Online-Only Supplemental Materials

Table S1. Description and psychometric properties of study measures

Construct	Measure	Description	Validity [a]	Reliability [b]
Medication adherence	Adherence to Refills and Medications Scale for Diabetes (ARMS-D)	11-item measure asking about medication taking and refill adherence. Responses range from 1="none of the time" to 4="all of the time"; summed to produce a score ranging from 11-44; we reserve scored items so higher scores indicate better adherence.	Validated against other self-report measures and objective refill adherence measures (1); Independently predicts HbA1c (2)	Cronbach's α = 0.74
Medication adherence	Summary of Diabetes Self-Care Activities - medications subscale (SDSCA-MS)	2-item measure asking separately for each prescribed diabetes medication: "On how many of the last seven days did you take this medication?" and "On how many of the last seven days did you take the correct number of (pills/injections) for this medication?" Responses range from 0–7; responses averaged; higher scores indicate better adherence.	Correlates with other self-report measures of medication adherence (2); Independently predicts HbA1c (2)	Average interitem correlation across medications queried: 0.80
Physical Activity	International Physical Activity Questionnaire - short form (IPAQ-SF)	2 items asking separately about days and time spent doing different levels of physical activity (vigorous, moderate, and light). Scoring instructions result in a total score in MET minutes/week.	Correlates with accelerometer data (3) and with other objective and subjective physical activity measures (4)	Review reports test-retest reliability >0.65 (3) [c]
Dietary behavior	Personal Diabetes Questionnaire - subscale assessing use of dietary information for decision making (PDQ)	3 items asking about frequency of using information on the number of calories, carbohydrates, and grams of fat in foods to make decisions about what to eat. Responses range from 1="never" to 6="1 or more times per day"; reverse scored as appropriate and averaged; higher scores indicate	Correlates with other self-report diet measures (5) and with HbA1c (6)	Cronbach's α = 0.83

		more use of information to make dietary decisions.		
Diabetes self-efficacy	Perceived Diabetes Self-Management Scale, 4-item version (PDSMS-4)	4 items asking about confidence in managing diabetes. Responses range from 1="strongly disagree" to 5="strongly agree"; reverse scored as appropriate and summed to produce a score ranging from 5-20; higher scores indicate better self-efficacy.	Full 8-item measure **c**orrelates with self-reported self-care activities and measures of HbA1c (7)	Cronbach's $\alpha = 0.68$

HbA1c, Hemoglobin A1c; MET, metabolic equivalent of task

[a] Based on prior studies

[b] Internal consistency of baseline measure in this study

[c] Internal consistency not relevant because of the different types of activity assessed

Table S2. Most common "top 4" barriers with associated prevalence and averaged scores for the REACH and Control groups

Barrier to diabetes medication adherence	REACH Group		Control Group	
	n (%) ranking barrier in top 4	Mean (SD)	n (%) ranking barrier in top 4	Mean (SD)
I'm disappointed when my medicine doesn't improve my diabetes right away.	66 (26.1%)	6.5 (3.0)	63 (24.9%)	6.3 (2.7)
I think brand name medicine works better than generic medicine.	65 (27.7%)	7.0 (2.8)	59 (23.3%)	6.9 (3.0)
I'm afraid of experiencing a side effect from my diabetes medicine.	63 (24.9%)	6.6 (2.8)	43 (17.0%)	6.7 (2.8)
I forget to take my medicine.	53 (20.9%)	4.4 (2.7)	52 (20.6%)	4.3 (2.7)
I worry that taking diabetes medicines for a long time will be bad for me.	51 (20.2%)	7.2 (3.0)	49 (19.4%)	6.7 (2.4)
I feel burned out with having to take diabetes medicines.	52 (20.6%)	6.8 (2.3)	48 (19.0%)	6.9 (2.6)

Note: Mean (SD) presents statistics for the barrier score among the participants for whom it was scored as a top 4 barrier on a scale from 1 = "never" to 10 = "a lot."

Table S3: Point estimates, 95% CIs, and *p*-values for coefficients of HbA1c GEE model

Coefficient	Estimate	95% CI	*p* value
Intercept	3.501	[2.34, 4.66]	< 0.001
REACH	0.249	[-1.16, 1.66]	0.729
Month 6	-0.531	[-1.57, 0.506]	0.316
Month 12	1.002	[-0.396, 2.40]	0.160
Month 15	1.388	[-0.503, 3.28]	0.150
$HbA1c_0$	0.586	[0.442, 0.731]	< 0.001
REACH x Month 6	1.005	[-0.298, 2.31]	0.131
REACH x Month 12	-0.355	[-2.18, 1.47]	0.703
REACH x Month 15	-0.702	[-2.87, 1.47]	0.526
REACH x $HbA1c_0$	-0.0598	[-0.233, 0.114]	0.500
Month 6 x $HbA1c_0$	0.0713	[-0.0539, 0.196]	0.265
Month 12 x $HbA1c_0$	-0.0941	[-0.264, 0.0759]	0.278
Month 15 x $HbA1c_0$	-0.150	[-0.383, 0.0834]	0.208
REACH x Month 6 x $HbA1c_0$	-0.123	[-0.276, 0.0304]	0.116
REACH x Month 12 x $HbA1c_0$	0.0610	[-0.158, 0.280]	0.585
REACH x Month 15 x $HbA1c_0$	0.105	[-0.161, 0.370]	0.440

$HbA1c_0$, baseline HbA1c
p-value for overall REACH effect: 0.260

Table S4: Point estimates, 95% CIs, and *p*-values for coefficients of HbA1c GEE model restricted to those with baseline HbA1c≥8.5% at baseline (n=219)

Coefficient	Estimate	95% CI	*p* value
Intercept	5.120	[3.696, 6.544]	< 0.001
REACH	-0.435	[-0.839, -0.031]	0.035
Month 6	0.228	[-0.147, 0.602]	0.233
Month 12	0.172	[-0.278, 0.621]	0.455
$HbA1c_0$	0.433	[0.290, 0.576]	< 0.001
REACH x Month 6	-0.302	[-0.737, 0.134]	0.175
REACH x Month 12	0.217	[-0.366, 0.800]	0.466

$HbA1c_0$, baseline HbA1c
p-value for overall REACH effect: 0.0364

Additional Detail on Methods & Analyses

Randomization

Within 1 week of completing all enrollment procedures, participants were randomized 1:1 to receive the REACH intervention or control. A second randomization, among those who were assigned REACH in the first randomization, assigned 1:1 the additional FAMS intervention or no additional intervention; yielding a 2:1:1 (control : REACH : REACH+FAMS) randomized design. Dr. Greevy, who had no participant contact and no role in recruitment in the study, executed the randomization algorithm using the statistical software program R (v.3.5.1). Given the nature of the interventions, the research assistants who interacted with the participants and the participants themselves were aware of what treatments they were receiving.

Treatment assignments were randomly allocated via sequential rematched randomization (8). This form of restricted randomization is a variation of optimal matched randomization (9) and matching on-the-fly (10). It has been shown to achieve, on average, much better balance in covariate distributions between treatment arms than simple randomization or conventional stratified randomization (8). Explained briefly, a nearly optimal stratified randomization would create N/2 strata for N participants, where each stratum contained exactly two participants who were similar, but rarely identical, in their covariates. Within each stratum, one participant would be randomized to the intervention and the other to control. Matching subjects prior to randomization creates this nearly optimal stratification. Sequential rematched randomization redefines these strata at each stage of the randomization under the constraint that participants never change their treatment assignments and like assignments cannot match to each other. The matching was based on similarity in baseline HbA1c, insulin status, race, age, duration of diabetes, gender, income, and education via a reweighted Mahalanobis distance (11). Only these

covariates are directly balanced by the algorithm. Covariates that are highly correlated with them will be indirectly balanced. Covariates that are fairly independent of them will be balanced as well as they would be with simple randomization. That is, if using a *p*-value as a measure of covariate balance, we would expect a fairly independent covariate not included in the matching algorithm to have about a 5% chance of being unbalanced at a p-value < 0.05 level. Just as with a trial using simple randomization, direct covariate adjustment or inverse probability of treatment weighting may be used to account for covariates that are unbalanced by chance.

Subgroup Analyses

Analyses. Data visualizations suggested effect modification by baseline HbA1c, which is visually evidenced by Figure 2B in which those with a higher baseline HbA1c demonstrate a larger estimated effect at 3 and 6 months than those with a lower baseline HbA1c. To explore additional subgroup effects in a context maximizing our ability to detect such differences, this supplement includes analyses based a subset of data including participants with a baseline HbA1c≥8.5% (69 mmol/mol; n=219), which was the approximate mean value for baseline HbA1c. We sought to evaluate whether there was evidence of effect modification by participant or intervention characteristics. Participant characteristics of interest included minority race/ethnicity (including any participant who was not non-Hispanic White) and an indicator of socioeconomic disadvantage. Participants were considered disadvantaged if they reported meeting one or more of the following criteria: annual household income <$25,000, years of educational attainment <12, homeless or uninsured at baseline. Among those with HbA1c≥8.5%, 61% were classified as minority and 63% were classified as disadvantaged. Intervention characteristics of interest included random assignment to REACH + FAMS as compared to REACH only, and text message frequency choice at 6 months (i.e., low-dose vs. not).

Ordinary least squares linear regression was used to address each of these questions, using Huber-White (12) standard errors to obtain point estimates and CIs, and including linear adjustment for baseline HbA1c in all models. For the analysis of participant characteristics, we first stratified the chained-equations imputation procedure according to the four induced minority/disadvantaged status categories in order to avoid biasing evidence of effect modification toward the null.

Results. We found no evidence of effect modification either by minority (interaction term -0.17, p=0.753) nor disadvantaged status (interaction term -0.37, p=0.489).

We found evidence of effects of both REACH only (-0.75%; 95% CI: [-1.38%, -0.12%]) and REACH + FAMS (-0.65%; 95% CI: [-1.26%, -0.05%]). We did not find sufficient evidence of 12-month effects for either REACH only (-0.31; 95% CI: [-1.00%, 0.39%]) or REACH + FAMS (-0.31%; 95% CI: [-0.96%, 0.51%]). Moreover, we did not find evidence of an additive effect of FAMS beyond REACH at 6 or 12 months. After 6 months, 56% of participants chose low-dose and 44% chose to continue receiving daily texts. We did not find evidence that the 12-month mean HbA1c differed between individuals based on their frequency choice after 6 months. There was no effect of choosing low-dose on HbA1c at 12 months (0.24%; 95% CI: [-0.54%, 1.02%]).

Weighting Theory-Based Barriers to Adherence

Analyses. To understand treatment effects on the barrier sum scores, we needed to account for imbalance in baseline barrier sum scores across conditions. We employed inverse probability of treatment weighting based on predictions from a logistic propensity score model including the following covariates: baseline HbA1c, gender, age, education, and baseline barrier sum. Each continuous covariate included cubic splines with two knots placed at the first and

second tertiles.

To maximize our ability to detect mediation of barrier scores, we examined mediation of the 3-month barrier scores on 6-month outcomes SDSCA-MS and HbA1c. For each mediation analysis, we fit a total effect model and a mediation model. We used Rubin's imputation rules to get an estimate and confidence interval for each. We checked out total effect estimates against those obtained in the GEE analyses. We determined a bootstrapped error by bootstrapping each model. The HbA1c mediation model was restricted to participants with baseline HbA1c≥8.5% (69 mmol/mol; n=219), as detailed in analyses above.

Results. We found evidence of a significant treatment effect on barrier sum scores for the first three months of the intervention period (-1.98; 95% CI: [-3.56, -0.39]), although we are not able to conclude evidence of an effect at 6 months (-1.27; 95% CI: [-3.05, 0.50]). We did not see subsequent treatment effects on barriers addressed by the intervention from months 3-6 or months 6-12. Total effects analyses were similar to the GEE model estimates of effects at 6 months, but with wider confidence intervals as expected due to the propensity scores. The evidence of indirect effects was weak, with endpoints of the confidence intervals leading to very different conclusions. We could not conclude a presence nor absence of mediation on SDSCA-MS or HbA1c at 6 months. Total effect on SDSCA-MS in this analysis was 0.444, 95% CI [0.218, 0.670], and indirect effect was 0.0658; bootstrapped 95% CI [-0.046, 0.178]. Total effect on HbA1c, restricted to those with elevated baseline HbA1c, was –0.743; 95% CI [–1.480, –0.011], and indirect effect was 0.0218; bootstrapped 95% CI [–0.271, 0.315].

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