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Socioeconomic status and anxiety as predictors of antidepressant treatment response and suicidal ideation in older adults

Alex Cohen, PhD
Department of Social Medicine, Harvard Medical School, 641 Huntington Avenue, Boston, MA 02115

Stephen E. Gilman, ScD
Departments of Society, Human Development and Health and Epidemiology, Harvard School of Public Health, 677 Huntington Ave. Boston, Massachusetts 02115

Patricia R. Houck, MSH, **Katalin Szanto, MD**, and **Charles F. Reynolds III, MD**
Intervention Research Center for the Study of Late-Life Mood Disorders Thomas Detre Hall of the Western Psychiatric Institute and Clinic University of Pittsburgh, 3811 O'Hara Street, Pittsburgh, PA 15213

Abstract

Background—Independent analyses have shown that socioeconomic status (SES) and anxiety are predictors of response to antidepressant treatment in older adults. SES status has also been demonstrated to be associated with the occurrence of suicidal ideation.

Aim—To determine whether SES differences and baseline anxiety independently contribute to poor depression treatment response and the occurrence of suicidality *or* that higher levels of anxiety in depressed elderly with low SES explain the differences in these outcomes. We hypothesized that neighborhood SES status will predict response to treatment and the occurrence of suicidal ideation even when controlling for baseline levels of comorbid anxiety.

Method—Secondary analyses of data from the Maintenance Treatment for Late-Life Depression Trials

Results—Regression analyses indicate that neighborhood SES remains an independent predictor of response to treatment and the likelihood of experiencing suicidal ideation, while comorbid anxiety remains a predictor of response to treatment.

Conclusion—These findings indicate the importance of treating anxiety symptoms during treatment of late-life depression and, at the same time, addressing barriers of treatment response related to low SES.

Keywords

socioeconomic status; late-life depression; comorbid anxiety; social determinants of health; response to antidepressant treatment

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Introduction

We recently reported that response to treatment for late-life depression varied significantly according to a patient's pre-treatment socioeconomic status (SES) [1]. In the Maintenance Treatment for Late-Life Depression (MTLD) Trial [2,3], participants who lived in middle- or high-income neighborhoods were significantly more likely to respond to treatment than residents of low-income neighborhoods (e.g., median household incomes <$25,000; hazard ratio=1.61.; 95% CI, 1.07, 2.42). In addition, residents of middle- or high-income neighborhoods were less likely to experience suicidal ideation during the course of treatment (odds ratio=0.46; 95% CI, 0.24, 0.87) than residents of low-income neighborhoods.

Our findings regarding lower SES and higher levels of depressive and suicidal symptoms during treatment, as well as those of other studies showing similar patterns of associations [4–7], suggest that the social and contextual factors [8–13] that increase the risk of depression in community samples [14–19] also prolong recovery from depression during treatment. Potential implications of these findings for clinical practice are that lower SES patients entering treatment for depression should be considered at higher risk for treatment failure, and may benefit from more intensive or different interventions [20,21].

Since our study, Andreescu et al. [22] analyzed data from MTLD, and reported that higher pre-treatment levels of anxiety – as measured by the Brief Symptom Inventory [23] – were associated with longer time to treatment response. These findings are consistent with prior studies on the effect of baseline levels of anxiety and subsequent response to treatment for late-life depression [24–26,7,27]. Andreescu et al. discussed the importance of treating anxiety symptoms alongside treatment for depression, but emphasized the need for future research to determine the optimal treatment strategies for older adults suffering from depression and co-occurring symptoms of anxiety.

Viewed together, results from the MTLD and other trials suggest that there are multiple categories of patient characteristics that are identifiable at the time of treatment initiation and which provide important prognostic value [e.g.,24,28,29,7]. However, not all prognostic factors are equal. The identification of prognostic factors such as SES, which reflects a complex set of attributes of an individual's social milieu [30,31], is likely to have implications for the treatment of depression that are different from the implications of identifying other factors such as pre-existing symptoms of anxiety. It is therefore important to determine the relative importance, i.e., the independent effects, of pre-treatment sociodemographic and pre-treatment clinical factors on depressive and suicidal symptoms during treatment.

Accordingly, we reanalyzed data from the MTLD trials to determine whether SES and anxiety independently contribute to poor treatment response and a higher probability of experiencing suicidal ideation. If the effects of SES are reduced after adjusting for baseline levels of anxiety symptoms, this would suggest that social inequalities in response to treatment for late-life depression are due partly to the effects of anxiety symptoms among lower SES patients. Such a finding would be consistent with epidemiologic studies that show significant SES gradients in the risk of anxiety [32–35]. Alternatively, the previously observed effects of baseline anxiety symptoms might be better attributed to SES, in that it has effects on a wide range of health outcomes, and has been hypothesized as a "fundamental cause" of health disparities [36]. Lastly, it is possible that anxiety and SES jointly influence the course of anti-depressant treatment. If that is the case, it would indicate the importance of future research examining diverse clinical samples to better understand moderators of treatment efficacy and effectiveness [37–39], and of integrating information on pre-treatment factors into the design of randomized trials.

Methods

The analyses reported here are based on a sample of 248 subjects who participated in the open-label, non-randomized phases of the two MTLD clinical trials. Comprehensive descriptions of the design of the these trials are available elsewhere [2,3]. Severity of participants' depressive symptoms were measured weekly with the 17- item Hamilton Rating Scale for Depression (HRSD) [40]. Scores on the HRSD were used to generate indicators of treatment efficacy with response defined as HRSD scores of 10 or less for at least 3 consecutive weeks [41]. Suicidal symptoms during the course of treatment were assessed with HRSD item 3. Participants who reported recurrent thoughts of death or wishes to be dead, had active suicidal ideation, or had attempted suicide were classified as experiencing suicidality. We analyzed outcomes during the acute and continuation phases of the trials, which together lasted up to 26 weeks.

SES was measured by tertiles of median census tract annual income based on data from the 2000 U.S. Census [42], and patients' own educational level (categorized as <12 years, 12 years, 13–15 years, and ≥16 years). Baseline anxiety symptoms were assessed with the Brief Symptom Inventory [23]. We also adjusted for patient demographic factors (age, sex, race/ethnicity, and marital status), and other baseline clinical characteristics (first vs. recurrent episode of depression, age at first onset of depression, duration of current episode, concurrent medical burden, and HRSD score).

Cox proportional hazards regression [43] was used to examine the effects of SES and baseline anxiety on the likelihood of response to antidepressant treatment, which was defined as achieving HRSD scores ≤10 for at least 3 consecutive weeks [41]. Repeated-measures generalized logit regression [44] was used to model the presence of suicidal symptoms at each week during treatment. For both outcomes, we estimated a model for baseline anxiety alone, SES alone, and for all covariates together.

Results

The demographic and clinical characteristics of the MTLD participants, shown in Table 1, are presented separately for each category of census tract income. At the time of the baseline interview, prior to treatment initiation, there was no significant association between anxiety symptoms and SES, measured either by median census tract income or patients' educational attainment. This provides an initial indication that the prognostic value of SES and of anxiety symptoms at baseline operate independently from one another.

Results of proportional hazards models of treatment response are shown in the first two columns of Table 2. In the first column of Table 2, adjusted hazard ratios for SES and anxiety are shown from two separate models, one with the SES variables and another with baseline anxiety. The second column shows the adjusted hazard ratios from a model with SES and baseline anxiety together. Hazard ratios for SES were virtually unchanged after accounting for baseline anxiety in the model. The likelihood of treatment response was significantly higher among residents of middle income neighborhoods (HR, 1.77; 95%CI, 1.16–2.71), and marginally higher among residents of high-income neighborhoods (HR, 1.32; 95%CI, 0.80–2.19). In the aggregate, residents of middle- and high-income census tracts combined were 1.63 (95%CI, 1.08–2.46) times more likely to respond to treatment than residents of low-income census tracts. Baseline anxiety symptoms were also predictive of treatment response, as reported by Andreescu et al. [22], but the reduction in the likelihood of response associated with anxiety symptoms was entirely independent of SES (HR, 0.74 [95%CI, 0.61–0.91] without controlling for SES, and HR, 0.73 [95%CI, 0.60–0.89] controlling for SES).

This general pattern holds when examining the effects of SES and baseline anxiety symptoms on suicidal ideation during the course of treatment: SES and anxiety exert independent effects.

Repeated-measures generalized logit regression analyses (Table 2, columns 3 and 4) suggest that, compared to residents of low-income neighborhoods, those in middle-income and high-income neighborhoods, independent of comorbid anxiety, were less likely to report suicidal ideation during treatment (OR, 0.48 [95%CI, 0.27–0.94] and OR, 0.39 [95%CI, 0.16–0.94], respectively). When comorbid anxiety is added to the model, the result is virtually unchanged for residents of middle-income neighborhoods (OR, 0.53 [95%CI, 0.29–0.96]) and slightly reduced for residents of high-income neighborhoods (OR, 0.44 [95%CI, 0.19–1.04]). The odds ratio for middle- and high-income groups combined was 0.51 (95%CI, 0.28–0.90). Independent of SES, baseline anxiety symptoms were associated with a marginally higher odds of suicidal ideation during the course of depression treatment (OR,1.45; [95%CI, 0.98– 2.14]).

The presence of independent effects of SES and baseline anxiety symptoms on depressive symptoms during the course of treatment suggests a marked elevation in the persistence of depression among the low SES individuals who were experiencing anxiety symptoms at the time of treatment initiation. To examine the combined influence of these factors, we evaluated the potential interactive effects of residence in a low-income neighborhood and anxiety on the outcomes of interest. There was no evidence of an interaction between SES and anxiety in the model for treatment response. However, there was suggestive evidence of an interaction between SES and anxiety in the model for suicidal ideation (p=0.08). The results of the model for suicidal ideation (Table 2, Column 4) in which the SES*anxiety interaction was added points to a somewhat stronger effect of baseline anxiety symptoms on suicidal ideation among individuals in low-income neighborhoods (OR, 2.10; 95%CI, 1.31–3.35) than among individuals who lived in middle- and high-income (aggregated) neighborhoods (OR, 1.63; 95% CI, 0.52–5.15).

As in our previous research [1], educational status was not a significant predictor of either likelihood of response or odds of suicidal ideation in the regression models that included baseline comorbid anxiety.

Discussion

The objective of this study was to investigate whether SES has an independent effect (over and above anxiety) in predicting treatment response and suicidal ideation in depressed older adults. Identification of pre-treatment factors that are related to the prognosis of depression would aid clinicians in the recognition of patients at higher risk for treatment failure. However, research to date is inconclusive about whether the clinical implications of SES as a prognostic factor differ from the clinical implications of baseline clinical factors, e.g., severity of depressive symptoms and comorbid anxiety [45,46,26,27]. Research by Areán et al. [47] suggests that low-income older adults respond to collaborative care for depression to the same extent as their higher-income counterparts. Research by Gum et al. [20] suggests that achieving good response to treatment among low-income older adults with psychiatric comorbidity requires clinical case management or cognitive-behavioral group therapy to retain patients in treatment. Work by Miranda et al. [21] is congruent with these findings. It demonstrates that low-income, African-American young women who are depressed respond well to either medication or psychotherapy interventions, but that enrollment and retention in treatment require intensive outreach, reimbursement for transportation, and provisions for child care. Nevertheless, we are left with the question of whether late-life depression among low-income individuals requires more intensive treatments, different treatments, or both. Our previous work [1] does not provide an answer. Although lower-income older adults were less likely to respond to combination (medication plus psychosocial) treatment and more likely to experience suicidal ideation during treatment than higher-income subjects in the MTLD trials, we do not know whether greater intensity of treatment would have eliminated these differences.

Although the secondary analyses reported here do not answers these questions, either, our findings do suggest that the social worlds which put older adults at elevated risk of depression also act to reduce the effectiveness of antidepressant treatments. At the same time, we cannot dismiss the consequences of psychiatric comorbidity. Adding baseline anxiety to the original regression models did not reduce substantially the effects of neighborhood SES (census tract median household income) as a predictor of response to treatment. Nor did neighborhood SES reduce substantially the effects of baseline comorbid anxiety as a predictor. This suggests that neighborhood and clinical characteristics are independent predictors of response to treatment for late-life depression. At the same time, there is intriguing evidence of an interaction effect on the experience of suicidal ideation the negative consequences of comorbid anxiety may be amplified for individuals who live in low SES neighborhoods. This apparent association, which is broadly consistent with other research concerning the interactive effects of risk factors for depression [48–54] and suicidal ideation [55], indicates a need for more research on the interactions among the "emotional, physical, and social factors that determine risk for suicide in the older adults" [56].

In conclusion, we suggest that, identifying, disentangling, and addressing social and clinical prognostic factors should become a major focus of clinical research in psychiatry. To that end, we urge clinical trial investigators to follow the advice of Kraemer et al. [37,38] and conduct secondary data analyses to explore the social factors (e.g., income, wealth, education, race/ethnicity, characteristics of neighborhoods) that may act as moderators of treatment effects. However, such analyses will not be possible unless investigators: 1) employ sampling methods that ensure ample representation of individuals from a wide range of social worlds and ensure sufficient power in clinical trials to detect moderators of treatment efficacy [57]; and, 2) collect detailed data on the socioeconomic status of participants in clinical trials [58]. Together, sampling methods, data collection and analytic strategies to explore moderators of treatment effects will make it possible for clinical research to sort out the independent effects of comorbid symptoms and other clinical factors from the effects of SES.

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Table 1

Baseline demographic & clinical characteristics of the income groups: mean (SD) or % (n)

	<$25,000 N=40	$25,000–$50,000 N=158	≥$50,000 N=50	F or X^2	df	p
DEMOGRAPHICS						
Education (years)	12.5 (2.9)	12.5 (2.5)	13.2 (2.5)			
<12	30 (12)	21 (33)	16 (8)	1.66	2,244	.19
12	35 (14)	45 (71)	35 (17)	6.17	6	.40
13–15	15 (6)	18 (29)	24 (12)			
≥16	20 (8)	16 (25)	24 (12)			
Age (years)	71.8 (8.6)	71.4 (6.9)	71.9 (7.8)			
59–69	48 (19)	35 (56)	36 (18)	0.15	2,245	.86
70–75	15 (6)	38 (60)	34 (17)	7.65	4	.11
76+	38 (15)	27 (42)	30 (15)			
Sex: Women	70 (28)	76 (120)	74 (37)	0.61	2	.74
Race: Caucasian	80 (32)	94 (149)	96 (48)	10.42	2	.005
Marital Status						
Not Married	23 (9)	18 (28)	20 (10)			
Married	28 (11)	44 (70)	42 (21)	3.84	4	.43
Widowed	50 (20)	38 (60)	38 (19)			
BASELINE CLINICAL CHARACTERISTICS						
CIRS-G score	8.6 (3.1)	8.4 (3.8)	8.6 (3.6)	0.09	2,239	.91
1st vs. Recurrent Depression	80 (32)	76 (120)	76 (38)	0.31	2	.86
Age of onset	54.4 (19.4)	54.5 (19.2)	55.0 (19.2)	0.02	2,245	.98
Late onset ≥60	40 (16)	47 (75)	46 (23)	0.72	2	.70

	<$25,000 N=40	$25,000–$50,000 N=158	≥$50,000 N=50	F or X^2	df	p
Weeks of Current Episode[a]	43 (59) med=15	69 (162) med=25	42 (47) med=25	0.58	2,245	.56
Folstein MM	28.3 (2.6)	28.6 (1.8)	28.9 (1.3)	1.26	2,239	.28
HRSD 17 total	22.0 (4.0)	22.0 (4.1)	20.9 (4.2)	1.44	2,245	.24
Suicidality 2+	33 (13)	20 (32)	14 (7)	4.72	2	.09
BSI Anxiety	1.29 (1.01)	1.40 (0.90)	1.22 (0.87)	0.79	2,239	.45

[i] Natural log transformation prior to statistical comparison

TABLE 2

Effects of SES and comorbid anxiety on treatment response and suicidality

	1.	2.	3.	4.
	Treatment response[a]		Suicidality[b]	
	Separate analyses of SES and anxiety[c]	Joint analyses of SES and anxiety[d]	Separate analyses of SES and anxiety[c]	Joint analyses of SES and anxiety[d]
	HR (95% CI)	**HR (95% CI)**	**OR (95% CI)**	**OR (95% CI)**
Socioeconomic Status				
Median household income				
Low	Reference	Reference	Reference	Reference
Middle	1.80 (1.18– 2.75)	1.77 (1.16–2.71)	0.48 (0.27–0.94)	0.53 (0.29–0.96)
High	1.25 (0.76–2.05)	1.32 (0.80–2.19)	0.39 (0.16–0.94)	0.44 (0.19–1.04)
Education (years)				
<12	Reference	Reference	Reference	Reference
12	0.98 (0.66–1.45)	0.97 (0.65–1.44)	0.97 (0.48–1.99)	0.98 (0.50–1.92)
13–15	1.03 (0.65–1.65)	1.06 (0.65–1.70)	0.72 (0.36–1.47)	0.70 (0.35–1.41)
≥16	1.09 (0.67–1.60)	1.10 (0.68–1.78)	0.93 (0.40–2.15)	0.89 (0.42–1.89)
BSI anxiety score at baseline	0.74 (0.61–0.91)	0.73 (0.60–0.89)	1.56 (1.02–2.37)	1.45 (0.98–2.14)

[a] Cox regression models of the time to treatment response. All models control for demographic (age, gender, race/ethnicity, and marital status) and baseline clinical (recurrent depression, age at onset, duration of current episode, medical illness burden, and severity of depressive symptoms) factors.

[b] Repeated-measures generalized logit models of suicidal symptoms during treatment. All models control for demographic (age, gender, race/ethnicity, and marital status) and baseline clinical (recurrent depression, age at onset, duration of current episode, medical illness burden, and severity of depressive symptoms) factors.

[c] Results shown from two separate models, one with income and education (plus control variables), and the other with baseline anxiety symptoms (and control variables). The results for SES were reported previously by Cohen et al. [1] for treatment response and suicidality; results for anxiety reported by Andreescu et al. [22] for treatment response.

[d] Results of a single, fully adjusted model with all SES, anxiety, and control variables.