



Preliminary Evidence for Sociotropy and Autonomy in Relation to Antidepressant Treatment Outcome

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Abstract

Sociotropy and autonomy are cognitive-personality styles that have been hypothesized to confer vulnerability to different presentations of major depressive disorder (MDD), which may respond differentially to treatment. Specifically, the profile of low sociotropy and high autonomy is hypothesized to indicate a positive response to antidepressant medication. The current study examines sociotropy and autonomy in relation to sertraline treatment response in individuals with MDD. As part of an ancillary study to the larger Establishing Moderators and Biosignatures of Antidepressant Response for Clinical Care (EMBARC) project, individuals with MDD participated in an 8-week trial of sertraline and completed a self-report questionnaire of sociotropy and autonomy. Discriminant function analyses were used to examine whether sociotropy and autonomy scores could distinguish antidepressant treatment responders (determined by a 50% or greater reduction in depressive symptoms) from non-responders. The sociotropy scale successfully discriminated sertraline treatment responders from non-responders. Further, lower sociotropy was associated with greater improvements in depressive symptomology following sertraline treatment. The current findings suggest individuals with MDD characterized by low sociotropy are more likely to benefit from sertraline. Given the promising results of the Sociotropy-Autonomy Scale in discriminating treatment responders from non-responders, the low resources necessary for administration, and the ease of translation into routine clinical care, the scale warrants further research attention.

Keywords Sociotropy · Autonomy · Major depressive disorder · Antidepressant · SSRI

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Introduction

Major Depressive Disorder is a highly prevalent illness that affects 20.6% of adults in the United States at some time in their life [8]. The first line of treatment in most pharmaceutical interventions for major depressive disorder is a selective serotonin reuptake inhibitor (SSRI); unfortunately, partial response and treatment resistance to first line antidepressant medication is common [5, 20–22]. Response rates to SSRIs have been estimated at under 50%, with full remission rates estimated at 33% or less [20, 22]. Further, response to treatment takes on average 4 weeks or longer, demonstrating the importance of matching patients to effective treatments on the first attempt [11]. Matching individuals with major depressive disorder to treatments could be aided by the identification of low-cost instruments which have the possibility of easy and swift translation into routine care, such as assessment of psychological variables that are related to treatment response [4]. Therefore, research that aims to improve treatment decision-making may benefit from the inclusion of such psychological factors.

Two psychological variables that show promise for predicting antidepressant treatment response are cognitive-personality styles of sociotropy and autonomy, first described by Beck [2]. Highly sociotropic individuals are prone to interpersonal dependency, characterized by a high need for close relationships, and tend to be very concerned with how they are viewed by others whom they often work hard to please. Individuals high in autonomy are characterized by a heavy emphasis on personal achievements, independence, and control. It was hypothesized that highly sociotropic individuals are vulnerable to depression following interpersonal loss, while autonomous individuals are vulnerable to depression as a result of perceived life failures. It is generally expected that an individual demonstrates either a predominantly sociotropic or autonomous style, though a combination of both is possible [2]. Sociotropy and autonomy dimensions have shown relative stability over time in individuals with depressive disorders despite changes in depressive symptomology with treatment, with observed test–retest correlations of 0.77 for sociotropy and 0.72 for autonomy [1, 10, 19]. Critically, sociotropic and autonomous styles may confer vulnerability to different depression symptom presentations with implications for treatment outcomes [2].

Indeed, sociotropy and autonomy relate to different clinical features or subtypes of depressive disorders, which may respond differentially to antidepressant treatment [12, 13, 15, 16]. Preliminary evidence has suggested the combination of low sociotropy and high autonomy predicts a positive response to pharmacotherapy [12, 13]. Consistent with this, Scott et al. [19] found that higher autonomy scores predicted lower symptom severity after 3 months of antidepressant treatment and recovery from depression at 6 months. These findings lead Scott et al. [19] to hypothesize that highly sociotropic individuals with major depressive disorder would show a symptom presentation that would be unlikely to respond to antidepressant medication. The current study tests this hypothesis by examining whether sociotropy and autonomy can discriminate treatment responders from non-responders. The current study uses the revised Sociotropy-Autonomy Scale [3]. Although this version improved psychometric properties, it has not been directly studied in relation to antidepressant treatment outcomes.

Study Aims

This study aimed to extend prior work by examining the relationship of sociotropy and autonomy to sertraline treatment outcomes in people with major depressive disorder (MDD) using the revised version of the Sociotropy-Autonomy Scale [3]. We assessed the accuracy by which these cognitive-personality styles can distinguish between sertraline responders and non-responders. We hypothesize that (1) indices of sociotropy and autonomy would differentiate treatment responders from non-responders, specifically: a profile of relatively low sociotropy and relatively high autonomy will be associated with positive sertraline response; (2) sociotropy will be negatively associated with percent change in depressive symptoms following treatment; and (3) autonomy will be positively associated with percent change in depressive symptoms. These results would suggest that assessment of sociotropy and autonomy hold promise for indicating which individuals with major depressive disorder are more likely to improve with sertraline medication.

Methods

Participants

Participants in this study were recruited from the Establishing Moderators and Biosignatures of Antidepressant Response for Clinical Care study (EMBARC; NCT01407094) [23]. EMBARC was a multi-site collaboration among the University of Michigan, the University of Texas Southwestern, Columbia University, and Massachusetts General Hospital supported by the National Institute of Health to examine biomarkers of treatment response in MDD without psychotic features. Participants in the EMBARC study were first randomized to receive either sertraline or a placebo during phase 1 of the study (first 8 weeks). During phase 2 of the study (second 8 weeks), sertraline non-responders (as determined by a reduction in depressive symptoms of 50% or less, [18]) were then randomized to receive bupropion or a placebo, and placebo non-responders were then randomized to receive either sertraline or a second placebo.

At phase 1, the EMBARC study screened and enrolled outpatients (age range: 18–65 years) meeting criteria for MDD without psychotic features based on the Structured Clinical Interview for DSM-IV Axis I Disorders [6]. Participants had a Quick Inventory of Depressive Symptomatology (QIDS) score of 14 or higher, indicating moderate depression at both the screening and randomization visits [17]. To minimize clinical heterogeneity, only patients reporting early-onset (before age 30 years) MDD that was chronic (episode duration > 2 years) or recurrent (≥ 2 recurrences including the current episode) were enrolled. Additional exclusion criteria are published elsewhere [14].

This ancillary study was approved by the EMBARC team in order to collect and assess cognitive styles and personality traits that may differentiate treatment responders from non-responders. IRB approval was obtained from the boards at each of the 4 sites. Participants who signed a consent to be contacted for future research studies were contacted by phone or in person at the end of their final (Week 16) follow up visit in the larger EMBARC project and asked about their interest in participating in this project. Participants who completed participation in the larger EMBARC study within the past six months and were in

the sample of patients with MDD were eligible to enroll in this study. Healthy controls from the larger EMBARC project were excluded from this study.

Thirty-seven individuals were contacted for participation in this ancillary study. Of the 37 participants contacted, 30 have completed this ancillary study, 2 declined to participate, and 5 were unreachable. Of the 30 who completed the ancillary study, 4 were excluded from these analyses as they were randomized to receive a placebo, and 2 were abnormal terminations that did not complete the antidepressant trial. 17 of the participants originated from the University of Michigan site; 3 are from UT Southwestern; and 4 are from Massachusetts General Hospital. All participants provided signed informed consent.

Thus, data from 24 participants with MDD (Mean Age=38.25, SD=14.78, Range: 18–65, 62.5% Female, 87.5% Caucasian) were included in the current study. Descriptive statistics of sample demographics and variables of interest, broken down by treatment response (responder vs. non-responder), are presented in Table 1.

Participants who indicated they were willing to participate in the ancillary study were given a questionnaire packet with mailing materials to take home. The packet included 10 questionnaires; the Sociotropy-Autonomy Scale is of interest to the current study. Compensation for participation in the ancillary study was \$10.

Measures

Depressive Symptomology Depressive symptoms were assessed with the 17-item Hamilton Rating Scale for Depression, which is a clinician-administered measure [7]. The HDRS

Table 1 Descriptive statistics of demographic characteristics of the sample, clinical variables of interest, and Sociotropy-Autonomy Scale (SAS) scores

	Responders		Non-responders		<i>F</i> or χ^2	<i>p</i>
	(N=9)		(N=15)			
	M	SD	M	SD		
Demographics						
Sex (M/F)	5/4		10/5		0.30	0.59
Race (Caucasian/Non-Caucasian)	7/2		14/1		1.24	0.26
Ethnicity (Hispanic/Non-Hispanic)	0/9		1/14		0.63	0.43
Age	41.56	16.11	36.27	14.12	0.711	0.408
Education (yrs)	16.11	3.37	16.18	2.55	0.003	0.957
Clinical characteristics						
HDRS baseline	17.67	5.57	16.73	5.19	0.17	0.68
HDRS raw change	12.78	5.37	1.4	4.56	30.74	<.001
HDRS reduction	72.0%	15.1%	4.0%	34.1%	31.51	<.001
SAS scale						
Sociotropy	54.22	9.42	68.20	16.75	5.22	0.032
Solitude/interpersonal insensitivity	25.89	10.75	23.87	8.63	0.26	0.617
Independence	40.00	6.60	39.20	7.09	0.08	0.786
Individualistic achievement	28.44	9.14	29.13	6.89	0.044	0.836
Total autonomy	94.33	18.41	92.20	18.11	0.08	0.784

HDRS = Hamilton Depression Rating Scale

was administered both in the first and last week of receiving sertraline treatment. Higher scores indicate more severe depressive symptomatology.

Sociotropy and Autonomy Sociotropy and autonomy were both assessed using the 74-item revised version of the Sociotropy-Autonomy Scale (SAS; [3]). This version consists of a sociotropy subscale and three subscales of autonomy: solitude/interpersonal insensitivity, independence, and individualistic achievement. Sociotropy consists of 28 items; autonomy subscales consist of 16, 16, and 14 items, respectively. For each item, participants rate the extent to which a statement applies to them on a 0–4 scale (0 = “never”, 4 = “all the time”). All subscales show acceptable internal consistency (alpha coefficients: 0.87 for sociotropy, 0.77 for solitude/interpersonal insensitivity, 0.78 for independence, and 0.76 for individualistic achievement; [3]).

Data Analysis

Participants who received sertraline either during the first phase of the study or during the second phase of the study (after a placebo non-response) were included in the present analyses, and HDRS percent change was calculated based on difference in HDRS scores from the first and last week they received sertraline treatment divided by baseline HDRS score. No differences were detected between phase 1 and phase 2 participants on any variables of interest (see Table 2).

The data were analyzed using IBM SPSS Version 26 [9]. Prior to conducting analyses, we assessed for outliers and ensured the statistical assumption of normality was met. No data points were deemed outliers. Participants were deemed responders if they exhibited a 50% or greater reduction in HDRS-17 scores over 2 months of antidepressant treatment [18].

One-way ANOVAs were conducted to examine differences in sociotropy and autonomy variables between treatment responders and non-responders. Subsequently, we assessed the discriminating ability of the sociotropy and autonomy scales using a separate discriminant function analysis for each of the 4 subscales (independent variable: SAS subscale, outcome: treatment response group). Additionally, we conducted bivariate correlations to examine whether sociotropy and autonomy subscales were linearly related to percent

Table 2 Comparison of Phase 1 and Phase 2 participants on variables of interest. Phase 1 participants did not significantly differ from Phase 2 Participants on any variables of interest to the current study

	Phase 1 (N=17)		Phase 2 (N=7)		F	p
	Mean	SD	Mean	SD		
HDRS Percent Change	19.0%	4.5%	54.0%	32.5%	3.55	0.07
HDRS Baseline Score	17.35	5.17	16.43	5.74	0.15	0.70
HDRS Raw Change Score	4.00	6.87	9.71	7.46	3.28	0.08
Sociotropy	63.94	15.84	60.57	16.65	0.22	0.65
Solitude/Interpersonal Insensitivity	25.18	9.33	23.29	9.83	0.20	0.66
Independence	38.18	7.24	42.71	4.39	2.36	0.14
Individualistic Achievement	29.65	6.62	27.00	9.98	0.59	0.45
Total Autonomy	93.00	19.17	93.00	15.52	<.001	1.00

HDRS = Hamilton Depression Rating Scale

change in depressive symptom severity (HDRS score). Autonomy subscales were each entered as individual variables for analyses, as they were suggested to be treated as three separate subscales by Clark and Beck [3].

Results

Descriptive statistics, broken down by treatment response, are presented in Table 1. Those who responded to antidepressant treatment reported significantly lower levels of sociotropy relative to treatment non-responders ($F(1, 22)=5.215, p=0.032$). Treatment responders and non-responders did not differ significantly on any of the three autonomy subscales (solitude/interpersonal insensitivity: $F(1, 22)=0.257, p=0.617$; independence: $F(1, 22)=0.075, p=0.786$; individualistic achievement: $F(1, 22)=0.044, p=0.836$).

Discriminant Function Analyses

Sociotropy The statistical assumption of Homogeneity of Covariance Matrices was satisfied as indicated by a non-significant Box's M of 2.921 ($F(1, 1185)=2.785, p=0.095$). The overall estimated function was significant (Wilk's Lambda=0.808, $\chi^2(1)=4.574, p=0.032$). The estimated function explained 23.7% of the variance in treatment response with a canonical correlation of 0.438. Classification analyses using the estimated function correctly classified 5/9 responders and 13/15 non-responders, with 55.6% sensitivity and 86.7% specificity. Overall, sociotropy correctly classified 75% of originally grouped cases.

Solitude/Interpersonal Insensitivity The statistical assumption of Homogeneity of Covariance Matrices was satisfied as indicated by a non-significant Box's M of 0.511 ($F(1, 1185)=0.486, p=0.486$). The overall estimated function was not significant (Wilk's Lambda=0.988, $\chi^2(1)=0.250, p=0.617$).

Independence The statistical assumption of Homogeneity of Covariance Matrices was satisfied as indicated by a non-significant Box's M of 0.053 ($F(1, 1185)=0.051, p=0.822$). The overall estimated function was not significant (Wilk's Lambda=0.997, $\chi^2(1)=0.073, p=0.786$).

Individualistic Achievement The statistical assumption of Homogeneity of Covariance Matrices was satisfied as indicated by a non-significant Box's M of 0.849 ($F(1, 1185)=0.808, p=0.369$). The overall estimated function was not significant (Wilk's Lambda=0.998, $\chi^2(1)=0.043, p=0.836$).

Correlational Analyses

Correlations between Sociotropy-Autonomy subscales and percent change in HDRS scores are depicted in Table 3. Consistent with the above finding that sociotropy discriminated treatment responders from non-responders, correlation results revealed decreased sociotropy score was associated with increased percent change in depressive symptomology ($r(22)=-0.557, p=0.005$). Autonomy subscales were not significantly associated with percent change in HDRS score (p -values > 0.05).

Table 3 Correlations between Sociotropy-Autonomy Scales (SAS) and percent change in depressive symptoms (HDRS-17) following sertraline treatment

SAS subscale	Correlation between SAS subscale and percent change in HDRS-17	
	<i>r</i>	<i>p</i>
Sociotropy	-0.557	0.005
Solitude/Interpersonal Insensitivity	0.012	0.957
Independence	-0.251	0.236
Individualistic achievement	-0.043	0.843
Total autonomy sum	-0.108	0.617

HDRS = Hamilton Depression Rating Scale

Discussion

The current study examined relationships between sociotropy, autonomy, and antidepressant treatment outcome, using the revised Sociotropy-Autonomy Scale [3]. We found that lower sociotropy was associated with greater reduction in depressive symptomology following sertraline treatment. Further, the sociotropy subscale was able to successfully discriminate treatment responders from non-responders (classifying 75% of individuals correctly). The current findings are consistent with the hypothesis that a relatively high level of sociotropy indexes a profile of major depressive disorder that is likely to respond poorly to first-line antidepressant treatment [2, 15, 16, 19].

Results of the current study extend prior work suggesting that the Sociotropy-Autonomy Scale can differentiate antidepressant treatment responders and non-responders [12, 13, 19]. The current study did not find that the autonomy subscale was associated with treatment outcome, as was previously observed [12, 13, 19]. However, given an individual typically shows one predominant style [2], the current finding regarding low sociotropy is generally consistent with prior findings that the profile of lower sociotropy relative to autonomy indicates a positive treatment response [12, 13]. It is possible that differences in measures of autonomy may have contributed to discrepancies in findings regarding the relationship between autonomy and treatment response; the revised version of the SAS used in the current study made more substantial changes to the autonomy subscale(s) than sociotropy [3]. In the current study, the revised sociotropy subscale alone successfully discriminated sertraline responders from non-responders, suggesting this subscale may be more useful in indicating treatment response.

Limitations

Findings of the current study should be interpreted in light of the small sample size. Additionally, the study sample consisted of participants from both phase 1 and phase 2 of the EMBARC study. Although no significant differences on SAS scores between phase 1 and phase 2 participants were observed, the different lengths of treatment duration (8 weeks versus 16 weeks) should be noted. A final limitation of importance is the post-treatment assessment of sociotropy and autonomy, which may have biased findings. SAS scores may have been influenced by post-treatment drops in depressive symptoms; although, research

has suggested sociotropy and autonomy to be personality constructs that are relatively stable over time in populations with depressive disorders receiving pharmacotherapy treatment [1, 10, 19]. Future research should further ascertain this by administering the SAS scale both pre and post-treatment in a larger MDD sample.

Conclusions

Our findings suggest individuals low in sociotropy are more likely to benefit from SSRI medication for major depressive disorder. Due to the relatively low amount of resources necessary to administer the Sociotropy-Autonomy Scale and the ease of translation into routine clinical care, the current study suggests the scale shows promise for predicting antidepressant treatment response, and therefore warrants further research.

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References

1. Bagby RM, Gilchrist EJ, Rector NA, Dickens SE, Joffe RT, Levitt A, Levitan RD, Kennedy SH. The stability and validity of the sociotropy and autonomy personality dimensions as measured by the revised personal style inventory. *Cognit Ther Res*. 2001;25(6):765–79. <https://doi.org/10.1023/A:1012975524455>.
2. Beck AT. Cognitive therapy of depression: new perspectives. In: Clayton PJ, Barnett JE, editors. *Treatment of depression: old controversies and new approaches*. New York: Raven Press; 1983. p. 265–90.
3. Clark DA, Beck AT. Personality factors in dysphoria: a psychometric refinement of beck's sociotropy-autonomy scale. *J Psychopathol Behav Assess*. 1991;13(4):369–88. <https://doi.org/10.1007/BF00960448>.
4. Clarke DE, Kuhl EA. DSM-5 cross-cutting symptom measures: a step towards the future of psychiatric care? *World Psychiatry*. 2014;13:314–6. <https://doi.org/10.1002/wps.20154>.
5. Fava M, Davidson KG. Definition and epidemiology of treatment-resistant depression. *Psychiatr Clin N Am*. 1996;19(2):179–200. [https://doi.org/10.1016/S0193-953X\(05\)70283-5](https://doi.org/10.1016/S0193-953X(05)70283-5).
6. First MB, Spitzer R, Gibbon M, William J. *Structured clinical interview for DSM-IV-TR axis I disorders—non-patient edition (SCID-I/NP)*. New York: Biometric Research Department New York State Psychiatric Institute; 2002.
7. Hamilton M. Development of a rating scale for primary depressive illness. *Br J Soc Clin Psychol*. 1967;6:278–96. <https://doi.org/10.1111/j.2044-8260.1967.tb00530.x>.
8. Hasin DS, Sarvet AL, Meyers JL, Saha TD, Ruan WJ, Stohl M, Grant BF. Epidemiology of adult DSM-5 major depressive disorder and its specifiers in the United States. *JAMA Psychiatry*. 2018;75:336–46. <https://doi.org/10.1001/jamapsychiatry.2017.4602>.
9. IBM Corp. *IBM SPSS statistics for windows* (No. 26). Armonk: NY IBM Corp; 2019.
10. Moore RG, Blackburn IM. The stability of sociotropy and autonomy in depressed patients undergoing treatment. *Cognit Ther Res*. 1994;20:69–80. <https://doi.org/10.1007/BF02229244>.
11. Nierenberg AA, Farabaugh AH, Alpert JE, Gordon J, Worthington JJ, Rosenbaum JF, Fava M. Timing of onset of antidepressant response with fluoxetine treatment. *Am J Psychiatry*. 2000;157:1423–8. <https://doi.org/10.1176/appi.ajp.157.9.1423>.

12. Peselow ED, Robins CJ, Sanfilippo MP, Block P, Fieve RR. Sociotropy and autonomy: relationship to antidepressant drug treatment response and endogenous-nonendogenous dichotomy. *J Abnorm Psychol.* 1992;101:479–86. <https://doi.org/10.1037/0021-843X.101.3.479>.
13. Peselow ED, Sanfilippo MP, Difiglia C, Fieve RR. Melancholic/endogenous depression and response to somatic treatment and placebo. *Am J Psychiatry.* 1992;149:1324–34. <https://doi.org/10.1176/ajp.149.10.1324>.
14. Pizzagalli DA, Webb CA, Dillon DG, et al. Pretreatment rostral anterior cingulate cortex theta activity in relation to symptom improvement in depression: a randomized clinical trial. *JAMA Psychiatry.* 2018;75:547–54. <https://doi.org/10.1001/jamapsychiatry.2018.0252>.
15. Robins CJ, Block P, Peselow ED. Relations of sociotropic and autonomous personality characteristics to specific symptoms in depressed patients. *J Abnorm Psychol.* 1989;98:86–8. <https://doi.org/10.1037/0021-843X.98.1.86>.
16. Robins CJ, Luten AG. Sociotropy and autonomy: differential patterns of clinical presentation in unipolar depression. *J Abnorm Psychol.* 1991;100:74–7. <https://doi.org/10.1037/0021-843X.100.1.74>.
17. Rush AJ, Trivedi MH, Ibrahim HM, et al. The 16-item quick inventory of depressive symptomatology (QIDS), clinician rating (QIDS-C), and self-report (QIDS-SR): a psychometric evaluation in patients with chronic major depression. *Biol Psychiatry.* 2003;54:573–83. [https://doi.org/10.1016/s0006-3223\(02\)01866-8](https://doi.org/10.1016/s0006-3223(02)01866-8).
18. Rush AJ, Kraemer H, Sackeim H, et al. Report by the ACNP task force on response and remission in major depressive disorder. *Neuropsychopharmacology.* 2006;31:1841–53. <https://doi.org/10.1038/sj.npp.1301131>.
19. Scott J, Harrington J, House R, Ferrier IN. A preliminary study of the relationship among personality, cognitive vulnerability, symptom profile, and outcome in major depressive disorder. *J Nerv Ment Dis.* 1996;184:503–5. <https://doi.org/10.1097/00005053-199608000-00008>.
20. Thase ME, Ninan PT, Musgnung JJ, Trivedi MH. Remission with venlafaxine extended release or selective serotonin reuptake inhibitors in depressed patients: a randomized, open-label study. *Prim Care Companion J Clin Psychiatry.* 2011;13.<https://doi.org/10.4088/PCC.10m00979blu>
21. Thomas L, Kessler D, Campbell J, Morrison J, Peters TJ, Williams C, Lewis G, Wiles N. Prevalence of treatment-resistant depression in primary care: cross-sectional data. *Br J Gen Pract J R Coll Gen Pract.* 2013;63:852–8. <https://doi.org/10.3399/bjgp13X675430>.
22. Trivedi MH, Rush AJ, Wisniewski SR, et al. Evaluation of outcomes with citalopram for depression using measurement-based care in STAR*D: implications for clinical practice. *Am J Psychiatry.* 2006;163:28–40. <https://doi.org/10.1176/appi.ajp.163.1.28>.
23. Trivedi MH, McGrath PJ, Fava M, et al. Establishing moderators and biosignatures of antidepressant response in clinical care (EMBARC): rationale and design. *J Psychiatr Res.* 2016;78:11–23.

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