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Social functioning and age across affective and non-affective psychoses

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Abstract

Both non-affective and affective psychoses are associated with deficits in social functioning across the course of the illness. However, it is not clear how social functioning varies among diagnostic groups as a function of age. The current study examined the relationship between social functioning and age in schizophrenia (SZ), schizoaffective disorder (SZA), and psychotic bipolar disorder (PBD). We found that individuals with PBD had the highest functioning while individuals with SZ had the poorest. The functioning of individuals with SZA fell in between the other groups. We also found that older ages were associated with poorer functioning. Although there was not a significant diagnostic group by age interaction, visual inspection of our data suggests a subtly steeper trajectory of decline in PBD. These results indicate that a decline in social functioning with may be an important area of unmet need in treatment across psychotic disorders.

Keywords

schizophrenia; schizoaffective disorder; bipolar disorder; social functioning; MCAS

1. Introduction

Non-affective psychosis, such as schizophrenia (SZ), is associated with social dysfunction (e.g., Blanchard et al, 1998; Strauss et al, 1974), including less social interest, fewer social activities, and less pleasure obtained from social interactions than healthy individuals (e.g., Chapman et al, 1976; Meehl, 1990). Social dysfunction is not only present in acute stages of the illness but also in the premorbid, prodromal, and residual stages (e.g., Comparelli et al, 2012; Haas et al, 1992; Kwapil, 1998; Perlick et al, 1992). This dysfunction is associated with other poor outcomes, including decreased quality of life, poorer community and

occupational functioning, and greater chronicity (e.g., Hoe et al, 2012; Penn et al, 1997; Perlick et al, 1992; Strous et al, 2004).

While affective psychoses, such as bipolar disorder with psychotic features (PBD), were generally considered to have better outcomes than non-affective psychosis (e.g., Marneros et al, 1990; McGlashan, 1984), evidence suggests that as few as one third of patients with bipolar disorder achieve functional recovery over time (Huxley et al, 2007). At least partial disability is reported in approximately 80% of patients with PBD; as many as 65% of patients reported being unemployed even after clinical recovery, and patients experience significant disability in daily living and social functioning (Goldstein et al, 2009; Hua et al, 2011; Rosen et al, 1983; Sanchez-Moreno et al, 2009). Similar to non-affective psychosis, social dysfunction in BD is present during periods of both symptom exacerbation and euthymia (e.g., Sanchez-Moreno et al, 2009), with up to 65% of individuals failing to obtain pre-morbid levels of social functioning after disease onset (Strakowski et al, 1998; Tohen et al, 2000).

Also similar to non-affective psychosis, social deficits in PBD are associated with other poor outcomes generally, including lower quality of life, poorer occupational functioning, and greater chronicity (Judd et al, 2002). Variables such as poor cognitive functioning predict poor community outcomes, including social dysfunction, in patients with BD as they do in SZ and related disorders (e.g., Barch, 2009; Donohoe et al, 2012; Green, 2006; Lewandowski et al, 2011a; Lewandowski et al, 2011b). The course of cognitive dysfunction appears to differ between patients with SZ and PBD (Lewandowski et al, 2011b). Individuals who go on to develop SZ tend to exhibit cognitive difficulties throughout the premorbid period with worsening at their first break followed by a more stable pattern of deficits over the course of the illness. In contrast, individuals with bipolar disorder tend to show typical premorbid cognitive development with evidence of cognitive dysfunction at onset followed by deficits associated with symptom exacerbations. Because cognitive functioning is strongly associated with community functioning, difference in the progression of cognitive deficits between diagnoses suggest that the course of community dysfunction may differ between these groups as well.

Although both non-affective and affective psychoses are associated with social deficits, it is not clear whether the groups differ in levels of social functioning. While some have suggested that social functioning is similarly poor between individuals with non-affective or with affective psychosis (e.g., Bellack et al, 1989; Simonsen et al, 2010), others have reported that those with affective psychosis have significantly better social functioning (e.g., Tarbox et al, 2012). Given that older ages are associated with greater social dysfunction in both SZ (Gould et al, 2012; Mueser et al, 2010) and bipolar disorder with and without psychotic features (Depp et al, 2007; Mueser et al, 2010), one possible reason for discrepant findings is differing age ranges of participants across investigations. Thus, the inclusion of a larger age range in a single study, specifically including participants with older ages (> 50 years), may help to clarify the relationship between social functioning and age across diagnostic categories.

Few studies have examined social functioning in patients with both prominent psychotic and affective features, such as patients with schizoaffective disorder (SZA). This disorder, which is characterized by both primary psychosis and prominent mood symptoms (American Psychiatric Association, 2000), is often considered together with SZ in investigations of social functioning (e.g., Corrigan et al, 1995; Lysaker et al, 2004; Pinkham et al, 2006; Roncone et al, 2002); however no studies have explicitly confirmed that patients with SZ and SZA experience the same degree of social dysfunction across the lifespan. Characterizing social functioning deficits across psychotic disorders may facilitate efforts to identify and remediate these deficits across diagnostic groups.

In the current study we aimed to clarify social functioning levels between patient groups and across the adult lifespan in a large, cross diagnostic sample of patients with psychosis ranging in age from 18–70 years. Based on previous findings we hypothesized that individuals with SZ would have the poorest social functioning while individuals with PBD would have the highest social functioning, with the social functioning of individuals with SZA falling between these groups. In addition, based on previous findings of regarding the course of cognitive dysfunction between the groups (i.e., greater differences in cognitive dysfunction between the groups at earlier ages and more similar levels of cognitive dysfunction at later ages), we also examined whether there was an interaction between age and diagnosis predicting social functioning. Specifically, we examined if as age increased, the social functioning of the PBD group would decline more rapidly and become more similar to the SZ group.

2. Methods

2.1. Participants

Patients with diagnoses of SZ (n=136), SZA (n=139), or PBD (n= 204) between the ages of 18 and 70 were included in this study. Subjects were originally recruited for a genetic association study of mood and psychotic disorders, as inpatients from an inpatient unit specializing in psychotic disorders (n = 332) or as outpatients through referrals or fliers posted at the hospital (n = 147). Patients were not eligible to participate if they had a history of head trauma with loss of consciousness, diagnosis of a developmental disorder, or psychosis secondary to a medical illness.

All procedures were approved by the hospital Institutional Review Board. Written informed consent was obtained from all participants after the study procedures were fully explained.

2.2. Materials

The Structured Interview for DSM-IV (SCID; First et al, 1998) was administered by trained clinicians who met routinely for reliability exercises and to discuss difficult cases and arrive at a consensus diagnosis. Rates of agreement were perfect (1.0) for SCID diagnoses (Ongur et al, 2009).

Social functioning was assessed using the Multnomah Community Ability Scale (MCAS; Barker et al, 1994). The MCAS assesses functioning in several domains including social interest, support, and activities, independence in daily living, and occupational or other

meaningful activity. We administered an abbreviated version of this assessment, eliminating items that directly assessed clinical symptoms (psychosis, M3; mood abnormality, M4; impulse control, M17), substance abuse (M16), intellectual functioning (M2), and medication compliance (M14) so as to assess functioning in a way that was less directly associated with clinical symptoms and cognition. Considering the previous month only, MCAS ratings were made following the SCID interview.

Clinical symptoms were assessed using the Positive and Negative Syndrome Scale (PANSS, Kay et al, 1987), which includes subscales for positive symptoms (e.g., hallucinations, delusions), negative symptoms (e.g., blunted affect, emotional withdrawal), and general psychopathology (e.g., somatic concerns, anxiety). Considering the previous month only, PANSS ratings were made following the SCID interview.

Information about medications at time of assessment was obtained from the discharge medication list (inpatients) or by patient report (outpatients). For participants with complete data, chlorpromazine (CPZ) equivalent dose was calculated; some data (e.g. dosage information) were missing in 9 subjects. For typical antipsychotics, we calculated CPZ equivalents based on the Schizophrenia Patient Outcomes Research Team recommendations (Lehman et al, 1998). For most atypical antipsychotics, we used the CPZ equivalent doses published by Woods (2003). For risperidone and paliperidone, we used guidelines by Gardner et al. (2010).

2.3. Procedure

2.3.1. Data Reduction—Because our primary interest in the current study was differences in social functioning (e.g., social interest, activities, support) between the groups and the total MCAS score tends to mask item variability (Hendryx et al, 2001), we performed a factor analysis on the MCAS data to determine whether there was a "social functioning" factor. To determine the appropriate number of factors to retain, we used Horn's Parallel Analysis (Horn, 1965), using the "paran" function (Dinno, 2009) in R (Ihaka et al, 1996). Five factors emerged from this analysis. The first factor was a social functioning factor, which accounted for 20% of the variance.[1] Three items had factor loadings of .4 or greater: Social Interest (.80; MCAS10; initiation of social contact, responsiveness to others' initiation), Social Network (.76; MCAS12; extensiveness of social support), and Meaningful Activity (.74; MCAS13; involvement in activities that include others). Thus, we created a composite social functioning score by adding the standardized scores on these three items. Because the MCAS is designed to assess functioning in patient populations, we would expect healthy adults to score at or near 15 on social functioning.

2.3.2. Statistical Approach—Demographic information and symptom ratings of the groups were compared using ANOVAs. When significant differences were detected among the three patient groups, pairwise comparisons between the groups were conducted using

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[1]Acceptance of Illness (MCAS8) and Cooperation with Treatment Providers (MCAS15) loaded on the second factor. Acceptance of Illness (MCAS 8) also loaded on the third factor along with Ability to Manage Monday (MCAS6) and Independence of Daily Living (MCAS7). Social acceptability (MCAS9) loaded on the fourth factor, and Independence of Daily Living (MCAS7) and Physical Health (MCAS1) loaded on the fifth factor.

Tukey's Honestly Significant Difference tests. A linear regression was conducted examining the effects of diagnosis and age on both social functioning (composite variable created from the MCAS) and community functioning (variable based on total MCAS score) after controlling for demographic variables (sex, race) and CPZ equivalents that significantly differed among the groups.

3. Results

3.1. Group differences in demographic information

First, we examined diagnostic group differences in demographic information (Table 1). The groups did not significantly differ in level of education, level of parental education, or inpatient status at time of testing, all ps >.32. Although approaching significance, the groups did not differ in reports of lifetime hospitalizations, χ^2 (4, N = 263) = 7.67, p = .1.[2] Groups differed significantly by sex, χ^2 (2, N = 479) = 9.78, p < .01, and race (Caucasian vs. non-Caucasian), χ^2 (2, N = 479) = 12.43, p < .01. Specifically, there were fewer women and fewer Caucasian individuals in the SZ group than the other groups.

The groups significantly differed in age, F(2, 476) = 4.80, p < .01. The SZA group was significantly older than the PBD group, p < .01, but the SZ did not differ from either the SZA or PBD groups, both ps > .2. Also, the groups significantly differed in duration of illness [DOI], F(2, 389) = 4.17, p < .05.[3] The SZA group had a significantly longer DOI than the PBD group, p < .05, but the SZ group did not differ from either the SZA or PBP groups in DOI, both ps > .2. Age and DOI were highly correlated, r = .75, p < .001. The groups significantly differed in CPZ equivalents, F(2, 476) = 9.79, p < .001. Although the SZ and SZA groups did not differ from each other, both were significantly greater than the PBD group, both ps < .01 (SZ, SZA > PBD).

3.2. Group differences in clinical symptoms

Clinical symptoms by diagnosis are presented in Table 1. Diagnostic groups did not differ on the positive or general subscale of the PANSS, both ps > .51; however, the groups significantly differed on the negative subscale, F(33) = 1.63, p < .05. Follow-up comparisons revealed that the SZ group had significantly higher scores on the negative subscale compared to both the SZA and PBD groups, and the SZA group had significantly higher scores compared to the PBD group, all ps < .001 (SZ > SZA > PBD). However, the negative subscale of the PANSS captures, among other types of negative symptoms, interest and initiation of social activities. Because these symptoms are closely related to our construct of interest, we did not statistically control for scores on the PANSS negative subscale in the regression analyses.

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[2]The number of lifetime hospitalizations was not collected for 68 individuals in the SZ group, 57 individuals in the SZA group, and 99 individuals in the PBP group.
[3]Duration of illness information was not collected for 20 individuals in the SZ group, 34 individuals in the SZA group, and 33 individuals in the PBP group.

3.3. Diagnosis, age, and social functioning

For the regression analyses, the SZ group was used as the reference group. Due to the substantial amount of missing data regarding number of lifetime hospitalizations (> 41% from each group), this variable was not included in the regression analysis. In addition, given the strong correlation between age and DOI, DOI was not included in the model in order to avoid multicollinearity. Because CPZ equivalents were not normally distributed (kurtosis = 3.96, skewness = 1.7), we used a log transformation of CPZ equivalents in the regression analyses.

We conducted a multiple regression with group, age, race, sex, and log CPZ equivalents, as well as the interaction between group and age, predicting the MCAS composite social functioning score. The interaction was not significant, $F(2, 472) = 0.083$, $p = .92$, $\eta_p^2 = .0004$. Thus, we removed the interaction term from the model and re-ran the analysis. As can been seen in Table 2, both group (PBD, $p < .001$; SZA, $p < .05$) and age ($p < .01$) were significant predictors of social functioning. As can be seen in Figure 1, social functioning differed among the diagnostic groups (PBD > SZA > SZ). As also can be seen in Figure 1, there was no statistically significant diagnosis X age interaction.

Also, race was predictive of social functioning, $p < .01$. That is, non-Caucasian status predicted worse social functioning. There was a trend for log CPZ equivalents to significantly predict social functioning ($p = .053$), and sex was not a significant predictor of social functioning ($p = .17$).

3.4. Diagnosis, age, and community functioning

We also conducted a multiple regression with group, age, race, sex, and log CPZ equivalents, as well as the interaction between group and age, predicting the total MCAS score. The interaction was not significant, $F(2, 472) = 0.044$, $p = .96$, $\eta_p^2 = .0002$. Thus, we removed the interaction term from the model and re-ran the analysis. As can been seen in Table 3, both group (PBD, $p < .001$; SZA, $p < .05$) and age ($p < .01$) were significant predictors of the MCAS total score. Also, CPZ equivalents were predictive of the MCAS total score, $p < .01$. That is, higher levels of CPZ predicted worse community functioning. There was a trend for race to significantly predict MCAS total score ($p = .092$), and sex was not a significant predictor of community functioning ($p = .35$).

4. Discussion

In the current study, we examined social functioning across three diagnostic groups (SZ, SZA, and PBD) using a clinician-rated measure. We included a large age range, specifically including participants with older ages (> 50 years), to help clarify the relationship between social functioning and age across these groups. We found that groups differed by diagnosis across the lifespan. Specifically, individuals with SZ had the poorest functioning while individuals with PBD had the highest functioning. The social functioning of individuals with SZA fell in between the other two groups. We also found a significant effect of age, with older ages associated with worse social functioning across all groups.

Although the PBD group had better social functioning than the SZ and SZA groups at age 18, all three patient groups exhibited social impairment compared to what would be expected in a healthy population. This suggests that – like patients with SZ and SZA – patients with PBD are already impaired in social functioning at an early stage of illness. Extensive work has been conducted with individuals at risk for the development of schizophrenia-spectrum disorders (for a review, see Correll et al, 2010), and recent work has focused on defining a bipolar risk syndrome (Bauer et al, 2008; Conus et al, 2008; Hauser et al, 2007; Ozgürdal et al, 2009). The current finding of a mild degree of impairment in social functioning at the earlier stages of PBD supports its inclusion in the conceptualization of a bipolar risk syndrome.

Although individuals with SZA are often grouped with individuals with SZ, the current study suggests that individuals with SZA may be less impaired than individuals with SZ. It may benefit investigators who include individuals diagnosed with SZ and SZA in a single study to be aware of social functioning differences. This may be especially important in intervention studies focused on amelioration of social deficits to consider that the baseline level of functioning between these groups differs.

Our findings provide evidence that increasing age is associated with poorer social functioning across diagnoses; however, we did not find a significant age by diagnosis interaction, suggesting that social functioning was associated with age in a similar way across diagnoses (Figure 1). While the interaction term was non-significant, visual inspection of our data suggests a subtly steeper trajectory of decline in patients with PBD. Differences in social functioning between diagnoses may mirror previously reported differences in trajectories of cognitive functioning over the course of illness and with repeated exacerbations, although examination of this relationship is beyond the scope of this work. In any event, decline in social functioning with age in both affective and non-affective psychoses suggests that this may be an important area of unmet need in treatment across psychotic disorders.

Although our sample was large with over 135 people in each of the three diagnostic groups, the current study utilized cross-sectional data. Thus, it is possible that individuals who are older but higher functioning are not in treatment and therefore not captured in the current study. However, any individual who is higher functioning, independent of diagnostic group, may be equally as likely to drop out of treatment making this an unlikely explanation for the current findings. In addition, the current study included both individuals who had been recently hospitalized and those who had not. It is possible that psychotic symptom exacerbation in those who had been recently hospitalized may obscure a more accurate reflection of social functioning within a diagnostic category. However, because the number of inpatients vs. outpatients did not differ between groups, we do not believe this affected the primary findings in the current study. Also, because the current study focused on social functioning, it did not include any neurocognitive measures. In schizophrenia, some research suggests neurocognitive factors are predictive of one's ability to socially problem solve in a laboratory setting (Addington et al, 2000). In addition, it has been reported that social cognition, or "the mental operations underlying social interactions" (Schmidt et al, 2011, p. S41), actually mediates the relationship between neurocognition and social functioning

(Horan et al, 2012). Thus, future research may include neurocognitive and social cognitive measures to examine these relationships across non-affective and affective psychoses.

With the vast majority of participants in the current study taking medications, we found that the SZ and SZA groups were prescribed significantly more antipsychotic medication than the PBD group. We also found a trend for CPZ equivalents to predict social functioning. These medications have been found to have multiple effects on brain activity. For example, some have found a relationship between antipsychotic mediation usage and ventral striatum activity (e.g., Juckel et al, 2006; Kirsch et al, 2007; Schlagenhauf et al, 2008), an area of the brain associated with reward processing. If reward processing is impacted by medication use, people may be less likely to find social situations rewarding. Subsequently, they may be less likely to engage in such situations, leading to a decreased in social functioning. A recent report of patients randomized to either an antipsychotic dose-reduction treatment condition or maintenance treatment condition found that patients in the dose reduction group experienced greater functional recovery over 7 years, suggesting that antipsychotic load may indeed adversely affect community functioning. (Wunderink et al, 2013). Of note, increasing antipsychotic medication dosage has not been consistently associated with poor community functioning, particularly in the short-term (Rosen et al, 1981).The effect of medication usage can be addressed directly in future work by investigating social functioning in unmedicated individuals at their first psychotic break who are followed over time.

6. Conclusions

The current study is consistent with previous work that has reported deficits in social functioning in both non-affective and affective psychoses. This study has extended previous work by clarifying the level of social functioning across the life span in these disorders. Results indicated that through the life span, individuals with SZ had the poorest functioning followed by individuals with SZA and individuals with PBD.

Acknowledgments

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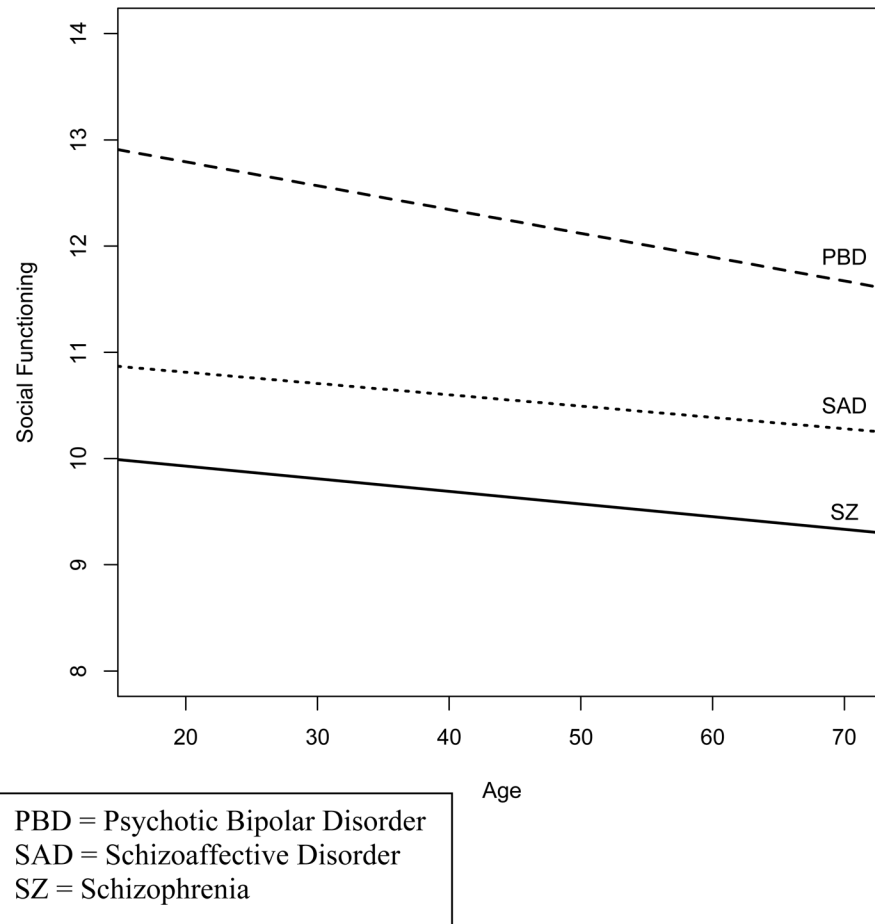
Figure 1.
The relationship between social functioning and age by diagnosis.

Table 1

Demographic information by diagnosis

	SZ (n = 136)	SZA (n = 139)	PBD (n = 204)
Age	38.84 (13.5)	39.60 (12.72)	35.24 (12.44)
Range	18–66	18–64	18–70
% Female	33.8	50.1	50
% Caucasian	65.4	82.0	79.4
Education[a]	4.19 (1.42)	4.54 (1.59)	4.87 (1.53)
Parental education[a]	5.06 (1.46)	5.29 (1.31)	5.33 (1.24)
Duration of illness	14.81 (13.45)	17.61 (12.01)	13.29 (11.10)
Lifetime hospitalizations			
% with 0 – 1 hospitalizations	22.06	12.08	25
% with 2 – 4 hospitalizations	35.29	30.12	33.92
% with 5 or more hospitalization	42.65	57.8	41.08
% Inpatient	74.3	68.3	66.7
% Taking antipsychotics	84.6	79.1	75.5
Chlorpromazine equivalents	485.19 (416.65)	436.97 (430.31)	307.97 (324.89)
Range	0–1800	0–2413	0–1920
% taking mood stabilizers	17.6	51.8	82.8
% taking antidepressants	33.8	47.5	19.1
% taking benzodiazepines	33.1	48.2	40.7
PANSS Positive	20.72 (5.66)	20.27 (7.07)	18.89 (8.35)
PANSS Negative	18.19 (8.28)	14.91 (6.19)	10.26 (3.77)
PANSS General	34.60 (10.28)	34.63 (9.36)	31.81 (10.47)
MCAS total score	39.97 (7.93)	42.22 (7.14)	45.75 (6.44)
Range	18–55	25–54	29–55
MCAS social functioning score	9.73 (3.13)	10.6 (2.8)	12.45 (2.53)
Range	3–15	5–15	5–15

Note:

[a] Education is coded based on the SCID Education and Work History scale: 1=grade 6 or less; 2=grade 7–12 (without graduating); 3=high school grad or equivalent; 4=part college; 5=graduated 2 year college; 6=graduated 4 year college; 7=part graduate/professional school; 8=completed graduate/professional school;

[b] Percentage do not add to 100% due to reports of being in no treatment or "other programs"; PANSS = Positive and Negative Syndrome Scale; MCAS total max possible = 55; MCAS social functioning score max possible = 15

Table 2

Summary of Multiple Regression Analysis for Social Functioning (N = 479)

	B	SE(B)	β	t	p
Intercept	−.43	.16		−2.73	**.007**
Diagnosis					
SZ	(reference)				
PBD	.72	.09	.39	7.69	**<.001**
SZA	.20	.10	.10	2.02	**.04**
Age	−.007	.003	−.09	−2.17	**.03**
Race					
Caucasian	(reference)				
Non-Caucasian	.29	.09	.14	3.28	**.001**
Sex					
Male	(reference)				
Female	.11	.08	.06	1.36	.17
CPZ equivalents	−.09	.05	−.08	−1.94	.053

Note: $F_{(6, 472)} = 17.97$, $p < .001$; $R^2 = 0.1756$

Table 3

Summary of Multiple Regression Analysis for MCAS total (N = 479)

	B	SE(B)	β	t	p
Intercept	–.14	.18		–0.76	.44
Diagnosis					
SZ	(reference)				
PBD	.68	.11	.33	6.31	**<.001**
SZA	.28	.11	.12	2.38	**.02**
Age	–.01	.003	–.14	–3.20	**.001**
Race					
Caucasian	(reference)				
Non-Caucasian	.17	.10	.07	1.69	.09
Sex					
Male	(reference)				
Female	.09	.09	.04	0.95	.34
CPZ equivalents	–.17	.05	–.13	–3.10	**.002**

Note: $F(6, 472) = 13.51$, $p < .001$; $R^2 = 0.1357$