1 to 5 of the metabolic syndrome criteria. As the number of metabolic syndrome criteria increased, the SSPG concentration also increased; however, the magnitude of difference appeared greater between individuals who met no criteria and more than 3 compared with any difference among those individuals meeting 3, 4 or 5 criteria.

Discussion

In a diverse population of psychiatric patients treated with a single SGA, we show that insulin resistance is the strongest predictor of CRP among the variables examined. In addition, insulin resistance may explain a significant amount of the variance in the relationship between CRP and other presumed predictors such as obesity and metabolic syndrome components in patients treated with SGAs.

In CATIE, patients treated with olanzapine for 3-months had the greatest increase in CRP compared with three other SGAs and perphenazine (Meyer *et al*, 2009). Olanzapine treatment was also associated with the greatest increase in weight gain (Lieberman *et al*, 2005), but change in BMI accounted for less than 1% of the change in CRP. The authors postulated that certain antipsychotics such as olanzapine may have direct effects on inflammation. However, insulin resistance was not measured, and olanzapine treatment can have both weight-dependent (Sowell *et al*, 2002; Sacher *et al*, 2008; Newcomer *et al*, 2009)

and weight-independent (Newcomer *et al*, 2002; Henderson *et al*, 2005; Henderson *et al*, 2006; Kim *et al*, 2009) associations with insulin resistance. Thus, it is also possible that change in insulin resistance was also contributing to the difference in CRP concentration among drug groups in CATIE.

Although no previous study has evaluated the relationship between CRP and a direct measure of insulin resistance, there have been a few studies in patients treated with SGAs that have measured CRP concentrations and an indirect measure of insulin resistance, the homeostasis model assessment of insulin resistance (HOMA-IR). HOMA-IR is a calculation based on fasting glucose and insulin concentrations (Matthews *et al*, 1985). Although it correlates with direct measures of insulin resistance, HOMA-IR accounts for less than 40% of the variability in insulin resistance (Kim *et al*, 2004), and therefore is a less precise measure of insulin action than SSPG. Possibly a reflection of the increased variability of this measure, the two studies measuring CRP and HOMA-IR in patients switched to olanzapine are inconsistent. Thus, one study showed a significant increase in both HOMA-IR and CRP concentrations after switching to olanzapine therapy in 60 patients with schizophrenia (Baptista *et al*, 2007a) while another in 37 patients with schizophrenia showed a decrease in HOMA-IR and no significant change in CRP levels (Baptista *et al*, 2007b). Another cross-sectional study reported no association between CRP concentrations and HOMA-IR in patients treated with clozapine, olanzapine, and first generation antipsychotics and in relatives of patients taking clozapine and olanzapine (Carrizo *et al*, 2008). The differences among these results and with ours may be secondary to limitations of HOMA-IR as a surrogate of insulin resistance and/or to differences among the study populations. For example, while Carrizo *et al*. found no association between CRP and HOMA-IR, CRP was positively associated with BMI and waist circumference—variables that normally increase with measures of insulin resistance (Farin *et al*, 2006). In addition, the patients treated with first generation antipsychotics had the highest CRP concentration among all the drug and nondrug groups, but they were also the leanest and had the lowest prevalence of the metabolic syndrome. This is contrary to the positive association seen between CRP and BMI in their study and contrary to the positive association seen between CRP and metabolic syndrome in our study and others (Ridker *et al*, 2003; Meyer *et al*, 2009). Despite these differences, one consistent finding among all the studies is that the change in CRP concentration seem unrelated to the type of antipsychotic treatment.

Although insulin resistance was the strongest predictor of CRP in our study, our results also show that it explains only a quarter of the variance in CRP. This degree of association is similar to results recently reported in the general population when insulin resistance was measured using the euglycemic clamp ($r^2=0.23$) (Bluher *et al*, 2005). That insulin resistance does not account for the majority of the variance in CRP should not be surprising given that CRP is a nonspecific marker of inflammation and also varies with infection and other inflammatory processes (Du Clos *et al*, 2004). CRP has also been associated with various psychiatric phenotypes (Fan *et al*, 2007; Akanji *et al*, 2009). However, as insulin resistance was not assessed, these psychiatric illness associations may be mediated by differences in insulin action rather than specific disease presentations. Therefore, caution should be taken to associate CRP with clinical variables when insulin resistance has not been evaluated.

On this note, metabolic syndrome criteria are often used as a surrogate of insulin resistance. However, as seen in Figure 1, there can be great overlap in degree of insulin resistance between individuals who meet varying numbers of the criteria. This likely explains the weaker correlation between CRP and number of metabolic syndrome criteria ($r=0.25$) compared with SSPG ($r=0.52$). CATIE reported almost identical relationship between number of metabolic syndrome criteria and CRP ($r=0.27$) in their population. Therefore, metabolic syndrome criteria have some limitations as a surrogate measure of insulin resistance.

A major limitation of this study is that the study population is relatively small; therefore smaller magnitude associations may not be detected. In addition, as it was a cross-sectional study, only associations and not causations can be shown. However, this is the first study to report on the relationship between CRP and insulin resistance using a direct measure of insulin action in a population of diverse patients treated with SGAs. In so doing, we find that insulin resistance is the major predictor of CRP among the variables evaluated and may explain some of the variance in CRP concentration in individuals treated with antipsychotics with different metabolic risks.

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Fig. 1.

There is a moderate positive relationship between number of metabolic syndrome criteria and a specific measure of insulin resistance (SSPG concentration, $r=0.55$, $p<0.001$); however, there is great overlap in SSPG values in patients meeting from between 1 to 5 of the metabolic syndrome criteria. The horizontal line across the box represents the median and the boundaries of the box represent the lower and upper quartiles.

Table 1

Demographic and metabolic characteristics of the SGA population

	Median	Interquartile Range
N	64	--
Age (years)	49	(40, 54)
BMI (kg/m ²)	33	(28, 36)
Waist Circumference (cm)	112	(97, 117)
Systolic Blood Pressure (mmHg)	117	(113, 132)
Diastolic Blood Pressure (mmHg)	73	(67, 77)
Fasting Glucose (mmol/L)	5.3	(5.1, 5.7)
Triglyceride (mmol/L)	1.4	(0.9, 2.3)
HDL-C (mmol/L)	0.9	(0.8, 1.2)
SSPG (mmol/L)	9.9	(6.2, 13.4)
CRP (mg/L)	2.3	(1.1, 5.2)
	N	%
Male	36	59
NonHispanic White	46	72
Current smoker	34	53
BMI ≥ 30 kg/m ²	43	67
Metabolic Syndrome	33	52
No. of Metabolic Syndrome Criteria		
0	3	5
1	9	14
2	19	30
3	16	25
4	12	19
5	5	8
Criteria Prevalence		
Waist Circumference	52	81
Blood Pressure	23	36
Glucose	23	36
Triglyceride	24	38
HDL-C	46	72

SSPG, steady-state plasma glucose, high values represent greater degrees of insulin resistance; HDL-C, high density lipoprotein cholesterol ; CRP, C-reactive protein

Table 2

Predictors of CRP

Variable	r	P
SSPG	0.52	<0.001
Waist Circumference	0.45	<0.001
Systolic Blood Pressure	0.17	0.18
Diastolic Blood Pressure	0.03	0.82
Fasting Glucose	0.12	0.33
Triglyceride	0.25	0.05
HDL-C	-0.12	0.35
No. of Metabolic Syndrome criteria	0.25	0.046

SSPG, steady-state plasma glucose, high values represent greater degrees of insulin resistance; HDL-C, high density lipoprotein cholesterol