ORIGINAL ARTICLE

Preventing Depression in Age-Related Macular Degeneration

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Context: Age-related macular degeneration is a prevalent disease of aging that may cause irreversible vision loss, disability, and depression. The latter is rarely recognized or treated in ophthalmologic settings.

Objective: To determine whether problem-solving treatment can prevent depressive disorders in patients with recent vision loss.

Design: Randomized, controlled trial.

Setting: Outpatient ophthalmology offices in Philadelphia, Pennsylvania.

Patients: Two hundred six patients aged 65 years or older with recent diagnoses of neovascular age-related macular degeneration in one eye and pre-existing age-related macular degeneration in the fellow eye.

Intervention: Patients were randomly assigned to problem-solving treatment (n=105) or usual care (n=101). Problem-solving treatment therapists delivered 6 sessions during 8 weeks in subjects' homes.

Main Outcome Measures: Outcomes were assessed at 2 months for short-term effects and 6 months for maintenance effects. These included *DSM-IV*–defined diagnoses of depressive disorders, National Eye Institute Vision Function Questionnaire–17 scores, and rates of relinquishing valued activities.

Results: The 2-month incidence rate of depressive disorders in problem-solving–treated subjects was significantly lower than controls (11.6% vs 23.2%, respectively; odds ratio, 0.39; 95% confidence interval, 0.17-0.92; $P=.03$). Problem-solving treatment also reduced the odds of relinquishing a valued activity (odds ratio, 0.48; 95% confidence interval, 0.25-0.96; $P=.04$). This effect mediated the relationship between treatment group and depression. By 6 months, most earlier observed benefits had diminished, though problem-solving treatment subjects were less likely to suffer persistent depression ($\chi^2_{1,3}=8.46$; $P=.04$).

Conclusions: Problem-solving treatment prevented depressive disorders and loss of valued activities in patients with age-related macular degeneration as a short-term treatment, but these benefits were not maintained over time. Booster or rescue treatments may be necessary to sustain problem-solving treatment's preventative effect. This study adds important new information to the emerging field of enhanced-care models to prevent or treat depression in older persons.

Trial Registration: clinicaltrials.gov Identifier: NCT00042211

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DEPRESSION ACCOMPANIES many medical and neurological disorders in older persons and increases their risk of disability and death.[1] Because it is often considered an inevitable consequence of disease, preventing depression may seem improbable. Such an effort is necessary, however, because depression is a common, costly disorder that exaggerates disability and is often unrecognized and untreated in medical settings.[2-4] Current efforts to reduce depression's public health impact, therefore, have emphasized prevention.[5]

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There have been few clinical trials of interventions to prevent depression in older adults with complicated medical problems. Most have targeted poststroke depression using pharmacologic interventions.[6] Age-related macular degeneration (AMD) is another serious medical problem with a high incidence rate of depression. It provides an appealing model for depression prevention research because its severity is readily quantified, it affects men and women equally, and has no biological symptoms that overlap with depression and therefore avoids the problem of confounding behavioral outcomes with underlying disease symptoms.

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Age-related macular degeneration is the most common cause of legal blindness in the United States and affects almost 10 million people.[7,8] It is caused by deterioration of the macular region of the retina and can lead to various pigment changes, such as geographic atrophy (a dry form of AMD) and choroidal neovascularization (neovascular, or wet, AMD).[9] Patients with bilateral AMD are often overwhelmed by their newfound difficulty of engaging the world and are frustrated by the challenge of remaining independent. As a result, many develop persistent depression and suffer further functional decline.[10] We have found, for example, that almost 30% of patients with bilateral AMD will develop a depressive disorder within a few months of their second eye becoming affected.[11]

This article reports results from the Preventing Depression in AMD Trial, a randomized, controlled clinical trial that compared the efficacy of problem-solving treatment (PST) with usual care in preventing depressive disorders in patients with AMD. Problem-solving treatment is a behavioral treatment that teaches problem-solving skills and posits that inaccurate appraisals of problems and difficulty deriving and implementing adaptive solutions can lead to depression.[12]

We tested the hypothesis that PST would reduce the incidence of depressive disorders at 2 months (short-term effects) and 6 months (maintenance effects) after implementation compared with usual care and, secondarily, that it would improve subject ratings of vision function and prevent loss of valued activities.

METHODS

SAMPLE RECRUITMENT

We recruited subjects from the retinovitreous clinics of Wills Eye Hospital in Philadelphia, Pennsylvania. The inclusion criteria were being older than 64 years and having neovascular AMD in one eye diagnosed within the preceding 6 months and preexisting AMD in the fellow eye. We chose these parameters to identify nondepressed patients with recent bilateral visual impairment who were at high risk for depression. These risk characteristics define a selective preventive intervention, which, according to the Institute of Medicine's recent classification of preventive interventions,[5] targets a population subgroup whose biological, psychological, or social characteristics increase their risk of disorder. By contrast, an indicated preventive intervention targets higher-risk persons who already have detectable signs of disorder but do not meet diagnostic criteria. For this trial, we excluded patients with *DSM-IV*–defined diagnoses of depressive disorders or current treatment for depression. Many such patients were excluded prior to the baseline assessment based on medical record evidence of depression. This accounts for the low rate of depression at the baseline assessment. We also excluded patients with cognitive impairment or confounding eye conditions. Thomas Jefferson University's institutional review board approved the informed consent form and procedures to access medical records to identify eligible subjects.

To obtain the sample, we reviewed ophthalmologists' dictated reports of fluorescein angiograms, confirmed diagnoses with treating ophthalmologists, and contacted potential subjects. They were patients who had previously signed a document permitting review of their medical records for research purposes. This method of ascertainment was objective and verifiable, avoided relying on ophthalmologists to identify eligible cases, and prevented other selection biases. We used a random-numbers table, sealed envelopes containing treatment assignments, and a fixed randomization scheme with a 1:1 allocation ratio to assign subjects to 1 of the 2 study groups. We used a permuted random block design to ensure balance between treatment groups according to their time of patient enrollment. Block sizes (4 or 6) were chosen at random to mask the blocking process. The outcome assessors were unaware of subjects' treatment assignment in this single-masked study.

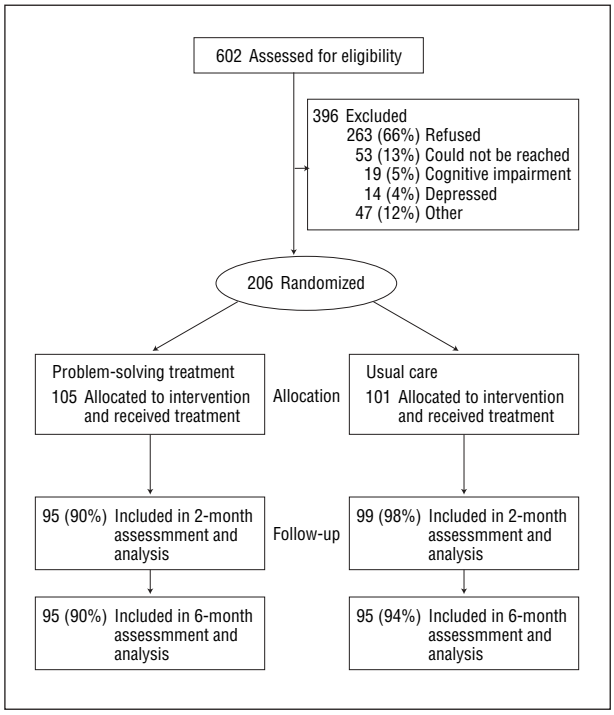

Figure. Patient flowchart.

The **Figure** depicts the sample's enrollment, randomization, and assessment. From December 2001 to July 2005, we identified 602 potentially eligible patients; of these, 206 (34.2%) were randomized to the PST (n=105) or usual care (n=101) group. The major reasons for nonparticipation (n=396) were refusal (n=263 [66%]); inability to contact patient (n=53 [13%]); cognitive impairment (n=19 [5%]); depression (n=14 [4%]); and other reasons (n=47 [12%]). Nonparticipating patients were similar to enrolled subjects in their demographic characteristics, visual acuity, and responses to a screening measure of depression. During the trial, 17 (8.3%) subjects dropped out of the study (11 PST subjects and 6 controls). In the PST group, 9 cited loss of interest for dropping out; 1 was too ill; and 1 died. They did not differ significantly from retained subjects on any clinical or demographic characteristics that would suggest an increased risk of depression (data not shown). Among controls, 3 cited loss of interest, 2 were ill, and 1 died.

PST VS USUAL CARE INTERVENTIONS

Problem-solving treatment is a manual-driven psychological treatment that teaches problem-solving skills.[11] It addresses negative perceptions that may interfere with finding practical solutions to problems and teaches the following problem-solving skills: (1) defining problems, (2) establishing realistic goals, (3) generating, choosing, and implementing solutions, and (4) evaluating outcomes. Subjects are encouraged to use

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these skills routinely to develop practical compensatory strategies to achieve valued functional goals and thereby prevent depression.

Subjects randomized to both PST and usual care continued to receive treatment as usual from their ophthalmologists or other health care providers. Usual care subjects were offered PST once the clinical trial was completed. During the trial, no subjects in either treatment group received outside specialty mental health treatment. There were no statistically significant differences in the proportions of subjects (PST vs usual care, respectively) who received low-vision rehabilitation (35.1% vs 34.7%; $P=.96$), used optical devices (80.9% vs 78.9%; $P=.74$), or were treated with antidepressant medications (3.2% vs 3.2%; $P>.99$).

PST TRAINING AND FIDELITY ASSESSMENT

Problem-solving treatment–trained therapists (2 nurses and 1 master's-level counselor) delivered 6 in-home PST sessions (45-60 minutes long) during 8 weeks to subjects randomized to PST. All therapists received extensive training by M. T. H., which included reviewing the PST treatment manual, watching training videotapes, and treating 5 practice patients. To maintain treatment fidelity, all PST sessions were audiotaped; M. T. H. reviewed one-third of randomly selected tapes during the trial and rated therapists on key problem-solving steps, communication skills, and efficient use of time.[13] All items were rated on a 6-point scale (0=very poor, 5=very good), with scores higher than 3 indicating a satisfactory performance. Mean scores across therapists ranged from 3.70 (SD, 1.30) for implementing decision-making guidelines to 4.97 (SD, 0.24) for interpersonal effectiveness.

ADEQUACY OF MASKING

Personnel masked to treatment assignment completed central data collection, measurement, and data entry. Only the project director, statistician, and PST therapists were aware of treatment assignment. Following randomization, the research nurses instructed all subjects on the purpose and importance of masked treatment assignment and requested that subjects not reveal any information about their study participation. We assessed rates of breaches in masking and found that unmasking occurred in 26 PST and 11 control subjects (18.0%). In all instances, subjects inadvertently revealed their treatment assignment. We compared depression rates in unmasked and masked subjects to determine whether unmasking influenced rates of diagnosing depression and found no difference ($P=.80$).

ASSESSMENTS

Research nurses with extensive training in psychiatry and ophthalmology obtained informed consent and completed all assessments in subjects' homes. Demographic characteristics included age, race, sex, years of education, and marital status.

The primary outcome was a *DSM-IV*–defined diagnosis of major or minor depression. The research nurses administered the modified Schedule for Affective Disorders and Schizophrenia and the Structured Interview Guide for the Hamilton Depression Rating Scale (HDRS) to rule out depression at baseline, to obtain history of depression treatment, and to diagnose a depressive disorder at 2 and 6 months.[14,15] Interrater reliability for nurse ratings was established ($\kappa=0.96$). We also used the 24-item HDRS to quantify depressive symptoms. Possible scores ranged from 0 to 75, with higher scores indicating more severe depression. Scores less than 7 are considered normal.[15]

To provide objective measures of vision, we assessed best-corrected distance visual acuity and contrast sensitivity in each eye separately using the Lighthouse Ferris-Bailey Early Treatment Diabetic Retinopathy Study chart and the Pelli-Robson contrast sensitivity chart, respectively.[16,17] Scores were converted to the logarithm of the minimum angle of resolution (logMAR) and log contrast, with higher scores indicating greater impairment.[18]

To provide a subjective measure of vision disability (ie, self-reported difficulty level on vision-dependent daily activities), we used the National Eye Institute Vision Function Questionnaire (NEI VFQ-17), a subset of the original NEI VFQ-52 that conforms to an interval scale.[19,20] The items rate difficulty of completing tasks, such as reading newsprint and pursuing hobbies, and are scored on a 5-point scale ranging from no difficulty to stopped a task due to impaired vision (relinquished activity). Scores were summed (range, 17-85), with higher scores indicating worse function. Subjects rated the personal value of all tasks.

Medical comorbidity was assessed using the Chronic Disease Score, which is derived from a weighted sum of medications taken for chronic illness.[21] The score predicts health care use, costs, hospitalization rates, and mortality, with higher scores indicating greater medical disease burden.[22]

STATISTICAL ANALYSIS

We based the sample size calculation on data showing a 30% incidence rate of depression in patients with recent vision loss due to AMD.[10] We hypothesized that PST would reduce that rate by 50%. To detect this difference with 80% power and α set at 0.05 (1-tailed), 95 subjects were required per group. Efficacy analyses were planned at 2 months to assess short-term effects and at 6 months to assess maintenance effects.

To assess the adequacy of randomization, we compared PST and usual care subjects on demographic and baseline clinical characteristics using χ^2 for categorical data and 1-way analyses of variance for continuous data. To compare PST with usual care, we employed a modified intent-to-treat approach, with subjects remaining in their assigned groups for all analyses but, because not all subjects reached study end points, outcome analyses were based on available cases.

Differences between treatment groups on categorical outcomes (depressive disorder and relinquishing valued activities) were analyzed using a logistic regression model with terms for treatment, time, and their interaction after adjusting for significant covariates. Two baseline variables—higher (worse) NEI VFQ-17 and HDRS scores—independently predicted depressive disorders and were included as covariates (both $P<.05$). Generalized estimating equation methods were used to account for the correlation among the repeated measures from each subject. Differences between treatment groups on continuous outcomes (NEI VFQ-17) were analyzed using a mixed-effects regression model adjusted for baseline scores. A random intercept term was included to account for correlations among repeated measurements from the same subject. Because of the skewness of HDRS scores, we used a Kruskal-Wallis test to examine change over time in this variable.

We tested the hypothesis that preventing the loss of valued activities would mediate the relationship between treatment group and depression diagnosis at 2 months. To do this we used Baron and Kenny's[23] approach to, first, establish significant intercorrelations between treatment group, depression, and relinquished activities and then to determine whether the association between treatment group and depression was either nonexistent or substantially reduced when loss of activities was included.

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Table 1. Baseline Demographic and Clinical Characteristics by Treatment Assignment[a]

Characteristic	Problem-Solving Treatment (n=105)	Usual Care (n=101)
Demographic characteristic		
Mean (SD) age, y	81.3 (5.4)	81.0 (6.3)
Female sex, %	65.7	74.3
White race, %	98.1	99.0
Married, %	42.9	40.6
Mean (SD) education, y	12.4 (2.3)	12.7 (3.5)
Clinical characteristic		
Mean (SD) best distance acuity, logarithm of the minimum angle of resolution	0.56 (0.33)	0.64 (0.44)
Mean (SD) best contrast sensitivity, log contrast sensitivity	0.71 (0.43)	0.61 (0.44)
Mean (SD) Chronic Disease Score[b]	5.5 (2.8)	5.0 (3.2)
Mean (SD) National Eye Institute Vision Function Questionnaire–17 score[c]	34.28 (14.12)	34.77 (12.80)
Mean (SD) Hamilton Depression Rating Scale score[d]	2.10 (2.07)	2.25 (2.36)
Underwent previous depression treatment, %	3.4	1.5

[a] There were no significant differences between groups.
[b] Scores ranged from 0 to 15, with higher scores indicating worse health status.
[c] Scores ranged from 16 to 76, with higher scores indicating worse vision function.
[d] Scores ranged from 0 to 75, with higher scores indicating worse depression.

Table 2. Outcomes for Problem-Solving Therapy (PST) and Usual Care Subjects at 2 and 6 Months

	Acute Effect[a]				Maintenance Effect[b]			
Measure	PST (n=95)	Usual Care (n=99)	OR (95% CI)	*P* Value	PST (n=95)	Usual Care (n=95)	OR (95% CI)	*P* Value
Depression, No. (%)[c]	11 (11.6)	23 (23.2)	0.39 (0.17-0.92)	.03	20 (21.1)	26 (27.4)	0.65 (0.33-1.39)	.29
No. of lost activities (%)[d]	22 (23.2)	37 (37.4)	0.48 (0.25 to 0.96)	.04	29 (30.5)	42 (44.2)	0.53 (0.28-1.01)	.06

	Acute Effect[a]				Maintenance Effect[b]			
	PST (n=95)	Usual Care (n=99)	*F*	*P* Value	PST (n=95)	Usual Care (n=95)	*F*	*P* Value
Mean (SE) change in NEI VFQ-17 score[e]	0.96 (7.97)	– 1.35 (7.80)	$4.17_{1,193}$	.04	– 0.97 (8.88)	– 2.45 (9.64)	$1.22_{1,189}$	.27
Mean (SD) change in HDRS score[f]	– 0.35 (2.88)	– 0.58 (2.96)	$0.298_{1,191}$	.59	– 1.03 (4.12)	– 1.04 (4.32)	$0.000_{1,189}$	.99

Abbreviations: CI, confidence interval; HDRS, Hamilton Depression Rating Scale; NEI VFQ-17, National Eye Institute Vision Function Questionnaire–17; OR, odds ratio.
[a] At 2 months.
[b] At 6 months.
[c] *DSM-IV*–defined diagnosis of a depressive disorder.
[d] Valued activity relinquished.
[e] Change from baseline. Scores ranged from 5 to 85. Positive scores indicate improved function.
[f] Change from baseline. Scores ranged from 0 to 75. Negative scores indicate worse depression.

To determine whether treatment was related to course of depression, we classified subjects as never having depression or as having incident, remitted, or persistent depression and compared the distribution of these outcomes by treatment group using a χ^2 test.

RESULTS

Problem-solving treatment and usual care subjects had no clinically or statistically significant differences in their baseline characteristics (**Table 1**). From baseline to 6 months, there were no statistically significant changes in visual acuity by treatment group (mean change, PST, –0.11 logMAR units [SD, 0.32 logMAR units]; usual care, –0.05 logMAR units [SD, 0.33 logMAR units]; $F_{1,180}=2.06$; $P=.15$]. **Table 2** compares incidence rates of depressive disorders, rates of relinquishing valued activities, and changes in NEI VFQ-17 and HDRS scores at months 2 and 6 by treatment assignment.

SHORT-TERM PREVENTIVE EFFECTS

At 2 months, 11 (11.6%) PST subjects (9 with major depression and 2 with minor depression) and 23 (23.2%) usual care controls (14 with major depression and 9 with minor depression) developed a depressive disorder. Problem-solving treatment subjects had less than half the odds of developing a depressive disorder as controls (odds ratio [OR], 0.39; 95% confidence interval [CI], 0.17-0.92; $P=.03$). The number of patients needed to be treated with PST to prevent 1 case of depression was 9 (95% CI, 5-72). The large CI

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Table 3. Course of Depression[a]

Course of Depression	Problem-Solving Treatment (n=95)	Usual Care (n=95)
Persistent[b]	4 (4.2)	16 (16.8)
Remitted[c]	7 (7.4)	6 (6.3)
Incident[d]	16 (16.8)	10 (10.5)
None[e]	68 (71.6)	63 (66.3)

[a] Subjects evaluated at 2 and 6 months. $\chi^2_{1,3}$=8.46; *P*=.04. All values are given as number (percentage).
[b] Depression diagnosis at 2 and 6 months.
[c] Depression diagnosis at 2 months but not at 6.
[d] Depression diagnosis at 6 months but not at 2.
[e] No depression diagnosis at 2 or 6 months.

reflects the size of the sample and the low incidence rate of depression.

The reduction in risk attributable to PST was 11% (95% CI, 0.6-21.0). To account for the unknown effects of attrition (11 PST subjects and 6 controls dropped from the study), we imputed depression status for missing subjects at 2 months under 4 scenarios. In the worst case (ie, all missing PST subjects were depressed and all controls were not), the OR for PST vs usual care was 0.89 (95% CI, 0.43-1.82; *P*=.75). For the best case (ie, no PST subjects were depressed and all controls were depressed), the OR was 0.33 (95% CI, 0.14-0.76; *P*=.01). If all missing subjects were depressed or if no missing subjects were depressed, the ORs were 0.78 (95% CI, 0.38-1.60; *P*=.50) and 0.38 (95% CI, 0.16-0.88; *P*=.02), respectively. These data provide the context in which to interpret our results.

Table 2 also shows that fewer PST subjects than controls relinquished a valued activity (OR, 0.48; 95% CI, 0.25-0.96; *P*=.04). We tested a mediation model to determine whether relinquishing a valued activity mediated the relationship between PST and depression using a logistic regression with depression diagnosis at 2 months as the outcome. Treatment group was entered on the first step and significantly predicted depression (OR, 2.31; 95% CI, 1.06-5.06; *P*=.04). This relationship diminished when activity loss was added (OR for treatment group, 2.04; 95% CI, 0.92-4.54; *P*=.08). Activity loss, however, was significant (OR, 2.55; 95% CI, 1.18-5.50; *P*=.02). These results suggest that PST prevented depression to the extent that it prevented loss of a valued activity.

Changes in NEI VFQ-17 scores, a subjective measure of vision-related task difficulty, showed that PST subjects had improved subjective vision function despite no change in objective acuity, whereas controls showed decline ($F_{1,193}$=4.17; *P*=.04) with no change in acuity. This change in NEI VFQ-17 score translates clinically to less difficulty on at least 1 vision-related functional activity for PST subjects and comparatively greater difficulty for controls. There were no significant differences in HDRS score changes over time between treatment groups.

MAINTENANCE EFFECTS

At 6 months, the prevalence of depressive disorders was not significantly different between treatment groups: 20 (21.1%) PST subjects (15 with major depression and 5 with minor depression) and 26 (27.4%) controls (14 with major depression and 12 with minor depression) developed a depressive disorder (OR, 0.65; 95% CI, 0.33-1.39; *P*=.29). At 2 months, however, PST subjects were almost half as likely to relinquish valued activities as controls (OR, 0.53; 95% CI, 0.28-1.01; *P*=.06), though this difference did not meet the customary level of statistical significance. There were no significant differences on NEI VFQ-17 (*P*=.27) or HDRS (*P*=.99) scores.

COURSE OF DEPRESSION

Table 3 demonstrates that the course of depression at 6 months differed by treatment group ($\chi^2_{1,3}$=8.46; *P*=.04). Only 4 of the 11 PST subjects (36.4% [4.2% of all PST subjects]) who were depressed at 2 months remained depressed at 6 months compared with 16 of 22 controls (72.7% [16.8% of all controls]). From months 2 to 6, following cessation of active treatment, more PST subjects than controls (16.8% of all PST subjects vs 10.5% of all controls) developed a depressive disorder. This trend accounted for PST's reduced treatment effect at 6 months and suggests that additional PST treatment may have been necessary to prevent depression in these subjects. In particular, the subjects with incident depression in the PST group had higher mean HDRS scores at baseline (4.0 [SD, 2.1]) and at 2 months (3.67 [SD, 3.0]) compared with subjects who never became depressed (1.35 [SD, 1.6] and 1.26 [SD, 1.5], respectively; both comparisons *P*<.001). It appears that PST mitigated their risk of depression initially but that its benefit decreased once it was removed.

COMMENT

We found that PST prevented depressive disorders as a short-term preventive treatment but that its effect did not persist over time. Although some may consider this to be a negative outcome, we found substantial evidence that PST is not the same as usual care. For one, we observed the hypothesized 50% risk reduction at 2 months and found that preventing the loss of valued activities mediated the relationship between PST and depression. We underestimated, however, the incidence of depression following cessation of treatment. This suggests that booster treatments for all PST subjects or rescue treatments for those with low-level depressive symptoms may have been necessary to sustain PST's ability to prevent depressive disorders. The former is a continuation of the selective intervention strategy that targets an asymptomatic high-risk group, whereas the latter is an indicated preventive strategy in that it targets patients with what may be early signs of depression.[5] In fact, our data and a recent review of brief interventions to prevent depression in older persons suggest the feasibility and effectiveness of these approaches.[24]

When we examined depression as a continuous measure of symptoms, we found no significant changes or differences between treatment groups. This was expected given that we enrolled subjects without depression. Although a notable minority developed a depres-

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sive disorder over time, the preponderance of HDRS scores in the sample remained low throughout the study. Nevertheless, our data indicate that even a few depressive symptoms, below even what is considered subsyndromal depression, increase the risk for subsequent depressive disorder. This suggests that indicated preventive interventions that target patients with low-level depressive symptoms may be more efficient.

Second, we found that the incidence rate of depression in controls was lower than we had observed in an earlier observational study.[11] We suspect that frequent contact with study personnel and the expectation of receiving PST following the clinical trial accounted for this. Thus, we believe that diminution of treatment efficacy over time in PST subjects, nonspecific benefits for controls, and insufficient statistical power may account for the findings.

Third, the course of depression differed in PST and control subjects. Depression in controls, at least from 2 to 6 months, was more likely to persist, whereas in PST subjects, it was more likely to remit. Whether depression at 6 months in PST subjects would similarly remit is unknown.

From a research design perspective we have shown that systematic sampling, successful randomization and masking, protocol-driven treatment, assessment of multiple relevant outcomes, and maintenance of treatment fidelity are feasible in psychosocial intervention studies. The generalizability and specificity of this study, however, may be limited by the unique visual characteristics of the sample, the uncertain effects of attrition, and the absence of an attention control treatment group. Our current National Eye Institute–funded clinical trial to improve vision function in AMD randomizes subjects to PST and supportive therapy (a placebo psychological treatment) to control for nonspecific treatment effects. However, our finding in this study, that preventing loss of valued activities mediated PST's efficacy to prevent depression at 2 months, suggests a specific treatment effect: that PST-treated subjects may have been able to develop compensatory strategies to continue to pursue valued activities.

Currently, depression is a major public health problem because it causes suffering, disability, and excess health care costs and is often unrecognized and untreated.[1] Although physicians have been more inclined to prescribe antidepressant medications in recent years, the effectiveness of this practice is unknown.[25] In fact, many older persons are dissatisfied with such treatment and do not adhere to prescribed medications.[26,27] These trends have prompted the development of innovative, collaborative care models to treat depression, but substantial barriers prevent their broad implementation.[28-31] As a result, depression remains a difficult condition to treat.[32] Thus, preventing depression may be more sensible, and perhaps more cost-effective, than waiting to treat it once it occurs or failing to treat it at all.

Our study adds important new information to the ongoing study of enhanced-care models to prevent or treat depression. We have shown that PST can prevent depression and influence its course, at least temporarily, and help people with visual impairments preserve valued activities. In the face of progressive vision loss, these are positive findings. They add to other studies that demonstrate the value of depression-management specialists working collaboratively with physicians to treat and, as our data suggest, prevent depression in older patients.[28-31] In a larger sense, our study suggests that a practical, problem-solving intervention like PST may prevent depression in other diseases. Because its treatment approach is relatively generic, non–mental health specialists can readily deliver PST to patients with other disabling conditions, such as heart disease, cancer, and stroke. Moreover, because Medicare reimburses nurses (about $50 per session) to deliver interventions like PST to patients in their homes, such treatments are ready to move from the research realm to routine clinical care.

Future studies might investigate the optimal timing of PST (perhaps to include booster or rescue treatments) to prevent depression for longer time periods and evaluate its cost-effectiveness. Smit et al,[33] for example, who reported that 1 in every 5 cases of late-life depression is a new case, recommended directing prevention efforts to people with subsyndromal symptoms to generate the best health benefits against the lowest costs. In this context, we view our study as part of the larger effort to find ways to manage depression in medical settings. As the population ages, increasingly large numbers of older adults will become disabled and depressed. Whether their disability is due to AMD or other chronic diseases, finding ways to prevent depression is clearly important.

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