Factors Mediating the Effects of a Depression Intervention on Functional Disability in Older African Americans

Laura N. Gitlin, PhD, Sarah L. Szanton, PhD, CRNP,*† Jin Huang, MA, PhD,‡ and David L. Roth, PhD‡*

OBJECTIVES: To determine factors mediating the effects of a depression intervention for older African Americans on functional disability and, secondarily, whether functional improvements mediate intervention effects on depressive symptoms.

DESIGN: Structural equation modeling to examine mediators in a secondary analysis of a randomized trial with 4-month follow-up.

SETTING: Philadelphia region.

PARTICIPANTS: Community-dwelling African Americans (≥55) with depressive symptoms living in an urban area (N = 208).

INTERVENTION: Up to 10 one-hour sessions over 4 months conducted by licensed social workers who provided care management, referrals and linkages, stress reduction techniques, depression knowledge and symptom recognition, and behavioral activation techniques.

MEASUREMENTS: Main outcome was self-reported functional difficulty level for 18 basic activities. Mediators included depression severity (Patient Health Questionnaire), depression knowledge and symptom recognition, behavioral activation, and anxiety.

RESULTS: At 4 months, the intervention had positive effects on functional difficulty and all mediators ($P < .001$). Separate structural equation models indicated that two factors (reduced depressive symptoms (23.5% mediated) and improved depression knowledge and symptom recognition (52.9% mediated)) significantly mediated the intervention's effect on functional disability. Enhancing behavioral activation and decreasing anxiety were not found to mediate improvements in functional disability. The two significant mediators jointly explained 62.5% of the intervention's total effect on functional disability. Functional improvement was not found to mediate the intervention's effect on depressive symptoms.

CONCLUSION: This multicomponent depression intervention for African Americans has an effect on functional disability that is driven primarily by enhancing symptom recognition and decreasing depressive symptoms. Reduction of functional difficulties did not account for improvements in depressive symptoms. Nonpharmacological treatments for depressive symptoms that enhance symptom recognition in older African Americans can also reduce their functional difficulties with daily living activities.
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Key words: depression; functional disability; mediation analysis

Late-life depression is the most prevalent mental health condition in older adults. It is associated with poor quality of life, functional disability, and mortality.[1,2] To address depression, a wide range of nonpharmacological interventions such as problem solving and cognitive and behavioral activation therapies have been developed, tested, and proven efficacious in various clinical, community, and home settings.[3–10] This robust body of research has shown that interventions effectively reduce depressive symptoms and also afford other important benefits, including improvements in physical function.[11–13] However, it is unclear whether the multiple benefits of these interventions are related or largely distinct and the underlying mechanisms by which nonpharmacological interventions affect separate outcomes such as depressive symptoms and physical function.

Mediation analysis provides an analytical framework for evaluating factors that explain how an intervention may effect or cause changes in a given outcome.[14–16] It is useful for evaluating causal mechanisms or whether changes in another variable can explain a proportion of the effects of an intervention on an outcome. Nevertheless, with few exceptions, mediational processes have not been

From the *Center for Innovative Care in Aging, Johns Hopkins University; †School of Nursing, Johns Hopkins University; and ‡Division of Geriatric Medicine and Gerontology, Center on Aging and Health, Johns Hopkins University, Baltimore, Maryland.

Address correspondence to Laura N. Gitlin, Professor/Director, Center for Innovative Care in Aging, Johns Hopkins University, 525 North Wolfe Street Suite 316, Baltimore, MD 21205. E-mail: lgitlin1@jhu.edu

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examined in depression trials.[17,18] Previously, the effects of mediators of a home-based multicomponent intervention (Get Busy Get Better, Helping Older Adults Beat the Blues (GBGB), formerly referred to as Beat the Blues) on depressive symptoms were examined. Increasing behavioral activation, enhancing depression knowledge and symptom recognition, and decreasing anxiety each independently mediated a significant proportion of the intervention's effect on depressive symptoms and jointly explained more than 60% of the intervention's total effect on depressive symptoms. That these measures reflect the intervention's treatment components suggests that they work in concert and are mutually necessary for maximal benefits on depressive symptoms to occur.[19] It was also previously reported that the intervention not only reduced depressive symptoms, but also afforded important benefits in other outcomes, including functional disability, anxiety, depression knowledge and symptom recognition, and behavioral activation.[20]

This study systematically builds upon this previous work and is an effort to further the understanding of the interrelationships between depression and physical function in this population. The purpose was threefold: to examine whether the three previously identified mediators of depressive symptom severity (behavioral activation, depression knowledge and symptom recognition, anxiety) of GBGB also mediate the positive effects of the intervention on functional disability; to examine whether improvements in depressive symptoms explain treatment benefits on functional disability; and the reverse, to examine whether improving functional disability mediated a proportion of the treatment effects on depressive symptoms.

Previous research suggests reciprocal relationships between measures of mental health and functional disability, with each being a risk factor for and strongly associated with the other,[21–25] but the relationship between changes in depression and functional disability due to an intervention targeting depression is unknown. It is plausible that an intervention designed to reduce depressive symptoms affects functional disability by improving mood. With improved mood, participants may become more active and participate in daily activities with less difficulty. In this scenario, depression contributes to excess disability (e.g., disability in addition to that caused by underlying and possibly immutable biomedical factors or underlying impairments). The reverse is also plausible; improvements in physical functioning may contribute to better mood in addition to the intervention's direct effect on depressive symptoms. With improved physical function, participants may become more physically and emotionally activated, which in turn may contribute to improved mood. Understanding the mechanisms by which one factor affects the other in a depression intervention can enhance the design of future interventions and guide clinical expectations when implementing an evidence-based depression program.

Behavioral activation, depression knowledge and symptom recognition, and anxiety were considered as mediators of functional disability given their role in mediating treatment effect on depressive symptoms.[19] If each measure or a combination thereof explained treatment effects on functional disability, then it could be concluded that similar mechanisms account for disparate treatment outcomes in depression trials. If the opposite were the case, then specific factors that need to be present in a depression treatment to obtain different specified desired outcomes will have been identified.

METHODS

Study Sample and Procedures

As reported elsewhere[20,26] the sample was a convenience sample of participants recruited in two ways: from a short-term in-home support program for medically compromised individuals who were routinely screened for depressive symptoms and offered information about the study and other mental health services if screened positive and from the community, using media announcements and presentations at local events and social agencies. Two hundred eight English-speaking, cognitively intact (Mini-Mental State Examination score >24) African Americans aged 55 and older who scored 5 or higher on the Patient Health Questionnaire (PHQ-9),[27] a measure of depressive symptoms, on two sequential testing occasions over 2 weeks were enrolled in the trial from these two sources. Individuals with a history of serious mental illness, with a life-limiting illness, who were enrolled in another depression trial, or who were living in assisted living or nursing home facilities were ineligible. Antidepressant medication use did not preclude study participation.

Analyses for this study were based on 177 participants (85%) with complete baseline and 4-month follow-up data for the variables of interest in the mediation analyses. Of the 31 participants with incomplete data, 29 did not complete the 4-month follow-up (missing depressive symptom severity or functional disability scores). A comparison of 177 participants included in the analyses with 31 who were not revealed no large or statistically significant differences at baseline in basic characteristics, mediators, depressive symptom severity, or functional disability (all $P > .05$).

Participants recruited from the two venues (in-home support group, community) differed at baseline in certain characteristics as anticipated. Participants from the in-home group (n = 51) were older; had more pain and health conditions; and were more functionally disabled, less educated, and less likely to be employed (all $P < .05$) than those in the community group (n = 126). The groups did not statistically differ in measures of mediation. Because a stratified randomization scheme based on recruitment source (in home vs community) was used, the intervention and control groups were comparable at baseline. There were no large or statistically significant differences at baseline between in-home participants assigned to intervention and control groups or between community participants assigned to intervention and control groups.

Those eligible and willing to participate in the trial provided written consent using an approved institutional review board form, completed a baseline home interview, and were randomized to receive the GBGB program immediately (treatment condition) or 4 months later (wait-list control). All participants were reassessed at 4 and 8 months at home using the same interview battery

conducted by assessors masked to participant group allocation.

Intervention

The intervention has been described elsewhere.[19,20,26] It involved up to 10 one-hour sessions at home conducted over 4 months by licensed social workers trained in the protocol. Interventionists assessed for unmet care management needs; developed a plan of action including referral and linkage to formal care and community and social services; provided education about depression including linking behavior and mood and helping participants learn how to identify their own symptoms, use specific strategies to manage symptoms early on, and talk about symptoms with physicians from different cultures and race; introduced different stress reduction techniques such as basic deep breathing; and introduced a behavioral activation process. The latter involved reviewing daily routines and mood fluctuations and then helping participants select behavioral goals and specific activities to add pleasure and personal satisfaction to routines. All participants received five treatment components (care management, referral and linkage, depression knowledge and symptom recognition, stress reduction, and behavioral activation), but the content of each component was customized to the participants' own needs, level of understanding, and behavioral goals.

The wait-list control group did not receive intervention contact between baseline and 4 months.

Measures

Research staff masked to group allocation administered all measures in this study, which reflected self-report of participants. Background characteristics included marital status (not married vs married or living as married); living arrangement (alone vs with others); sex; education (< high school, high school, > high school level of education); age; financial difficulty (0 = not very difficult to 3 = very difficult paying for basics); employment (employed vs unemployed); number of health conditions; and use of depression, anxiety, or pain medications (yes vs no).

The initial primary dependent variable was functional disability measured according to 18 items reflecting mobility and instrumental and basic activities of living.[28] Participants rated their level of difficulty (1 = no difficulty to 5 = unable to do because of health problem) with each item in the past month. A mean score was derived by summing across scores and dividing by the number of items, with lower scores indicative of fewer functional difficulties (Cronbach α for sample = .91). In secondary analyses, the functional disability index was evaluated as a mediator of the treatment effect on depressive symptoms at 4 months.

Indicators of Mediation

Depressive symptoms were assessed using the PHQ-9, a brief, psychometrically sound, 9-item self-report measure of severity of depression.[27] Because the scale was administered on two subsequent occasions 2 weeks apart to determine eligibility, the second screen was used as the baseline value. A total severity score was calculated by summing responses of nine items rated as occurring not at all (0), on several days (1), on more than half the days (2), or nearly every day (3). Possible scores ranged from 0 to 27 (α = .78 for sample).

Depressive symptom severity was initially examined as a mediator of treatment effect on functional disability, and then secondarily as an outcome measure to evaluate whether functional disability mediated treatment effects. Other measures of mediation included depression knowledge and symptom recognition, state anxiety, and behavioral activation.

Depression knowledge and symptom recognition was assessed using 10 items reflecting symptom awareness (can identify depression symptoms) and efficacy (know how to explain feelings, make self feel better by increasing activities) rated from 1 (not at all confident) to 4 (absolutely confident). A total mean score was derived by summing across items and dividing by the number of items (range 1–4). Higher scores represented greater knowledge or efficacy (α = .72 for sample).[29]

Anxiety was measured using the 10-item State Anxiety Scale, in which feelings ("I felt calm," "tense") are rated from 1 (very much) to 4 (not at all). A total anxiety score was computed as the mean of all items (α = .85 for sample). Higher scores indicated greater anxiety.[30]

Behavioral activation was assessed using a modification of the Behavioral Activation Scale. The modification involved eliminating 14 items (My work or schoolwork suffered…) not relevant to this sample and rewording three items to heighten their relevance. Participants rated the resulting 17 items from 0 (not at all) to 6 (completely). Items reflected positive engagement (accomplished goal, engaged in activities, did things even though hard because fit with goals), avoidance of difficult situations (there were certain things that I needed to do that I didn't do), or dwelling on negative feelings (spent time thinking about my past, people who have hurt me, mistakes I've made). A total activation score was computed as the mean of items (α = .83 for sample). Higher scores indicated greater activation.[31]

Statistical Analysis

The primary aim of the analyses was to determine whether improvements in four mediating variables (depressive symptom severity, depression knowledge and symptom recognition, anxiety, behavioral activation) independently and jointly mediated the beneficial effect of the intervention on functional difficulties at 4 months. A secondary aim was to determine whether improvements in functional difficulties mediated the beneficial effect of GBGB on depressive symptoms at 4 months.

For analyses, simple change scores (4-month minus baseline) were calculated for mediators (depressive symptom severity, depression knowledge and symptom recognition, anxiety, behavioral activation) and functional difficulties.[16] These change scores were then analyzed as a function of their baseline values and the intervention effect (Figure 1). All mediation analyses were conducted using Mplus Version 7 (Muthén & Muthén, Los Angeles, CA). The mediation models included only the corresponding baseline measurement of the mediator being investigated.

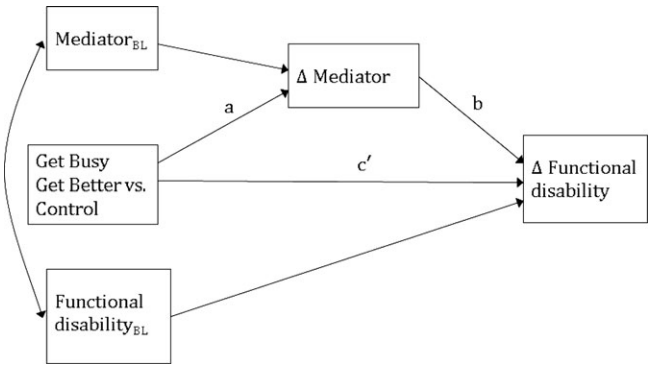

Figure 1. Two-wave mediation model used to examine mediators individually. BL = baseline observation. Δ = 4-month score minus baseline score.

No other covariates were included in analyses. The mediated or indirect effect represents the joint effect of the "*a*" and "*b*" paths on the outcome, and is estimated as "$a \times b$." The standard error of the $a \times b$ mediation effect was calculated using the Sobel method.[32] The c' path represents the unmediated or direct effect, and the sum of $a \times b$ and c' comprises the total (baseline-adjusted) effect of intervention on functional disability. These estimates were tested for statistical significance and used to estimate the proportion of the total effect that could be attributed to that mediator ($(a \times b)/((a \times b) + c')$).

After mediators were examined individually, those identified as statistically significant were examined simultaneously to test for independent versus overlapping mediation effects. The c' from those models represented any intervention effect on functional disability that was independent of the other mediating mechanisms and was used to calculate a jointly mediated proportion.

RESULTS

Participants were all African Americans with an average age of 69.5 ± 8.6 (range 55.6–96.1). Most were female (N = 140, 79.1%), had a high school education or more (N = 137, 77.4%), were not employed (N = 163, 92.1%), were not married (N = 155, 87.6%), and reported financial difficulties (N = 119, 67.2%). Participants reported an average of 6.6 ± 3.1 health conditions, and 91 (51.7%) were taking pain management medications. Most were not taking medications for mood; 37 (21.02%) reported taking an antidepressant and 28 (15.9%) reported taking an anti-anxiety medication.

At baseline, participants reported some functional difficulties (mean 1.96 ± 0.66) and moderate depressive symptomatology (mean 12.9 ± 4.9).

For other mediators, participants reported some to moderate anxiety (mean 2.52 ± 0.65), some confidence recognizing symptoms (mean 3.10 ± 0.43), and low activation (mean 2.89 ± 1.00) (Table 1). There were no differences between the intervention and control groups in background characteristics, mediators, or outcome measures at baseline.

Table 2 displays the correlations between variables analyzed in the mediation models. As expected, there were significant associations between the different mediating variables at baseline and for 4-month change scores.

Effects of GBGB on Dependent Variables and Mediators

As found in the main trial analyses[20] and using standard analyses of covariance with the baseline score as the covariate, GBGB had significant effects on all measures, including functional disability, and four mediators (depressive symptoms, behavioral activation, depression knowledge and symptom recognition, anxiety; all $P < .001$). For each, improvements were observed in the GBGB group that exceeded changes in the wait-list control group. Estimates for these effects on the mediators constitute the "a" paths in the mediation models (Table 3).

Single-Mediator Models for Functional Disability

Table 3 shows unstandardized estimates that correspond to the paths illustrated in Figure 1 for each combination of mediator and functional disability measure. These findings indicate highly significant mediation effects for depressive symptoms ($P < .05$) and depression knowledge and symptom recognition ($P < .01$). The proportion that depressive symptoms mediated was 23.5%, whereas the proportion that depression knowledge and symptom recognition mediated was twice that (52.9%). The analysis that examined whether changes in functional disability mediated changes in depressive symptoms did not reveal a statistically significant mediation effect.

Multiple-Mediator Models for Functional Disability

The single-mediator models with functional disability as the outcome variable indicated that changes in two of four tested mediators explained a portion of the intervention's effect on functional disability. As shown in Figure 2, when examining these two mediators simultaneously, the unmediated or direct effect of the intervention was no longer statistically significant ($\beta = -0.06$, $P = .35$), whereas the joint mediated effects explained 62.5% of the interventions' total effect on depressive symptoms. A comparison of the mediation effects ($a \times b$) indicated that changes in depression knowledge had a slightly stronger independent mediation effect than changes in depressive symptoms, but the difference was not statistically significant ($P = .13$).

DISCUSSION

This study is the first to the knowledge of the authors to examine whether reduction in depressive symptom severity from a nonpharmacological intervention would extend to improvements in functional disability through the specific pathways identified in a mediation model. Whether changes in functional disability mediated the relationship between intervention participation and effect on depressive symptoms was further examined. The analysis focused on older urban African Americans with significant health, pain, and financial concerns. Consistent with the findings for the entire sample of 208,[20] GBGB had significant positive effects on all measures; GBGB reduced functional

Table 1. Baseline Characteristics of Analytical Sample

Characteristic	Total Sample, N = 177	Treatment Group, n = 84	Control Group, n = 93	*P*-Value
Age, mean ± SD	69.5 ± 8.6	69.1 ± 8.7	69.8 ± 8.5	.59
Sex, n (%)				
Male	37 (20.9)	17 (20.2)	20 (21.5)	.84
Female	140 (79.1)	67 (79.8)	73 (78.5)	
Education, n (%)				
< High school	40 (22.6)	18 (21.4)	22 (23.7)	.34
High school or General Educational Development certificate	52 (29.4)	21 (25.0)	31 (33.3)	
> High school	85 (48.0)	45 (53.6)	40 (43.0)	
Employment status, n (%)				
Employed	14 (7.9)	10 (11.9)	4 (4.3)	.06
Unemployed	163 (92.1)	74 (88.1)	89 (95.7)	
Paying for basics, n (%)				
Not difficult at all	36 (20.3)	12 (14.3)	24 (25.8)	.20
Not very difficult	22 (12.4)	13 (15.5)	9 (9.7)	
Somewhat difficult	68 (38.4)	32 (38.1)	36 (38.7)	
Very difficult	51 (28.8)	27 (32.1)	24 (25.8)	
Marital status, n (%)				
Not married	155 (87.6)	70 (83.3)	85 (91.4)	.10
Married	22 (12.4)	14 (16.7)	8 (8.6)	
Number of health conditions, mean ± SD	6.6 ± 3.1	6.8 ± 2.8	6.4 ± 3.3	.45
Antidepressant medication, n (%)	37 (21.0)	21 (25.0)	16 (17.4)	.22
Anxiety medication, n (%)	28 (15.9)	11 (13.1)	17 (18.5)	.33
Pain medication, n (%)	91 (51.7)	43 (51.2)	48 (52.2)	.90
Functional disability, mean ± SD	2.0 ± 0.7	2.0 ± 0.7	2.0 ± 0.7	1.00
PHQ-9 score (second screen), mean ± SD	13.0 ± 4.9	13.1 ± 5.2	12.8 ± 4.7	.61
PHQ-9 score (second screen), n (%)				
0–4 (minimal or no depression)	0 (0.0)	0 (0.0)	0 (0.0)	.40
5–9 (mild depression)	52 (29.4)	25 (29.8)	27 (29.0)	
10–14 (moderate depression)	63 (35.6)	30 (35.7)	33 (35.5)	
15–19 (moderate to severe depression)	41 (23.2)	16 (19.1)	25 (26.9)	
≥20 (severe depression)	21 (11.9)	13 (15.5)	8 (8.6)	
Behavioral activation, mean ± SD	2.9 ± 1.0	2.9 ± 1.0	2.9 ± 1.0	.57
Depression knowledge, mean ± SD	3.1 ± 0.4	3.1 ± 0.5	3.1 ± 0.4	.43
Anxiety, mean ± SD	2.5 ± 0.7	2.5 ± 0.7	2.5 ± 0.6	.88

SD = standard deviation; PHQ-9 = Patient Health Questionnaire.

disability, depressive symptoms, and anxiety and enhanced depression knowledge and symptom recognition and behavioral activation, important outcomes for this group indicating overall improved quality of life.

The present mediation analyses along with earlier findings[19] provide an understanding of the pathways by which GBGB afforded positive effects on two distinct outcomes: depressive symptoms and functional difficulties with daily living activities. Improvements in two of the four mediators examined (change in depressive symptoms and depression knowledge and symptom recognition) mediated a substantial proportion of GBGB's effect on functional disability. When effects of the two mediators were examined jointly, the direct effects of GBGB on functional disability were minimized, suggesting that the intervention had its effects by improving the ability of participants to understand depression and recognize and actively manage symptoms coupled with decreasing depressive symptoms, with the former having a greater effect on functional outcomes. These two factors appear to be necessary conditions to affect functional disability for this sample in this intervention. Although enhanced behavioral activation and lessened anxiety did not affect changes in physical function, these factors mediated GBGB effects on depressive symptoms. The only mediator in common for improvements in function and depressive symptoms was enhanced knowledge about depression and confidence in being able to recognize and act on symptoms. This suggests that providing depressed older African Americans with education and strategies for preventing and managing symptoms has a dual benefit—reducing depressive symptoms and functional disability. The findings also suggest that slightly different pathways may account for dissimilar outcomes of nonpharmacological depression treatments.

Why would improvements in symptomatology and depression knowledge and symptom management versus other positive changes account for observed reductions in functional disability? At baseline, functional disability scores were significantly correlated with each of the mediators (all $P < .05$), such that having more disability was associated with higher depressive symptom and anxiety scores and lower knowledge about depression, efficacy in symptom management, and behavioral activation. These relationships are consistent with findings from other studies of persons with functional limitations[33,34] showing that disability is associated with quality-of-life domains and mood decrements, yet it appears that a critical pathway for addressing functional challenges in this depressed

Table 2. Summary of the Relationship Between Mediators at Baseline and 4 Months (N = 177)

		Baseline					Change at 4 Months				
Mediator	Group	Functional Disability	PHQ-9	Depression Knowledge	Behavioral Activation	Functional Disability	Functional Disability	PHQ-9	Depression Knowledge	Behavioral Activation	Anxiety
Mean ± SD		1.96 ± 0.66	12.93 ± 4.94	3.10 ± 0.43	2.89 ± 1.00	2.52 ± 0.65	−0.14 ± 0.48	−5.21 ± 6.04	0.25 ± 0.49	0.62 ± 1.18	−0.33 ± 0.69
Baseline											
Functional disability	0.00										
PHQ-9	0.04	0.20[b]									
Depression knowledge	−0.06	−0.45[c]	−0.21[b]								
Behavioral activation	0.04	−0.21[b]	−0.27[c]	0.42[c]							
Anxiety	−0.01	0.19[a]	0.35[c]	−0.29[c]	−0.50[c]						
Change at 4 month											
Functional disability	−0.18[a]	−0.42[c]	−0.06	0.24[b]	0.14	−0.07					
PHQ-9	−0.22[b]	0.17[a]	−0.50[c]	−0.05	0.00	0.00	0.17[a]				
Depression knowledge	0.34[c]	0.04	0.08	−0.52[c]	−0.16[a]	0.08	−0.34[c]	−0.34[c]			
Behavioral activation	0.27[c]	−0.06	0.05	−0.13	−0.54[c]	0.18[a]	−0.15	−0.30[c]	0.51[c]		
Anxiety	−0.22[b]	0.11	−0.05	0.05	0.11	−0.50[c]	0.06	0.36[c]	−0.37[c]	−0.45[c]	

[a] $P < .05$, [b] $.01$, [c] $.001$.
PHQ-9 = Patient Health Questionnaire.

group is to enhance their ability to self-manage symptoms —that is, to recognize and then feel more efficacious in their ability to adopt positive coping strategies such as engaging in meaningful activities. Previous research has not considered how managing mood disturbances factors into the disablement process, but mood may be easier to intervene on than other contributing factors to disability.

Having depressive symptoms and not knowing how to identify and manage them may represent what is referred to as excess disability. That is, it may make performing everyday activities of living more challenging. Because GBGB minimized both of these sources of disability, participants reported improved daily function.

Despite reciprocity between depressive symptoms and functional disability, for this sample, reducing functional challenges did not account for the effect of GBGB on reductions in depressive symptoms. Thus, although minimizing disability is an important consequence of depression treatments, it is not the pathway through which the intervention has its effects on depression severity. Rather, as reported elsewhere, other factors, namely, improving depression knowledge and symptom recognition, reducing anxiety, and enhancing behavioral activation levels, mediated the effects of the intervention on depressive symptoms. One explanation may be that this sample reported only "some" difficulties with daily activities, such that physical functioning was not the primary source of their depressive symptoms.

Taken as a whole, the findings of the current study show that different mechanisms may account for the positive benefits of depression treatments on distinct outcomes. Although the main outcome for depression treatments should be depression, other secondary treatment benefits are important as well, particularly given the interconnectedness of depressive symptoms, functioning, and well-being. Also, there is efficiency in a treatment if multiple outcomes can be realized. These results suggest that, for a depression intervention to affect daily functioning, individuals need to be provided with the knowledge and skills to manage their depressive symptoms actively. Knowledge and symptom recognition enhancements also improved depressive symptoms, so efficiency in intervention delivery in this respect was achieved, although other factors also accounted for the reduction in depressive symptoms and thus are necessary intervention ingredients.

A competing hypothesis is that participants in the treatment group may have become more sensitized to their depressive symptoms and consequently sought other treatments that account for the positive changes reported here. However, at 4 months, there were no large or statistically significant differences between the intervention and control groups as to their formal care and service use, and there was a slight decline in antidepressant use in the treatment group, as reported elsewhere.[35]

Given that no studies to the knowledge of the authors have examined mediators of depression interventions on functioning, it is unclear how the current results compare with those of other depression treatments. A recent trial to test the efficacy of behavioral activation to prevent depression in individuals with age-related macular degeneration and subsyndromal depressive symptoms found that the risk of depression was significantly lower for the treat-

Table 3. Summary of Single-Mediator Models on Functional Disability and Depressive Symptoms (N = 177)

Outcome	Mediator	*a*	*b*	*ab*	*c′*	*ab*/(*ab* + *c′*)
Functional disability	PHQ-9	−2.48[b]	0.02[b]	−0.04[a]	−0.13[a]	0.24
	Depression knowledge	0.31[c]	−0.29[c]	−0.09[b]	−0.08	0.53
	Behavioral activation	0.70[c]	−0.06	−0.04	−0.14[a]	0.22
	Anxiety	−0.32[c]	0.05	−0.02	−0.16[a]	0.11
PHQ-9	Functional disability	−0.18[b]	1.36	−0.24	−2.24[b]	0.10

$P <$ [a].05, [b].01, [c].001.
PHQ-9 = Patient Health Questionnaire.

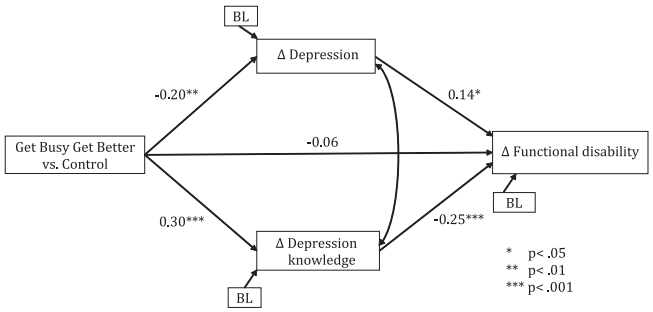

Figure 2. Multiple-mediator model of intervention effect on change in functional disability. BL = baseline observation. Δ = 4-month score minus baseline score.

ment group, although there were no changes in functional disability level, and staying socially engaged appeared to mediate or account for treatment benefits. These findings suggest that the reduction of depression risk may not affect daily function and that the pathways for improvements may differ according to study population.[36] Mediation analyses are an important analytical strategy for identifying the mechanistic variables or pathways by which treatments have their effects. This analytical framework should be applied in future depression trials to more fully comprehend how such interventions achieve their benefits because this may differ according to outcome, targeted population, and type of depression treatment.

These findings should be understood within the context of several limitations. First, the sample was a convenience sample and smaller than those in large multisite depression clinical trials. Nevertheless, this study compares favorably with intervention studies conducted in single community sites, which depend upon samples of convenience and with sample sizes less than 600.[3] Because men were underrepresented, it is not possible to determine whether pathways are different according to sex, as previous research suggested.[37] Because men and women experience depression differently, examining sex effects could help to refine treatment components and customize messaging.[38] Second, the sample represents one minority group and was recruited from one region, possibly limiting its generalizability to other minority groups or locations. Third, this study used a brief screen for depression, the PHQ-9, and the focus was on depressive symptoms. Future research examining depression disorders and using substantive clinical evaluations of depression would be important. Finally, at 4 months, the wait-list control group received the intervention. Thus, it was not possible to examine whether mediational effects at 4 months endured at 8 months.

Despite limitations, these findings contribute to understanding the value of nonpharmacological depression treatments and the mechanisms by which they achieve positive effects for a study population not well represented in previous depression trials. Older African Americans are at greater risk of depression than whites because of their higher rates of chronic illness, functional disability, health problems, and exposure to multiple social structural jeopardies, including financial strain and poor neighborhood quality, all risk factors for depression.[39,40] Because prevalence of depressive symptoms in older African Americans may be higher than previously considered (as high as 31%) for homebound or medically comprised individuals,[36–38] and because this group does not customarily receive depression care (detection and treatment) in primary care, identifying different models of mental health support is paramount for addressing persistent disparities. GBGB is a step in the right direction. The findings presented here and elsewhere support the importance of its replication and translation so that it is a deliverable service alternative for this underserved group.

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Author Contributions: Gitlin L.N.: principal investigator, developed study concept and design including intervention, oversaw scientific integrity and interpretation of data, primary responsibility for preparation of manuscript. Szanton S.L.: assisted in interpretation of study results and reviewed manuscript for accuracy. Huang J.: conducted all data analyses, constructed manuscript tables, assisted in

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