Collaborative Care Versus Screening and Follow-up for Patients With Diabetes and Depressive Symptoms: Results of a Primary Care–Based Comparative Effectiveness Trial

Jeffrey A. Johnson,[1,2] Fatima Al Sayah,[1,2] Lisa Wozniak,[2] Sandra Rees,[2] Allison Soprovich,[2] Weiyu Qiu,[2] Constance L. Chik,[3] Pierre Chue,[4,5] Peter Florence,[4] Jennifer Jacquier,[3] Pauline Lysak,[5] Andrea Opgenorth,[3] Wayne Katon,[6] and Sumit R. Majumdar[1,2,3]

Diabetes Care 2014;37:3220–3226 | DOI: 10.2337/dc14-1308

OBJECTIVE

Depressive symptoms are common and, when coexisting with diabetes, worsen outcomes and increase health care costs. We evaluated a nurse case-manager–based collaborative primary care team model to improve depressive symptoms in diabetic patients.

RESEARCH DESIGN AND METHODS

We conducted a controlled implementation trial in four nonmetropolitan primary care networks. Eligible patients had type 2 diabetes and screened positive for depressive symptoms, based on a Patient Health Questionnaire (PHQ) score of ≥10. Patients were allocated using an "on-off" monthly time series. Intervention consisted of case-managers working 1:1 with patients to deliver individualized care. The main outcome was improvement in PHQ scores at 12 months. A concurrent cohort of 71 comparable patients was used as nonscreened usual care control subjects.

RESULTS

Of 1,924 patients screened, 476 (25%) had a PHQ score >10. Of these, 95 were allocated to intervention and 62 to active control. There were no baseline differences between groups: mean age was 57.8 years, 55% were women, and the mean PHQ score was 14.5 (SD 3.7). Intervention patients had greater 12-month improvements in PHQ (7.3 [SD 5.6]) compared with active-control subjects (5.2 [SD 5.7], P = 0.015). Recovery of depressive symptoms (i.e., PHQ reduced by 50%) was greater among intervention patients (61% vs. 44%, P = 0.03). Compared with trial patients, nonscreened control subjects had significantly less improvement at 12 months in the PHQ score (3.2 [SD 4.9]) and lower rates of recovery (24%, $P < 0.05$ for both).

CONCLUSIONS

In patients with type 2 diabetes who screened positive for depressive symptoms, collaborative care improved depressive symptoms, but physician notification and follow-up was also a clinically effective initial strategy compared with usual care.

[1]School of Public Health, University of Alberta, Edmonton, Alberta, Canada
[2]Alliance for Canadian Health Outcomes Research, University of Alberta, Edmonton, Alberta, Canada
[3]Department of Medicine, University of Alberta, Edmonton, Alberta, Canada
[4]Alberta Health Services, Edmonton, Alberta, Canada
[5]Department of Psychiatry, University of Alberta, Edmonton, Alberta, Canada
[6]Department of Psychiatry and Behavioral Sciences, University of Washington, Seattle, WA

Corresponding author: Jeffrey A. Johnson, jeff.johnson@ualberta.ca.

Received 22 May 2014 and accepted 14 September 2014.

This article contains Supplementary Data online at http://care.diabetesjournals.org/lookup/suppl/doi:10.2337/dc14-1308/-/DC1.

Clinical trial reg. no. NCT01328639, clinicaltrials.gov.

Depression and depressive symptoms are common in patients with type 2 diabetes, with rates twofold higher than in the general population (1). Despite this, less than 50% of these patients are recognized as having depressive symptoms (1). Depression itself is considered a risk factor for the development of type 2 diabetes (2) and is associated with poorer self-care behaviors (3), worse glycemic control, increased risk of complications (4), decreased quality of life (5), and substantially higher health care costs (6).

Although the majority of individuals with diabetes and depression are treated in primary care settings (7), only a minority receive adequate treatment for depression (7,8). Given its high prevalence and effect on outcomes and the availability of effective treatments, screening for depression in patients with diabetes has been recommended by some (9,10). This is, however, not without controversy (11,12); for example, the Canadian Task Force for Preventive Medicine recently recommended against screening for depression in primary care in general or in high-risk patients such as those with chronic diseases (13). Much of the debate has surrounded the limited evidence base and the value of screening alone versus screening combined with coordinated care (11–14).

A growing body of literature has demonstrated value in collaborative care models for patients with depression (15,16) and, specifically, in patients with diabetes (17). These models are also considered to be cost-effective, although estimates of efficiency may be based on only a subset of controlled studies with large clinical effects that are not representative (18). In general, the controlled trials of collaborative care for depression in primary care settings suggest a small-to-moderate effect (16,17,19). Furthermore, most of the studies have been undertaken in the U.S. (19), although recent trials in Europe of collaborative care for depression demonstrated feasibility and modest effectiveness (20,21). We therefore sought to evaluate the comparative effectiveness of a collaborative model of care for patients with type 2 diabetes and depressive symptoms in the Canadian primary care setting while also determining the value of screening for depression itself when compared with usual care delivered outside the trial setting.

RESEARCH DESIGN AND METHODS

Overview

Our study design and rationale have been previously published (22,23). Our intervention was an adaptation of a nurse-led collaborative care model previously proven effective in randomized controlled trials in U.S. health maintenance organizations (24,25). We chose to evaluate this model using a pragmatic, controlled implementation trial. We used a variant of the cohort multiple randomized controlled trial design (22,26) and took advantage of sampling from the Alberta's Caring for Diabetes (ABCD) Cohort Study (23), a large, ongoing, annual survey of patients with type 2 diabetes. Initial contact with cohort participants included the administration of the Patient Health Questionnaire (PHQ). Those who screened positive for depressive symptoms (i.e., PHQ $\geq$10) were invited to participate and allocated to intervention or active control. We also identified patients from the ABCD Cohort who would have been eligible for our trial, but who were neither approached nor enrolled, to serve as independent nonscreened usual care control subjects. Ethics approval was granted from the University of Alberta Health Research Ethics Board (HREB #PRO00012663).

Setting and Population

We worked in collaboration with four primary care networks (PCNs) in nonmetropolitan Alberta. A PCN is akin to the "medical home" (27). Collectively, the four study PCNs represent 140 family physicians and 180,000 patients, with an estimated 10,000 patients with type 2 diabetes. Eligible patients were adults aged 18 years and older with type 2 diabetes and PHQ $\geq$10. We excluded patients with severe physical illness, serious psychiatric illness (e.g., bipolar disorder, schizophrenia, or under current care by a psychiatrist), those who were pregnant or breast-feeding, and those who were participating in other clinical trials. Patient enrollment started in November 2010, with the last patient visit in January 2013.

Recruitment and Allocation

Recruitment included three steps. First, a screening survey was mailed. Second, PCN staff confirmed eligibility and scheduled a baseline visit. Third, the care manager (CM) reconfirmed eligibility and obtained written informed consent. Patients were then allocated using a controlled "on-off" time series method (22,28). During month 1, patients were allocated to intervention; those enrolled in month 2 were allocated to active control, and so on. Research personnel, analysts, and investigators were masked to allocation status at all times, although neither the CM nor patients could be blinded. This method has been used in many studies. It reliably leads to balance in measured (and unmeasured) patient characteristics and meets criteria for internal validity sufficient for inclusion in the Cochrane Collaborations' Effective Practice and Organization of Care systematic reviews (29).

Intervention (Collaborative Care)

Our collaborative care model was an adaptation of the TEAMCare approach (24,25). The intervention involved a registered nurse CM who coordinated collaborative team management. The goal was to reduce depressive symptoms, achieve targets for cardiometabolic parameters, and improve lifestyle behaviors. The CM worked with the patient to develop a shared care plan, offered support and problem solving techniques to optimize self-management, and closely monitored treatment adherence and outcomes (24,25). The CM provided active in-person or telephone follow-up once to twice per month to reassess symptoms and assist patients in achieving goals. The CM consulted regularly with specialists (psychiatrists and endocrinologists) to review new cases and ongoing patient progress and discuss recommendations, based on locally developed and endorsed evidence-based care algorithms. The CM communicated recommendations to family physicians, who remained responsible for final treatment decisions and all prescriptions. Management of depressive symptoms involved the use of antidepressant medication and/or psychotherapy. Once patients achieved symptom amelioration (PHQ $<$10), a relapse prevention plan was developed while continuing to work toward cardiometabolic control and lifestyle modifications.

Members of the team in each PCN, including the CMs, specialists, physician

champion, and data administrative assistant, received extensive, on-going training facilitated by the original TEAMCare investigators (24,25). Training included an initial 2-day training session, an annual booster session, and monthly teleconferences with the CMs and specialists. The CMs also received standardized training in problem-solving therapy, behavioral activation, and motivational interviewing.

Active-Control Subjects (Screening and Follow-up)

All patients were screened using the PHQ. Each patient's family physician was notified by letter that the patient had recently screened positive for depressive symptoms. Patients allocated to active control received all subsequent follow-up from their family physician, at his or her discretion, without further additional active support from the CM. PHQ and additional measures were collected at baseline, 6, and 12 months, and the family physician was notified if any measures fell outside prespecified values.

Nonscreened Usual Care Control Subjects

Irrespective of trial participation, all patients with type 2 diabetes in the province were potentially eligible for our concurrent ABCD Cohort Study (23). ABCD cohort respondents completed a written questionnaire with self-reported measures that overlapped the measures in the TEAMCare study, including the PHQ8 (30). One item (suicide screen) from the PHQ9 is omitted in the PHQ8, and the latter was used because no follow-up was planned for the cohort participants who might positively respond to this item. Both versions are similarly scored, yield essentially identical results, and are considered equivalent (14,30). For clarity and brevity, we refer to these measures as PHQ throughout the article. We identified those participants in the ABCD Cohort with PHQ $\geq$10 at the baseline survey and evaluated their PHQ score from the subsequent 12-month survey during the same study period. Because the baseline PHQ scores were not calculated for these patients (i.e., only measured) nor were any family physician notification or any other clinical follow-up planned, we considered them an independent but concurrent and comparable nonscreened control group, representing the natural history of depressive symptoms outside of our trial (i.e., true "usual care").

Measures

Clinical outcomes were assessed at baseline, 6, and 12 months. Our primary outcome was change in the PHQ score over 12 months, where 5 points is considered an important difference (14,31). We also evaluated the rates of clinically significant recovery (PHQ improved by 50% of the baseline score) and amelioration of depressive symptoms (PHQ $<$10) at 12 months (14,20). Clinical and laboratory measures included BMI, waist circumference, A1C, systolic blood pressure (SBP), and LDL-cholesterol (32). Patient-reported outcomes were also collected from all participants using validated surveys. Health-related quality-of-life measures included the SF-12 (33) physical and mental composite summaries, the 5-level EQ-5D (34), and the Problem Areas in Diabetes 5-item (PAID-5) (35). Satisfaction with care was measured using the brief Patient Assessment of Chronic Illness Care (PACIC-11) (36,37).

Sample Size and Power

We estimated that a minimum total sample size of 120, with 60 in each arm, would provide 80% power to detect mean between-group differences of 5 points in the PHQ (14,31), assuming a repeated-measures correlation of 0.6, and two-tailed α of 0.05. This sample size provided more than 80% power to detect any between-group absolute differences in proportions of 15% or more (e.g., 45% of usual care patients achieve recovery vs. 60% of intervention patients). Anticipating a 40% attrition rate, we planned to recruit 160 patients.

Data Analysis

Using random-effects linear regression models, we compared the pooled estimates of improvements in each outcome from baseline to 6 months and 12 months, respectively, between the intervention and active-control groups. We used logistic regression to compare the proportion of subjects who achieved recovery (PHQ improved by 50%) and amelioration (i.e., PHQ $<$10) of depressive symptoms (14) at 12 months. We used an intention-to-treat framework and last-observation-carried-forward imputation of missing values. Because of concerns with the last-observation-carried-forward approach to missing data, we repeated all comparisons using individual growth-curve models, obtaining consistent results (results not shown). We also repeated all analyses adjusting for age, sex, BMI, and PCN site, again obtaining results similar to the main analysis (results not shown). We also directly compared 12-month changes and improvements in PHQ among trial patients with the independent nonscreened usual care control patients from the ABCD cohort with linear and logistic regressions, respectively, adjusting for baseline age difference. Finally, we stratified these three-group comparisons by baseline PHQ score $>$14. For all analyses, we used SAS 9.2 software (SAS Institute, Inc., Cary, NC), considered a P value $<$0.05 to be statistically significant, and did not adjust for multiple comparisons.

RESULTS

We mailed screening packages to 7,846 patients, with a response rate of 35% (n = 2,718), of whom 71% (n = 1,924) were initially eligible (Supplementary Data). Of these, 476 patients (25%) had PHQ $\geq$10. From this group, 157 patients were enrolled in the trial, with 95 allocated to intervention and 62 to active control. Forty-three patients (27%) did not complete the trial, with no significant difference between trial groups (29% vs. 24%; P = 0.47) (Supplementary Data). Three participants did not complete the survey at any time point, and thus, the analysis of secondary outcomes from the survey data was based on a sample of 154.

Baseline Characteristics

There were no important baseline differences between intervention and active-control patients (Table 1). The mean age overall was 57.8 (SD 9.8) years, 55% were women, and 75% had a BMI $>$30 kg/m^2. The mean PHQ score at baseline was 14.5 (SD 3.7), and just under one-half of patients were being treated with psychoactive medications. Patients had reasonably good cardiometabolic control, with mean A1C of 7.6% (SD 1.8%) (60 [SD 19.7] mmol/mol), SBP of 125.2 (SD 15.9) mmHg, and LDL-cholesterol of 2.2 (SD 0.8) mmol/L.

Table 1—Baseline characteristics for trial participants

Characteristic	Intervention $N = 95$	Active control $N = 62$	Usual care $N = 71$*	P value**
Sex, *n* (% female)	58 (61)	29 (47)	40 (56)	0.21
Age (year), mean (SD)	57.0 (10.5)	59.2 (8.5)	63.4 (11.3)	<0.001
Obese (BMI >30 kg/m^2), *n* (%)	71 (76)	44 (72)	47 (68)	0.49
Education less than high school, *n* (%)	11 (12)	10 (16)	14 (20)	0.60
Employed, *n* (%)	48 (52)	31 (51)	25 (36)	0.27
White, *n* (%)	81 (87)	49 (80)	66 (96)	0.07
Income, *n* (%)				
<$40,000	28 (30)	16 (26)	29 (46)	0.24
$40,000–$80,000	23 (25)	16 (26)	15 (24)	
≥$80,000	26 (28)	13 (21)	16 (25)	
Refused to answer	3 (3)	8 (13)	3 (5)	
Smoking, *n* (%)	23 (25)	15 (25)	13 (18)	0.42
Alcohol use, *n* (%)	70 (75)	41 (67)	42 (59)	0.09
Psychoactive medications***, *n* (%)	46 (48)	27 (44)	–	0.55
PHQ, mean (SD)	14.5 (3.8)	14.6 (3.5)	14.2 (3.4)	0.79
A1C (%), mean (SD)	7.5 (1.8)	7.8 (1.7)	–	0.18
A1C (mmol/mol), mean (SD)	58 (19.7)	62 (18.6)	–	0.18
SBP (mmHg), mean (SD)	126 (15.5)	124 (16.5)	–	0.29
LDL-cholesterol (mmol/L), mean (SD)	2.2 (0.9)	2.1 (0.8)	–	0.48

*Drawn from similar patients in ABCD cohort; medication and laboratory data were not available. **From t test for continuous data or χ^2 for categorical data. ***Antidepressants, anxiolytics, mood stabilizers, and/or antipsychotics.

Depressive Symptoms

Intervention patients had greater improvements in PHQ (7.3 [SD 5.6]) over 12 months compared with active-control subjects (5.2 [SD 5.7]), for a between-group difference of difference of 2.0 (95% CI 0.4, 3.7; $P = 0.015$) (Table 2 and Fig. 1). Clinically important recovery from depressive symptoms occurred in 61% of intervention patients compared with 44% of active-control subjects ($P = 0.03$).

Patients in the nonscreened usual-care control cohort were similar to trial patients in sex (54% female) and baseline PHQ scores (14.2), but were older than trial patients (Table 1). At 12 months, the nonscreened usual care control subjects had a PHQ improvement of 3.2 (SD 4.9) (Table 3), which was significantly less than improvements observed among the intervention ($P < 0.001$) or active-control ($P = 0.029$) patients, after adjusting for age. At 12 months, only 24% of the nonscreened usual care control subjects had recovered, and 39% achieved symptom amelioration ($P < 0.05$ for both) (Table 3). The improvements appeared to be greatest in those with more severe depressive symptoms at baseline (Supplementary Data).

Other Outcomes

No significant differences were observed in changes in A1C, SBP, or LDL-cholesterol between intervention and active-control groups (Table 2). All patients had substantially impaired health-related quality of life at baseline, but improvements were observed in all patients for all outcomes, with no differences between groups (Table 2). PAID-5 scores suggested intervention patients had a significantly greater reduction in diabetes-specific distress than active-control subjects ($P = 0.03$) (Table 2). For the PACIC-11 scores, there was a nonsignificant ($P = 0.25$) improvement of 18.1 (SD 30.0) points in the intervention group compared with 12.6 (SD 26.2) points for active-control subjects (Table 2).

CONCLUSIONS

As has been previously established, the collaborative care intervention implemented in our local setting led to reductions in depressive symptoms over and above care delivered by family physicians who were notified that their patients screened positive for depressive symptoms. In fact, most of the patients in our trial, who had moderately severe depressive symptoms at baseline, had large and clinically important improvements in their symptoms over 12 months. However, outside the trial, concurrent nonscreened patients with diabetes (whose family physicians were not notified of PHQ results) had far less improvement in depressive symptoms over 12 months than the intervention or active-control patients. Thus, our results suggest collaborative care models are effective outside of randomized trials and in different primary care settings and that screening for depressive symptoms and notification to the family physician may be an initial, and effective, strategy to achieve worthwhile improvements in the care of those with depressive symptoms and type 2 diabetes compared with usual care.

Although we evaluated this collaborative care model with a pragmatic implementation trial, we recognized some potential limitations. We used a controlled but nonrandom allocation of patients, which has greater potential for unbalanced study groups, particularly because an unblinded CM undertook enrollment and assessment. This might be considered an important threat to internal validity. However, we demonstrated that nonrandomized allocation led to study groups that, although unbalanced in number, had comparable baseline characteristics, and ultimately, we observed an effect size similar to that seen in randomized trials. We considered the available evidence for collaborative care models quite robust (15,17), including the randomized trials testing the efficacy of the specific TEAMCare model we implemented (24,25). Therefore, we did not see the need to conduct another randomized trial and were reluctant to allocate to a true unscreened (i.e., family physicians masked to PHQ status at baseline) control group (22).

Although relatively small, our minimal sample size calculation ($N = 120$) suggested adequate power to detect clinically important changes in the PHQ. In fact, the magnitude of the difference between our intervention and active-control groups (2.0 points, or an effect size of 0.56) is similar to previous trials of collaborative care (15,17,25). Although this difference is well below the previously suggested criterion (and our a priori standard) of 5 points (14,31), some controlled studies suggest that even small to moderate improvements

Table 2—Outcomes at 6 and 12 months for intervention and active-control patients

	Baseline Mean (SD)	6-month change Mean (SD)	12-month change Mean (SD)	Difference in change*	*P* value**
PHQ					
Intervention	14.5 (3.8)	−6.6 (5.0)	−7.3 (5.6)	2.0	0.015
Active control	14.6 (3.5)	−4.6 (5.8)	−5.2 (5.7)		
A1C (%)					
Intervention	7.5 (1.8)	−0.2 (1.1)	−0.2 (1.3)	−0.1	0.47
Active control	7.8 (1.7)	−0.4 (0.9)	−0.2 (1.1)		
A1C (mmol/mol)					
Intervention	58 (19.7)	−2.2 (12.0)	−2.2 (14.2)	−1.3	0.47
Active control	62 (18.6)	−4.4 (9.8)	−2.2 (12.0)		
SBP (mmHg)					
Intervention	126 (15.5)	−2.3 (13.8)	−4.8 (13.8)	2.5	0.75
Active control	124 (16.5)	−3.1 (21.1)	−2.5 (17.8)		
LDL-cholesterol (mmol/L)					
Intervention	2.2 (0.9)	−0.2 (0.6)	−0.2 (0.7)	0.08	0.39
Active control	2.1 (0.8)	−0.2 (0.6)	−0.1 (0.9)		
PCS-12					
Intervention	36.0 (10.0)	1.5 (6.3)	2.8 (6.9)	−0.7	0.54
Active control	34.9 (9.8)	2.6 (7.3)	3.1 (9.0)		
MCS-12					
Intervention	34.1 (7.7)	6.1 (8.1)	7.6 (9.5)	2.3	0.09
Active control	36.6 (7.3)	4.7 (9.7)	4.4 (8.4)		
EQ-5D					
Intervention	0.71 (0.16)	0.01 (0.11)	0.03 (0.10)	−0.02	0.23
Active control	0.67 (0.17)	0.04 (0.10)	0.04 (0.12)		
PAID-5					
Intervention	1.6 (1.0)	−0.4 (0.8)	−0.6 (0.8)	0.3	0.03
Active control	1.3 (1.0)	−0.1 (0.9)	−0.2 (0.9)		
PACIC-11					
Intervention	34.7 (27.9)	–	18.1 (30.0)	5.5	0.25
Active control	34.4 (25.0)	–	12.6 (26.2)		

*Difference in change between intervention and active control over 12 months adjusted for time. ***P* value from a random-effects model comparing changes in intervention and active-control patients at 6 and 12 months.

in depressive symptoms might be associated with reduced health care utilization and costs (19,38,39). We also recognize that attrition was somewhat higher than in previous studies, although it was less than we originally anticipated. In part this may be expected because we undertook this as an implementation study, with less control than in previous, and more rigorous, efficacy trials. Regardless, our attrition was nondifferential (~27% across both groups). Furthermore, when those who did or did not complete the full 12-month follow-up visits were compared, there was no difference in baseline A1C ($P = 0.24$), LDL-cholesterol ($P = 0.34$), or SBP ($P = 0.36$). There was a statistically significant difference in the baseline PHQ score, but it was of marginal clinical importance (14.1 for completers and 15.4 for noncompleters; $P = 0.04$). We therefore believe there was little bias due to attrition.

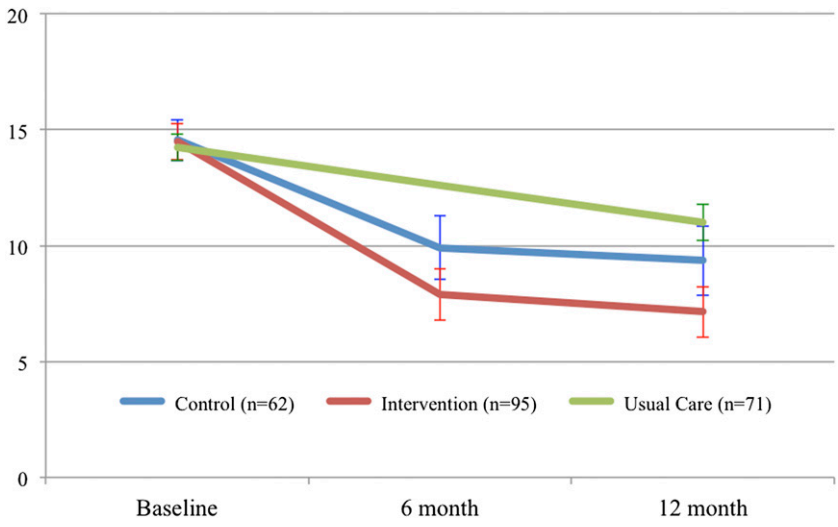

Figure 1—Change in the PHQ score over 12 months for collaborative care intervention vs. active-control trial patients and nonscreened usual care control subjects.

A major strength of our study was our consideration of the true natural history of depressive symptoms for patients who were not actively screened. In this context, the intervention and active-control patients in our trial both had substantially better improvements in PHQ scores than those potentially eligible but outside the trial. Indeed,

Table 3—Change in depressive symptoms over 12 months for trial and nonscreened usual care control patients

	Baseline Mean (SD)	12 months Mean (SD)	Change* Mean (SD)	*P* value**
PHQ score				
Intervention (*n* = 95)	14.5 (3.8)	7.1 (5.4)	−7.3 (5.6)	<0.001
Active control (*n* = 62)	14.6 (3.5)	9.4 (5.9)	−5.2 (5.7)	0.030
Usual care control (*n* = 71)	14.2 (3.4)	11.0 (5.2)	−3.2 (4.9)	–

	%	NNT*** *n*	*P* value**
Proportion recovered (PHQ improved by 50%)			
Intervention (*n* = 95)	61	3	<0.001
Active control (*n* = 62)	44	5	0.034
Usual care control (*n* = 71)	24		
Proportion achieving amelioration (PHQ <10)			
Intervention (*n* = 95)	68	3	<0.001
Active control (*n* = 62)	58	5	0.020
Usual care control (*n* = 71)	39		

NNT, number needed to treat. *Change from baseline to 12 months. **From linear (PHQ score change) or logistic (proportions) regressions comparing with usual care control subjects, after adjusting for age. ***NNT over 12 months to achieve amelioration compared with usual care control subjects.

the magnitude of the improvements in depressive symptoms seen in our active-control group (screening and notification with follow-up) compared with no screening was substantial, with an effect size of 0.63, similar to that seen in previous randomized trials (16,17,25) and in our own comparative effectiveness trial. This substantial effect, obtained with fewer and more sustainable resources (i.e., our active-control group), may be considered an important first step in improving the quality of care for patients with comorbid diabetes and depression. If, however, additional resources are available to implement and sustain more costly and complex collaborative care models, even greater improvements could be achieved. Moreover, the greatest improvements were observed in those with more severe depressive symptoms at baseline. These may be strategies to consider in the translation of our findings for the implementation of these interventions in primary care.

As noted above, however, recommendations for screening for depression in primary care are controversial (11,12). At the time we initiated our study, the Canadian Task Force for Preventive Care recommended screening for depression (9), similar to recommendations in the U.S. (10). Shortly after the completion of our study, however, the recommendations of the Canadian Task Force changed (13), making our approach discordant with the guidelines. The change in Canadian Task Force recommendations was not based on new evidence but rather on reinterpretation of the available evidence, where no randomized trials of depression screening exist (11,13). Although the guidelines, and controversy, are based on screening for depression, a more nuanced and clinically informed alternate view may be that there is value in a proactive strategy of case-finding patients with unresolved depressive symptoms (40). Although not randomized, our results nonetheless suggest that there are benefits to actively identifying patients with type 2 diabetes and depressive symptoms and notifying their physicians of their PHQ results. Indeed, given that half our trial population was already taking a psychoactive medication of some type, the issue may be more of inadequate or undertreatment of depression rather than just identifying new cases.

The change in PAID-5 score, a measure of diabetes distress, in the intervention compared with active-control patients was an interesting observation. It is likely that there is some degree of overlap between distress and depressive symptoms, but it is clearly not complete overlap. We recruited patients on the basis of depressive symptoms, not diabetes distress or poor diabetes control, and in fact, most patients already had good glycemic control, suggesting less diabetes distress in this sample of patients. We observed only a weak to moderate correlation between PAID and PHQ scores (0.17 at baseline, 0.46 at 12 months) and found that 30% of patients with moderate to severe depressive symptoms (PHQ >10) had no diabetes distress (PAID = 0) at baseline. Regardless, our intervention focused initially on improving quality of care for depressive symptoms, included problem solving and improving antidepressant therapy, and resulted in a significant improvement in depressive symptoms. Once these improvements were seen in patients, the CM then shifted focus to cardiometabolic care and treatments. Thus, it is likely the observed improvements in diabetes distress may have been secondary to reduced depressive symptoms. Nonetheless, it would be worthwhile to further explore the temporal relationship and patterns of improvements in symptoms of depression and diabetes distress.

In conclusion, we observed one in four patients with type 2 diabetes in the primary care setting had moderate to severe depressive symptoms. Substantial improvements in depressive symptoms occurred in most of the patients enrolled in our trial, with greater improvements achieved through collaborative care compared with our active-control group, which based on our design and results is perhaps better termed “enhanced usual care.” In more resource-constrained settings, our findings also suggest that active screening for depressive symptoms with family physician notification and follow-up may be an effective initial strategy to achieve worthwhile improvements in these high-risk patients compared with the status quo of usual primary care.

Funding. J.A.J. is a Senior Scholar with Alberta Innovates-Health Solutions (AIHS) and a Centennial Professor at the University of Alberta. S.R.M. is a Health Scholar funded by AIHS and holds the Endowed Chair in Patient Health Management funded by the Faculties of Medicine and Dentistry and Pharmacy and Pharmaceutical Sciences of the University of Alberta. This work was supported in part by grant from Alberta Health and a Canadian Institutes of Health Research (CIHR) Team Grant to the Alliance for Canadian Health Outcomes Research in Diabetes (reference No. OTG-88588),

sponsored by the CIHR Institute of Nutrition, Metabolism and Diabetes.

Duality of Interest. P.C. has received research and travel grants from Janssen, Pfizer, AstraZeneca, Lundbeck, Bristol-Myers Squibb, and Eli Lilly as a researcher and speaker. W.K. has received support to serve on advisory boards from Eli Lilly and has received honorariums for lectures from Pfizer, Forest, and Eli Lilly. No other potential conflicts of interest relevant to this article were reported.

Author Contributions. J.A.J. led the conception and design of the study. F.A.S., L.W., S.R., A.S., and W.Q. collected and/or analyzed data and reviewed and edited the manuscript. C.L.C., P.C., P.F., J.J., P.L., and A.O. provided clinical care as specialists and reviewed and edited the manuscript. W.K. served as a consultant in the implementation phase of this study and reviewed and edited the manuscript. S.R.M. contributed to conception and design of the study and data analysis and interpretation, and reviewed and edited the manuscript. J.A.J. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

References

1. Holt RIG, de Groot M, Lucki I, Hunter CM, Sartorius N, Golden SH. NIDDK international conference report on diabetes and depression: current understanding and future directions. Diabetes Care 2014;37:2067–2077
2. Knol MJ, Twisk JWR, Beekman ATF, Heine RJ, Snoek FJ, Pouwer F. Depression as a risk factor for the onset of type 2 diabetes mellitus. A meta-analysis. Diabetologia 2006;49:837–845
3. Katon WJ, Russo JE, Heckbert SR, et al. The relationship between changes in depression symptoms and changes in health risk behaviors in patients with diabetes. Int J Geriatr Psychiatry 2010;25:466–475
4. de Groot M, Anderson R, Freedland KE, Clouse RE, Lustman PJ. Association of depression and diabetes complications: a meta-analysis. Psychosom Med 2001;63:619–630
5. Ali S, Stone M, Skinner TC, Robertson N, Davies M, Khunti K. The association between depression and health-related quality of life in people with type 2 diabetes: a systematic literature review. Diabetes Metab Res Rev 2010;26:75–89
6. Simon GE, Katon WJ, Lin EHB, et al. Diabetes complications and depression as predictors of health service costs. Gen Hosp Psychiatry 2005; 27:344–351
7. Katon WJ, Simon G, Russo J, et al. Quality of depression care in a population-based sample of patients with diabetes and major depression. Med Care 2004;42:1222–1229
8. Remick RA. Diagnosis and management of depression in primary care: a clinical update and review. CMAJ 2002;167:1253–1260
9. MacMillan HL, Patterson CJS, Wathen CN, et al.; Canadian Task Force on Preventive Health Care. Screening for depression in primary care: recommendation statement from the Canadian Task Force on Preventive Health Care. CMAJ 2005;172:33–35
10. U.S. Preventive Services Task Force. Screening for depression in adults: U.S. preventive services task force recommendation statement. Ann Intern Med 2009;151:784–792
11. Thombs BD, Coyne JC, Cuijpers P, et al. Rethinking recommendations for screening for depression in primary care. CMAJ 2012;184: 413–418
12. Bland RC, Streiner DL. Why screening for depression in primary care is impractical. CMAJ 2013;185:753–754
13. Joffres M, Jaramillo A, Dickinson J, et al.; Canadian Task Force on Preventive Health Care. Recommendations on screening for depression in adults. CMAJ 2013;185:775–782
14. Kroenke K. Enhancing the clinical utility of depression screening. CMAJ 2012;184:281–282
15. Gilbody S, Bower P, Fletcher J, Richards D, Sutton AJ. Collaborative care for depression: a cumulative meta-analysis and review of longer-term outcomes. Arch Intern Med 2006;166: 2314–2321
16. Thota AB, Sipe TA, Byard GJ, et al.; Community Preventive Services Task Force. Collaborative care to improve the management of depressive disorders: a community guide systematic review and meta-analysis. Am J Prev Med 2012;42:525–538
17. van der Feltz-Cornelis CM, Nuyen J, Stoop C, et al. Effect of interventions for major depressive disorder and significant depressive symptoms in patients with diabetes mellitus: a systematic review and meta-analysis. Gen Hosp Psychiatry 2010;32:380–395
18. Gilbody S, Bower P, Sutton AJ. Randomized trials with concurrent economic evaluations reported unrepresentatively large clinical effect sizes. J Clin Epidemiol 2007;60:781–786
19. Jacob V, Chattopadhyay SK, Sipe TA, Thota AB, Byard GJ, Chapman DP; Community Preventive Services Task Force. Economics of collaborative care for management of depressive disorders: a community guide systematic review. Am J Prev Med 2012;42:539–549
20. Richards DA, Hill JJ, Gask L, et al. Clinical effectiveness of collaborative care for depression in UK primary care (CADET): cluster randomised controlled trial. BMJ 2013;347:f4913
21. Gensichen J, Petersen JJ, Von Korff M, et al. Cost-effectiveness of depression case management in small practices. Br J Psychiatry 2013; 202:441–446
22. Johnson JA, Al Sayah F, Wozniak L, et al. Controlled trial of a collaborative primary care team model for patients with diabetes and depression: rationale and design for a comprehensive evaluation. BMC Health Serv Res 2012;12: 258
23. Wozniak L, Rees S, Soprovich A, et al. Applying the RE-AIM framework to the Alberta's Caring for Diabetes Project: a protocol for a comprehensive evaluation of primary care quality improvement interventions. BMJ Open 2012; 2:e002099
24. Katon WJ, Von Korff M, Lin EH, et al. The Pathways Study: a randomized trial of collaborative care in patients with diabetes and depression. Arch Gen Psychiatry 2004;61:1042–1049
25. Katon WJ, Lin EH, Von Korff M, et al. Collaborative care for patients with depression and chronic illnesses. N Engl J Med 2010;363: 2611–2620
26. Relton C, Torgerson D, O'Cathain A, Nicholl J. Rethinking pragmatic randomised controlled trials: introducing the "cohort multiple randomised controlled trial" design. BMJ 2010; 340:c1066
27. Rittenhouse DR, Shortell SM. The patient-centered medical home: will it stand the test of health reform? JAMA 2009;301:2038–2040
28. Majumdar SR, Rowe BH, Folk D, et al. A controlled trial to increase detection and treatment of osteoporosis in older patients with a wrist fracture. Ann Intern Med 2004;141:366–373
29. Cochrane Effective Practice and Organisation of Care Group. EPOC Author Resources [Internet]. Available from http://epoc.cochrane.org/epoc-author-resources. Accessed 2 February 2014.
30. Kroenke K, Strine TW, Spitzer RL, Williams JBW, Berry JT, Mokdad AH. The PHQ-8 as a measure of current depression in the general population. J Affect Disord 2009;114:163–173
31. Manea L, Gilbody S, McMillan D. Optimal cut-off score for diagnosing depression with the Patient Health Questionnaire (PHQ-9): a meta-analysis. CMAJ 2012;184:E191–E196
32. Majumdar SR, Johnson JA, Bowker S, et al. A Canadian consensus for the standardized evaluation of quality improvement interventions in type 2 diabetes. Can J Diabetes 2005;29:220–229
33. Johnson JA, Maddigan SL. Performance of the RAND-12 and SF-12 summary scores in type 2 diabetes. Qual Life Res 2004;13:449–456
34. van Hout B, Janssen MF, Feng YS, et al. Interim scoring for the EQ-5D-5L: mapping the EQ-5D-5L to EQ-5D-3L value sets. Value Health 2012;15:708–715
35. McGuire BE, Morrison TG, Hermanns N, et al. Short-form measures of diabetes-related emotional distress: the Problem Areas in Diabetes Scale (PAID)-5 and PAID-1. Diabetologia 2010;53:66–69
36. Glasgow RE, Whitesides H, Nelson CC, King DK. Use of the Patient Assessment of Chronic Illness Care (PACIC) with diabetic patients: relationship to patient characteristics, receipt of care, and self-management. Diabetes Care 2005;28:2655–2661
37. Gugiu PC, Coryn C, Clark R, Kuehn A. Development and evaluation of the short version of the Patient Assessment of Chronic Illness Care instrument. Chronic Illn 2009;5:268–276
38. Katon W, Unützer J, Fan M-Y, et al. Cost-effectiveness and net benefit of enhanced treatment of depression for older adults with diabetes and depression. Diabetes Care 2006;29:265–270
39. Katon W, Russo J, Lin EHB, et al. Cost-effectiveness of a multicondition collaborative care intervention: a randomized controlled trial. Arch Gen Psychiatry 2012;69:506–514
40. Ducat L, Philipson LH, Anderson BJ. The mental health comorbidities of diabetes. JAMA 2014;312:691–692