Journal of the International Neuropsychological Society (2015), **21**, 468–472.

doi:10.1017/S1355617715000442

BRIEF COMMUNICATION

Measuring Cognition in Bipolar Disorder with Psychosis Using the MATRICS Consensus Cognitive Battery

Sarah H. Sperry,[1,2] Lauren K. O'Connor,[1] Dost Öngür,[1,3] Bruce M. Cohen,[1,3] Matcheri S. Keshavan,[3,4] AND Kathryn E. Lewandowski[1,3]

[1]Schizophrenia and Bipolar Disorder Program, McLean Hospital, Belmont, Massachusetts
[2]University of North Carolina at Greensboro, Greensboro, North Carolina
[3]Harvard Medical School, Boston, Massachusetts
[4]Beth Israel Deaconess Hospital, Boston, Massachusetts

(RECEIVED September 8, 2014; FINAL REVISION April 17, 2015; ACCEPTED May 11, 2015; FIRST PUBLISHED ONLINE July 6, 2015)

Abstract

Given the substantial overlap in cognitive dysfunction between bipolar disorder (BD) and schizophrenia (SZ), we examined the utility of the MATRICS Consensus Cognitive Battery (MCCB)—developed for use in SZ—for the measurement of cognition in patients with BD with psychosis (BDP) and its association with community functioning. The MCCB, Multnomah Community Ability Scale, and measures of clinical symptoms were administered to participants with BDP ($n = 56$), SZ ($n = 37$), and healthy controls (HC) ($n = 57$). Groups were compared on clinical and cognitive measures; linear regressions examined associations between MCCB and community functioning. BDP and SZ groups performed significantly worse than HC on most neurocognitive domains; BDP and HC did not differ on Social Cognition. Patients with BDP performed better than patients with SZ on most cognitive measures, although groups only differed on social cognition, working memory, verbal memory, and the composite after controlling for clinical variables. MCCB was not associated with community functioning. The MCCB is an appropriate measure of neurocognition in BDP but does not appear to capture social cognitive deficits in this population. The addition of appropriate social cognitive measures is recommended. (*JINS*, 2015, *21*, 468–472)

Keywords: Bipolar disorder, Cognition, Neuropsychology, Schizophrenia, Psychometrics, Psychosocial functioning

INTRODUCTION

Cognitive dysfunction is increasingly recognized as a key feature of bipolar disorder (BD). Neurocognitive deficits persist across illness stages including euthymia, with patients scoring an average of 2/3 to 1 *SD* below the mean (Robinson et al., 2006), and may be more severe in patients with BD with a history of psychosis (BDP) (Lewandowski, Cohen, Keshavan, & Ongür, 2011). Cognitive dysfunction in BDP appears to fall between the level of healthy controls and patients with schizophrenia (SZ) (Hill et al., 2013), although not all studies report differences between patient groups (Lewandowski et al., 2011). Cognitive deficits are associated with functional outcomes in both SZ and BD (Green, 2006) and are among the strongest predictors of future functioning (Lewandowski, Cohen, Keshavan, Sperry, & Ongür, 2013).

Patients with BD appear to exhibit deficits in social cognition, although findings in this domain are mixed. Social cognition is a multidimensional construct that is comprised of many aspects of social processing, and patients with BD may exhibit selective deficits in certain domains but not others. For instance, while patients with BD show deficits in emotion processing and Theory of Mind, they may not show broad deficits across all dimensions of social cognition (Samamé, Martino, & Strejilevich, 2012). Deficits in social cognition may be especially predictive of community outcomes in patients with SZ (Couture, Penn, & Roberts, 2006); the association between social cognition and community outcomes in BD is less clear (Van Rheenen & Rossell, 2014).

Given that cognitive deficits are numerous, severe and functionally impairing across the psychoses, development of assessments to measure key cognitive domains in patients with BD is essential for proper characterization of the nature

Correspondence and reprint requests to: Sarah H. Sperry, Department of Psychology, UNC-Greensboro, P.O. Box 26170, Greensboro, NC 27402-6170. E-mail: shsperry@uncg.edu

and course of cognitive dysfunction and the development of targeted treatments and monitoring of therapeutic success. The Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS) Initiative set forth to do just this through the development of a consensus cognitive battery (MATRICS Consensus Cognitive Battery; MCCB) assembled to assess key cognitive domains in SZ (Marder & Fenton, 2004). Despite growing evidence of significant and functionally relevant neurocognitive impairment in patients with BD, no such battery has been created for use with this population and there is a lack of consensus regarding appropriate neurocognitive assessment in terms of both domains to be assessed and measures to be used. Given evidence of substantial overlap in cognitive dysfunction between SZ and BD, particularly BDP, the MCCB may be appropriate for the assessment of cognition in this population.

The International Society for Bipolar Disorders (ISBD) conducted a meta-analysis of individual measures included in the MCCB and endorsed its use in patients with BD for assessment of most cognitive domains excluding reasoning/problem solving and social cognition (Yatham et al., 2010). Indeed, Burdick et al. (2011) found that both euthymic and symptomatic outpatients with BD performed worse than healthy controls on all MCCB domains except reasoning/problem solving and social cognition. Van Rheenen and Rossell (2013a) reported that patients only performed worse than controls on processing speed, visual learning, and working memory; however, their sample was heterogeneous, including both patients with BDI and BDII, and they did not report potentially relevant factors such as history of psychosis. To our knowledge, no studies to date have examined the relationship of MCCB with community functioning in patients with BD.

We aimed to investigate the sensitivity of the MATRICS Consensus Cognitive Battery in detecting cognitive deficits in patients with BDP. Here we present the first study to directly compare MCCB scores in BDP to both healthy participants and participants with SZ. We aimed to increase the homogeneity of our BD sample by including only those subjects with a history of psychosis, a feature that has been associated with greater illness burden and poorer cognitive functioning in previous studies. We hypothesized that (1) patients would perform worse than controls on the MCCB but better than patients with SZ, and (2) MCCB scores would be associated with community functioning in both patient groups.

METHOD

Participants

Participants were recruited through the Schizophrenia and Bipolar Disorder Program (SBDP) at McLean Hospital and via fliers posted around the hospital. Participants were recruited in the context of several separate but related studies: patients with BDP were recruited for a study of cognitive remediation, patients with SZ were recruited for a study of genotype and phenotype in psychosis or for a study of cognitive remediation, and HC were recruited through Craigslist or a study investigating brain metabolism. All procedures were approved by the McLean Hospital IRB. Participants were outpatients at the time of testing. Exclusion criteria for all participants included history of head trauma with loss of consciousness, history of seizure, and current substance abuse or dependence. Participants with BDP were excluded for Clozaril use in the cognitive remediation study due to its sedating side effects. HC participants had no history of a psychiatric diagnosis, no first-degree relatives diagnosed with a psychiatric illness, and no history of substance abuse or dependence.

Inclusion criteria included a DSM-IV (SCID-IV-TR) diagnosis of SZ, schizoaffective disorder depressed type, or BDP. BDP was defined as having at least one psychotic symptom (3 on at least one criteria A SCID-IV item) during a manic or depressed episode during the lifetime. Three participants with schizoaffective disorder depressed type were included in the SZ sample as Kern et al. (2011) combined these groups in the initial standardization studies of the MCCB. The final study sample included patients with BDP ($n = 56$), patients with SZ ($n = 37$), and HC ($n = 57$). All participants were between the ages of 18 and 55.

Materials

Diagnosis was confirmed by Structured Clinical Interview for DSM-IV (SCID-IV-TR) through patient interview, medical record review, and consultation with the participants' treatment provider(s). Clinical assessment included the Young Mania Rating Scale (YMRS; Young, Biggs, Ziegler, & Meyer, 1978), the Montgomery-Asberg Depression Rating Scale (MADRS; Montgomery & Asberg, 1979), and the Positive and Negative Syndrome Scale (PANSS; Kay, Fiszbein, & Opler, 1987). Community functioning was measured using an abbreviated version of the Multnomah Community Ability Scale (MCAS) as described in Lewandowski et al. (2013). The MCCB was used to measure neurocognitive and social cognitive functioning. The MCCB produces six domain scores and a composite. Domains include: Speed of Processing (Trail Making Test A; Brief Assessment of Cognition in Schizophrenia: Symbol Coding; Category Fluency); Attention/Vigilance (Continuous Performance Test: Identical Pairs); Working Memory (Wechsler Memory Scale Spatial Span; Letter Number Span); Visual Learning (Brief Visuospatial Memory Test); Verbal Learning (Hopkins Verbal Learning Test); Reasoning/Problem Solving (Neuropsychological Assessment Battery: Mazes); Social Cognition (Mayer-Salovey-Caruso Emotional Intelligence Test: Managing Emotions). Domain scores and the composite were converted to standard (T) scores based on MCCB age and gender adjusted norms.

Design and Procedure

Neuropsychological and clinical assessments were conducted in a single session lasting approximately 2 hrs. Procedures across all studies were standardized and the same study staff completed these assessments. The cognitive and clinical

https://doi.org/10.1017/S1355617715000442 Published online by Cambridge University Press

symptom assessments were always completed within the same visit. The diagnostic interview was administered before neurocognitive testing. Chlorpromazine equivalents (CPZ) were calculated (Baldessarini, 2012).

Statistical Analysis

Subjects were compared on demographic and clinical variables using analysis of variance (ANOVA) or Chi-Square. Cognitive domain and composite scores were compared by group using a One-Way ANOVA followed by pairwise comparisons. A subsequent analysis of covariance (ANCOVA) was used for pairwise comparisons between SZ and BDP on cognitive domains, covarying for PANSS score. Effect sizes were calculated using Cohen's D. To examine the association of cognition and community functioning, a series of linear regressions was performed predicting MCAS scores using neurocognitive scores as predictors after accounting for the effects of education, PANSS, MADRS total, and diagnosis. All analyses were performed using SPSS v.19.

RESULTS

Groups differed on age, sex, education, MCAS, MADRS, PANSS, and CPZ (Table 1). Correlations were conducted to examine the relationship between clinical and cognitive measures. PANSS was significantly correlated with Processing Speed, r = −.38, $p < .01$, Working Memory, r = −.29, $p < .01$, Verbal Memory, r = −.24, $p < .05$, and the Composite, r = −.28, $p < .01$; YMRS and MADRS were not correlated with any cognitive measures ($p > .05$).

A One-Way ANOVA examining cognitive performance by diagnosis indicated that groups differed on all domains and the Composite ($p < .001$ for all tests). To correct for multiple comparisons, all pairwise analyses were Bonferroni-corrected ($a = .006$) (see Table 2). Patients with BDP performed worse than HC on Processing Speed, Verbal Memory, Problem Solving, and the Composite; patients with BDP performed better than patients with SZ on all domains except Visual Memory and Problem Solving. Patients with SZ performed worse than healthy controls on all MCCB domains. All pairwise comparisons yielded moderate to large effect sizes. To account for differences in PANSS scores between patient groups, ANCOVAs comparing BDP and SZ groups on all MCCB variables covarying PANSS total score were conducted. After accounting for PANSS score, BDP and SZ no longer differed on Processing Speed [$F(1,91) = 6.25$; $p > .05$; $d = 0.78$] or Attention [$F(1,89) = 6.98$; $p > .05$; $d = 0.65$], although effect sizes were still in the moderate range.

A series of linear regressions was conducted examining the association between MCCB domains and community function after accounting for the effects of education, PANSS, MADRS, CPZ, and diagnosis. PANSS and MADRS were included as confounders as they were correlated with MCAS scores. We included patients only in this analysis, as there was very little variance in the HC group on MCAS scores (as would be expected based on the nature of the instrument). We found no associations between any MCCB domain and the MCAS ($p > .05$).

DISCUSSION

The present study examined the utility of the MCCB in the measurement of neuro- and social cognitive deficits in BDP and associations between MCCB score and community functioning. As expected, patients with BDP performed better than patients with SZ on most domains, although some domains were no longer significant after controlling for PANSS score.

This is the first study to investigate the cognitive profile of patients with BDP using the MCCB compared to two control groups: healthy participants and patients with SZ. Restricting our sample along a symptom dimension that has been associated with cognition may improve the interpretability of findings of MCCB performance in BDP compared to SZ and healthy controls.

Previous reports suggest that patients with BD perform approximately 0.4 to 1.2 *SD*s below the mean on most

Table 1. Demographic and clinical variables by group

	BDP ($n = 56$)	SZ ($n = 37$)	HC ($n = 57$)	Test statistic	
Age	30.50 (8.16)	33.95 (11.12)	25.67 (6.42)	F (2,148) = 11.45***	HC < BDP, SZ
Education[a]	5.43 (1.59)	3.97 (1.28)	5.53 (1.54)	F (2,148) = 14.08***	SZ < BDP, HC
% Caucasian	82.14%	87.5%	78.38%	X^2 (2,148) = 4.55[n.s]	BDP = SZ = HC
% Female	57.14%	13.51%	59.65%	X^2 (2,148) = 22.61***	SZ < BDP, HC
YMRS	5.21 (4.67)	5.38 (6.18)	—	F (1,92) = .02[n.s]	BDP = SZ
MADRS	12.13 (7.23)	9.00 (7.72)	—	F (1,92) = 3.94*	SZ < BDP
PANSS	46.61 (8.37)	55.73 (15.72)	—	F (1,92) = 13.24***	BDP < SZ
MCAS	47.91 (4.08)	43.51 (8.07)	54.74 (0.67)	F (2,148) = 67.79***	SZ < BDP < HC
CPZ	169.57 (182.53)	271.62 (235.13)	—	F (1,92) = 5.52*	BDP < SZ

[a]Education is coded based on the SCID: 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.
*$p < .05$.
***$p < .001$.

https://doi.org/10.1017/S1355617715000442 Published online by Cambridge University Press

Table 2. MCCB domain and composite T scores by group

	BDP (n = 56)	SZ (n = 37)	HC (n = 57)	HC vs. BDP Cohen's D	HC vs. SZ Cohen's D	BDP vs. SZ Cohen's D
ProcSpeed	46.79 (10.50)	38.43 (11.33)	59.89 (8.30)	F(1,111) = 54.30* d = 1.39	F(1,92) = 112.11* d = 2.23	F(1,91) = 13.25* d = 0.78
Attention	46.31 (9.91)	39.67 (10.87)	50.51 (8.37)	F(1,110) = 5.88$^{n.s}$ d = 0.46	F(1,91) = 29.28* d = 1.15	F(1,89) = 9.05* d = 0.65
WMemory	48.64 (9.68)	37.46 (10.94)	52.98 (8.12)	F(1,111) = 6.68$^{n.s}$ d = 0.49	F(1,92) = 62.20* d = 1.65	F(1,91) = 26.81* d = 1.10
Verbal	47.46 (9.77)	39.97 (7.48)	52.77 (8.48)	F(1,111) = 9.58* d = 0.58	F(1,92) = 55.99* d = 1.58	F(1,91) = 15.66* d = 0.84
Visual	44.30 (10.48)	39.30 (10.92)	48.88 (7.10)	F(1,111) = 7.40$^{n.s}$ d = 0.51	F(1,92) = 26.61* d = 1.09	F(1,91) = 4.92$^{n.s}$ d = 0.47
ProbSolving	46.68 (9.61)	45.84 (10.06)	51.32 (7.91)	F(1,111) = 7.86* d = 0.53	F(1,92) = 8.67* d = 0.62	F(1,91) = 0.16$^{n.s}$ d = 0.09
Social	50.75 (9.92)	40.62 (11.77)	53.98 (11.65)	F(1,111) = 2.52$^{n.s}$ d = 0.30	F(1,92) = 29.28* d = 1.14	F(1,91) = 20.00* d = 0.95
Composite	45.64 (9.03)	34.28 (10.58)	54.53 (6.80)	F(1,110) = 34.80* d = 1.11	F(1,91) = 126.55* d = 2.39	F(1,89) = 30.01* d = 1.17

Note. Bonferroni corrected p-values.
*$p < .006$.

measures, and patients with SZ perform approximately 1 to 1.7 *SD*s below the mean (Burdick et al., 2011; Kern et al., 2011); however, we found that on average, all three groups performed approximately 0.5 *SD* better than would be expected compared to MCCB norms and previous findings in BD, SZ, and HC studies using the MCCB (Burdick et al., 2011). Our samples may have been slightly higher functioning than is typical due to access to higher education in our recruitment area.

Consistent with the other two reports of MCCB in BD, we did not find social cognitive deficits in our BDP sample using the MSCEIT, although we did find significant deficits in social cognition in our SZ patients. While substantial evidence exists of deficits in some aspects of social cognition in patients with BD including deficits in facial emotion perception (Van Rheenen & Rossell, 2013b) and Theory of Mind (Samamé et al., 2012), social cognition is a multifaceted construct and not all domains appear to be equally affected in BD. Indeed, the specific nature of social cognitive deficits between SZ and BD may differ; thus, while the MSCEIT: Managing Emotions appears to detect significant deficits in patients with SZ, this measure may not adequately capture the social cognitive deficits commonly experienced by patients with BDP. Emotion perception and ToM appear to be among the most commonly reported areas of social cognitive dysfunction in BD. Since social cognitive deficits specific to BDP are not adequately captured by the MCCB, we recommend inclusion of one or more diagnosis-appropriate measures to assess specific aspects of social cognition when using the MCCB in this population.

Cognitive deficits are among the strongest predictors of community functioning in patients with SZ and BD. Unexpectedly, we did not find any associations between MCCB domains and community functioning in our sample. This may be due to our measure of community functioning, which assesses broad domain of functioning using patient report and may not be sensitive in evaluating current instrumental functional deficits associated with cognitive processing. Based upon recommendations by the FDA, the MATRICS Initiative suggests including an ecologically valid measure of psychosocial functioning, particularly performance-based measures that may have greater sensitivity in detecting functional deficits related to cognition (Green et al., 2008).

Several limitations of the present study should be noted. Data were collected from multiple separate but related studies, and several inclusion and exclusion criteria differed by diagnostic group including clinical symptomatology at baseline and use of clozapine. While we controlled for CPZ in some of our analyses, no subjects in the BDP group were prescribed clozapine, whereas clozapine use was not an exclusion for participants with SZ. We did not examine the effects of Lithium or mood stabilizers on cognition. Future studies would benefit from examining these differences. In addition, groups were not well matched on age and gender; however, MCCB scores are normed based on age and gender so these differences are not expected to affect the results. Lastly, analyses did not examine the effects of premorbid IQ on cognitive functioning, as these data were not available for the full sample.

The MCCB appears to be a valid measure of neurocognitive deficits in patients with BDP across a range of relevant domains at the group level; however, there is considerable cognitive heterogeneity in patients with BD (and other psychotic disorders). Several studies report that approximately 30 to 40% of patients with BD perform in the normal range on measures of neurocognition (Burdick et al., 2014; Lewandowski, Sperry, Cohen, & Ongür, 2014). Consistent with these reports, 37.5% of our BDP sample fell in the "normal" range, ≥ -0.5 *SD*s on the MCCB Composite. Group-level analyses may obscure a more precise examination of the cognitive heterogeneity common across the psychoses.

https://doi.org/10.1017/S1355617715000442 Published online by Cambridge University Press

The present findings suggest that the MCCB is an appropriate measure of neurocognitive functioning but not social cognition in BD, and highlights the lack of diagnostic specificity of most domains of the MCCB in the bipolar-schizophrenia spectrum. However, given that social cognition is a multidimensional construct, consideration should be given to the appropriateness of the MCCB social cognition measure for particular study populations. For instance, measures of emotion processing and ToM should be added to the MCCB when studying social cognitive dysfunction in BD. Many measures used to examine these constructs in BD are also well validated in SZ and can be used cross-diagnostically. In addition, these findings highlight the importance of using well-validated performance based measures of functional outcomes to elucidate whether cognitive improvements result in better community functioning.

ACKNOWLEDGMENTS

We thank the participants for their time and effort and Samira Pingali for her assistance with data collection. This work was supported by the National Institutes of Health (KEL, Grant #K23MH91210 and DO, Grant #R01MH094594, #R21MH096107); The Shervert Frazier Research Institute at McLean Hospital (KEL); NARSAD/BBRF Independent Investigator Award (DO). Dost Öngur served on the scientific advisory board for Eli Lilly. Kathryn Lewandowski served as a consultant for Clintara. Matcheri Keshavan has a grant with Sunovion. Sarah Sperry, Bruce Cohen, and Lauren O'Connor report no disclosures.

REFERENCES

Baldessarini, R.J. (2012). *Chemotherapy in psychiatry*. New York: Springer Verlag.

Burdick, K.E., Goldberg, T.E., Cornblatt, B.A., Keefe, R.S., Gopin, C.B., Derosse, P., … Malhotra, A.K. (2011). The MATRICS consensus cognitive battery in patients with bipolar I disorder. *Neuropsychopharmacology*, *36*(8), 1587–1592. doi: 10.1038/npp.2011.36

Burdick, K.E., Russo, M., Frangou, S., Mahon, K., Braga, R.J., Shanahan, M., & Malhotra, A.K. (2014). Empirical evidence for discrete neurocognitive subgroups in bipolar disorder: Clinical implications. *Psychological Medicine*, *44*, 3083–3096.

Couture, S.M., Penn, D.L., & Roberts, D.L. (2006). The functional significance of social cognition in schizophrenia: A review. *Schizophrenia Bulletin*, *32*, S44–S63. doi: 10.1093/schbul/sbl029

Green, M.F. (2006). Cognitive impairment and functional outcome in schizophrenia and bipolar disorder. *Journal of Clinical Psychiatry*, *67*(10), e12.

Green, M.F., Nuechterlein, K.H., Kern, R.S., Baade, L.E., Fenton, W.S., Gold, J.M., & Marder, S.R. (2008). Functional co-primary measures for clinical trials in schizophrenia: Results from the MATRICS Psychometric and Standardization Study. *The American Journal of Psychiatry*, *165*(2), 221–228. doi: 10.1176/appi.ajp.2007.07010089

Hill, S.K., Reilly, J.L., Keefe, R.S., Gold, J.M., Bishop, J.R., Gershon, E.S., & Sweeney, J.A. (2013). Neuropsychological impairments in schizophrenia and psychotic bipolar disorder: Findings from the Bipolar-Schizophrenia Network on Intermediate Phenotypes (B-SNIP) study. *American Journal of Psychiatry*, *170*(11), 1275–1284. doi: 10.1176/appi.ajp.2013.12101298

Kay, S.R., Fiszbein, A., & Opler, L.A. (1987). The positive and negative syndrome scale (PANSS) for schizophrenia. *Schizophrenia Bulletin*, *13*(2), 261–276. doi: 10.1093/schbul/12.2.261

Kern, R.S., Gold, J.M., Dickinson, D., Green, M.F., Nuechterlein, K.H., Baade, L.E., & Marder, S.R. (2011). The MCCB impairment profile for schizophrenia outpatients: Results from the MATRICS psychometric and standardization study. *Schizophrenia Research*, *126*(1-3), 124–131.

Lewandowski, K.E., Cohen, B.M., Keshavan, M.S., & Ongür, D. (2011). Relationship of neurocognitive deficits to diagnosis and symptoms across affective and non-affective psychoses. *Schizophrenia Research*, *133*(1-3), 212–217. doi: 10.1016/j.schres.2011.09.004

Lewandowski, K.E., Cohen, B.M., Keshavan, M.S., Sperry, S.H., & Ongür, D. (2013). Neuropsychological functioning predicts community outcomes in affective and non-affective psychoses: A 6-month follow-up. *Schizophrenia Research*, *148*(1-3), 34–37. doi: 10.1016/j.schres.2013.05.012

Lewandowski, K.E., Sperry, S.H., Cohen, B.M., & Ongür, D. (2014). Cognitive variability in psychotic disorders: A cross-diagnostic cluster analysis. *Psychological Medicine*, *44*(15), 3239–3248. doi: 10.1017/S0033291714000774

Marder, S.R., & Fenton, W.S. (2004). Measurement and treatment research to improve cognition in schizophrenia: NIMH MATRICS initiative to support the development of agents for improving cognition in schizophrenia. *Schizophrenia Research*, *72*, 5–9. doi: 10.1016/j.schres.2004.09.010

Montgomery, S.A., & Asberg, M. (1979). A new depression scale designed to be sensitive to change. *British Journal of Psychiatry*, *134*, 382–389.

Robinson, L.J., Thompson, J.M., Gallagher, P., Goswami, U., Young, A.H., Ferrier, I.N., & Moore, P.B. (2006). A meta-analysis of cognitive deficits in euthymic patients with bipolar disorder. *Journal of Affective Disorders*, *93*(1-3), 105–115. doi: 10.1016/j.jad.2006.02.016

Samamé, C., Martino, D.J., & Strejilevich, S.A. (2012). Social cognition in euthymic bipolar disorder: Systematic review and meta-analytic approach. *Acta Psychiatrica Scandinavica*, *125*(4), 266–280. doi: 10.1111/j.1600-0447.2011.01808.x

Van Rheenen, T.E., & Rossell, S.L. (2013a) An empirical evaluation of the MATRICS Consensus Cognitive Battery in bipolar disorder. *Bipolar Disorders*, *16*(3), 318–325. doi: 10.1111/bdi.12134

Van Rheenen, T.E., & Rossell, S.L. (2013b). Is the non-verbal behavioural emotion-processing profile of bipolar disorder impaired? A critical review. *Acta Psychiatrica Scandinavica*, *128*(3), 163–178.

Van Rheenen, T.E., & Rossell, S.L. (2014). Phenomenological predictors of psychosocial function in bipolar disorder: Is there evidence that social cognitive and emotion regulation abnormalities contribute? *Australian and New Zealand Journal of Psychiatry*, *48*(1), 26–35. doi: http://dx.doi.org/10.1177/0004867413508452

Yatham, L.N., Torres, I.J., Malhi, G.S., Frangou, S., Glahn, D.C., Bearden, C.E., & Chengappa, K.N. (2010). The International Society for Bipolar Disorders-Battery for Assessment of Neurocognition (ISBD-BANC). *Bipolar Disorders*, *12*(4), 351–363. doi: 10.1111/j.1399-5618.2010.00830

Young, R.C., Biggs, J.T., Ziegler, V.E., & Meyer, D.A. (1978). A rating scale for mania: Reliability, validity and sensitivity. *British Journal of Psychiatry*, *133*, 429–435.