The association of financial incentives for low density lipoprotein cholesterol reduction with patient activation and motivation

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ABSTRACT

There is growing interest in using financial incentives for patients to improve medication adherence, but few studies have evaluated whether financial incentives are associated with patients’ activation and motivation. We analyzed survey data collected as part of a randomized clinical trial conducted from 2011 to 2014 of four financial incentive interventions to reduce low density lipoprotein cholesterol (LDL-C) among patients at risk for atherosclerotic cardiovascular disease. The main trial included 1503 patients aged 18–80 and recruited from primary care practices affiliated with three health systems. Participants were randomized into four groups: patient financial incentives, primary care physicians (PCPs) incentives, patients and PCPs shared incentives, or no incentives for LDL-C control. Patient Activation Measure (PAM) and Treatment Self Regulation Questionnaire (TSRQ) surveys were administered at baseline and 12 months. Clinical outcomes were change in LDL-C at 12 and 15 months and average medication adherence as measured by electronic pill bottle opening. Mean changes in PAM and TSRQ scores were compared between patients eligible and not eligible for incentives. Clinical outcomes were tested against baseline and change in psychosocial measures using bivariate and multivariate regression. Change in PAM score and TSRQ autonomous subscore did not differ significantly between patients eligible and not eligible for incentives. Lower baseline and greater increase in TSRQ autonomous subscore were predictive of greater 15-month decrease in LDL-C. A financial incentive intervention to improve LDL-C control was not associated with changes in patients’ activation or autonomous motivation. Increases in patient autonomous motivation are predictive of long-term LDL-C control.

1. Introduction

Atherosclerotic cardiovascular disease (ASCVD) remains the leading cause of mortality among US adults. (National Center for Health Statistics. Health, United States, 2016) HMG-CoA reductase inhibitors (statins) have been shown to lower cholesterol and reduce the risk of myocardial infarction by about 30%. (Baigent et al., 2005; Downs et al., 1998; Pedersen et al., 2004; Ridker et al., 2009; Sacks et al., 1996; The Long-Term Intervention with Pravastatin in Ischaemic Disease (LIPID) Study Group, 1998) but adherence to statins is poor; 25–50% of patients prescribed statins discontinue them within 6 months to 1 year, and nonadherence at the end of 2 years is as high as 75%. (Brown and Russell, 2011) High nonadherence rates have been associated with significantly increased risk of cardiovascular-related hospitalizations and mortality. (Ho et al., 2009)

There has been growing interest in using direct financial incentives for patients to improve health behaviors such as medication adherence. (King et al., 2013; Loewenstein et al., 2013; Rice, 2013) Increasing numbers of studies have found that financial incentives can improve adherence to medications for preventing clotting or for reducing risk from ASCVD, HIV, latent tuberculosis, drug or alcohol dependence, or psychotic disorders. (DeFulio and Silverman, 2012; Kimmel et al., 2012; Noordraven et al., 2017; Volpp et al., 2008) However, few studies have evaluated whether financial incentives to improve medication adherence either are influenced by patients’ underlying motivation or activation, or whether motivation and activation change in the context of such an intervention. This could be important in at least two ways.

First, financial incentives may alter patients’ psychosocial states over the course of an intervention. In particular, there is concern that financial incentives may “crowd out” intrinsic motivation, such that levels of the desired behavior drop below baseline levels once financial incentives are removed. (Promberger and Marteau, 2013) To our
knowledge, only two previous studies have examined the impact of financial incentives for health-related behaviors on direct measures of intrinsic motivation. These studies found no evidence of crowding out of intrinsic motivation from financial incentive interventions for abstinence from substance abuse or for weight loss. (Ledgerwood and Petry, 2006; Sen et al., 2014)

Second, a number of psychosocial measures may be predictive of medication adherence. These include the Patient Activation Measure (PAM) and the Treatment Self Regulation Questionnaire (TSRQ). The PAM score measures a patient’s knowledge, skills, beliefs, and behaviors to manage their chronic condition. (Hibbard et al., 2004) The TSRQ is based on self-determination theory and measures various forms of a patient’s motivation. (Levesque et al., 2007) While the PAM and TSRQ measures were not designed to directly predict medication adherence, they have been associated with medication adherence in a number of chronic conditions. (Parchman et al., 2010; Umeukeje et al., 2015; Williams et al., 1998; Williams et al., 2009)

To date, most studies have relied on self-reported measures of medication adherence, which may be associated with recall biases. Fewer have relied on medication event monitoring systems (MEMS), which electronically document every time a medication bottle is opened and may account for some of the recall biases associated with self-reported measures. (Garfield et al., 2011)

The present exploratory analysis, done in the context of a study of financial incentives to improve low density lipoprotein cholesterol (LDL-C) control among a high-risk patient population, (Asch et al., 2015) aims to examine two main issues:

1. Are patients’ activation and motivation, as measured by PAM and TSRQ scores, associated with participation in a trial of financial incentives to improve LDL-C?
2. Are patients’ baseline or change in activation or motivation predictive of medication adherence or LDL-C control?

2. Materials and methods

2.1. Design and intervention

This study is an exploratory analysis of survey data collected as part of a multicenter, cluster-randomized, controlled trial of four financial incentive interventions to reduce LDL-C among patients with ASCVD risk (NCT01346189). A detailed description of the main trial is described elsewhere. (Asch et al., 2015) The study was reviewed by the Institutional Review Board at the main study institution. In brief, 340 primary care physicians (PCPs) and 1503 of their patients were randomized to one of four arms: physician incentives, patient incentives, shared physician-patient incentives, or control (no financial incentives). Patients were assigned quarterly goals to reduce their LDL-C by 10 mg/dl or more from the previous quarter’s target, or to achieve or maintain an LDL-C goal that varied by baseline risk (< 100 mg/dl for high risk patients, < 130 mg/dl for medium risk patients). The maximum amount that a patient in the patient incentive arm was eligible to receive was $1022 per year; this amount was halved in the shared incentives arm.

2.2. Sample

In the underlying cluster-randomized trial, recruitment of a PCP made their patients potentially eligible and assigned to the arm to which their PCP was randomized. Patients were recruited from the practices of participating PCPs from 3 health systems. Inclusion criteria included being aged 18 to 80; having a designated consenting PCP; and having a 10-year Framingham Risk Score (FRS) (Wilson et al., 1998) ≥ 20% or coronary artery disease equivalents with LDL-C ≥ 120 mg/dl (high risk patients); or a FRS of 10–20% with LDL-C ≥ 140 (medium risk patients).

Eligible patients provided informed consent and enrolled either by phone or online via the Way to Health platform, a web-based platform developed at one study institution for facilitating behavioral intervention studies. Baseline data, including demographics, socioeconomic status, and current medication usage, were collected from all patients at enrollment.

2.3. Psychosocial measures

The PAM and TSRQ survey instruments were administered at baseline and at 12 months either by phone or online via the Way to Health platform. Survey data were used to calculate PAM scores and TSRQ subscores.

The PAM-13 instrument measures a patient’s level of “activation,” defined as having the beliefs, knowledge, skills, and behavioral repertoire to successfully manage his/her chronic condition. It represents a validated short form of the original 22-item PAM questionnaire. (Hibbard et al., 2005) Responses are on a 4-point scale ranging from 1 (“strongly disagree”) to 4 (“strongly agree”), and are summed to produce a raw score ranging from 13 to 52. Raw scores are converted into activation scores ranging from 0 to 100, and then further classified into PAM levels: 1 (“believing the patient role is important,” score ≤ 47.0), 2 (“having the confidence and knowledge necessary to take action,” score of 47.1 to 55.1), 3 (“actually taking action to maintain and improve one’s health,” score of 55.2 to 67.0), or 4 (“staying the course even under stress,” score ≥ 67.1).

The TSRQ is a 15-item instrument that measures motivation based on self-determination theory, which proposes that autonomy is an essential factor for achieving sustainable behavior change. (Levesque et al., 2007) Responses are on a 7-point scale ranging from 1 (“not at all true”) to 7 (“very true”), and were used to calculate three subscores: “amotivation” (the absence of motivation); “controlled regulation” (behavior performed to obtain a reward or to avoid negative consequences or feeling guilty); and “autonomous regulation” (behavior performed because it is valued by the individual, perceived as part of the larger self, or engaged for their own sake), a category that includes intrinsic motivation. Subscores were calculated as the mean of the relevant questions, and range from 1 to 7. Higher scores on autonomous regulation are associated with positive health behaviors such as fruit and vegetable consumption, physical activity, glycemic control, long term abstinence from tobacco, and reduction in LDL-C, while higher scores on amotivation and controlled regulation are associated with negative health outcomes such as depression. (Levesque et al., 2007; Williams et al., 2004; Williams et al., 2006) Analysis of TSRQ was limited to the 33% of study participants who self-reported taking cholesterol-lowering medications at baseline, as TSRQ questions were focused on behaviors related to taking medications.

2.4. Clinical endpoints

The primary clinical endpoint was change in LDL-C from baseline to 12 months. Secondary endpoints included change in LDL-C from baseline to 15 months and average medication adherence, defined as the percentage of days that patients took their prescribed medications. Patients’ average daily adherence was measured using an electronic pill bottle (Vitality* GlowCaps), assuming that each time the electronic pill bottle was opened, a dose was taken.

2.5. Analysis

The patient was the unit of analysis for all outcomes. Demographic and baseline psychosocial measures were compared across the four study arms (ANOVA F test for continuous variables; Pearson’s chi-squared test or Fisher’s exact test for categorical variables).

To examine study question 1, the mean changes in PAM and TSRQ subscores (Student’s t-test) were compared between patients who were
eligible to receive incentives (patient and shared arms) and those who were not (physician and control arms).

To examine study question 2, the primary and secondary clinical outcomes were tested against the baseline and change in psychosocial measures (Wilcoxon rank sum test, Kendall trend test). Bivariate regression models were created with baseline and change in PAM level and TSRQ subscores as predictors, and 12-month change in LDL-C, 15-month change in LDL-C, and average adherence as dependent variables. Because the TSRQ autonomous regulation, controlled regulation, and amotivation subscores showed significant correlation, only the autonomous subscore was included in multivariate regression. Six multivariate regression models were created, with either baseline or change in PAM score and TSRQ autonomous subscore as predictors, and with either 12-month change in LDL-C, 15-month change in LDL-C, or average adherence as the dependent variable, controlling for various demographic and other factors.1

Analyses were conducted using SAS v9.3 (SAS Institute Inc., Cary, NC) and Stata 13.9 (StataCorp, College Station, TX).

3. Results

Of the 1503 patients randomized, 1496 completed baseline surveys. Demographic characteristics across study groups were similar (detailed data included in full randomized trial). (Asch et al., 2015) PAM score and TSRQ subscores were similar across study groups at baseline (Table 1). 84% of patients were at the highest PAM level, and 45% of patients were at the highest autonomous motivation subscore, with an average autonomous motivation subscore of 6.5 out of 7.

From baseline to 12 months, the PAM score and the TSRQ autonomous subscore both increased among patients eligible for incentives. However, the changes in PAM and TSRQ scores were not substantially different between patients who were and were not eligible for incentives (PAM score +1.3 among eligible vs +0.9 among not eligible; TSRQ autonomous subscore +0.13 eligible vs −0.02 among not eligible).

In bivariate analyses, associations were observed between patients' baseline or change in psychosocial measures and their clinical outcomes (Table 2). Patients who experienced increases in their PAM level had greater decreases in LDL-C at 12 months and 15 months. Patients with lower baseline TSRQ autonomous subscores, as well as patients who experienced increases in their TSRQ autonomous subscore, had greater decreases in LDL-C at 15 months.

In multivariable regression of clinical outcomes on PAM scores and TSRQ autonomous subscores, lower baseline TSRQ autonomous subscores and greater increase in TSRQ autonomous subscores were associated with greater 15-month decrease in LDL-C. Neither baseline nor change in PAM scores was associated with 12-month change in LDL-C, 15-month change in LDL-C, or average adherence (Tables 3 and 4). Tests of multicollinearity between PAM and TSRQ subscores revealed low-to-moderate levels of correlation (Spearman correlation coefficient 0.30 for baseline measures and 0.29 for change in measures).

4. Discussion

The present study, done in the context of a financial incentive intervention to improve LDL-C control among patients at high cardiovascular risk, aimed to examine two main issues: 1) are patients' activation or motivation, as measured by PAM and TSRQ scores, associated with participation in a trial of financial incentives to improve LDL-C control? and 2) are patients' baseline or change in activation or motivation predictive of long-term medication adherence or LDL-C control?

With regard to the first issue, the results indicate that neither patient activation nor autonomous motivation changed substantially over the course of the intervention. While an increase in PAM score was observed, the magnitude of the change was small. Furthermore, changes did not differ between patients who did and did not receive financial incentives, which suggests that financial incentives do not explain the small increases that were observed. However, participants had very high levels of both scores on enrollment. This may have created a ceiling effect that reduced the statistical power to detect meaningful changes.

Qualitative studies of financial incentives for smoking cessation have found that participants do not generally perceive that incentives directly increased their motivation to quit, even when the incentives significantly increased quit rates in a randomized trial. (van den Brand et al., 2018a; Van den Brand et al., 2018b) Rather, they attributed their success to their existing intrinsic motivation and saw the incentives as nice bonuses. It has been postulated that health-related behaviors represent behaviors for which individuals have high stated motivation, but face self-control or external constraints in maintaining those behaviors over time. As such, rather than increasing motivation, financial rewards may help maintain levels of intrinsic motivation over time by providing an immediate and tangible benefit to participating in a behavior the individual would like to do more of, consistent with what was observed in our study.

It is also important to note that there was no evidence that financial incentives reduced patient activation or autonomous motivation. When patient financial incentives are proposed as a means of increasing healthy behavior, one concern that is often raised is that extrinsic motivation may crowd out intrinsic motivation. (Frey and Jegen, 2001; Lepper et al., 1973) Results from this study suggest that the “crowding out” phenomenon does not apply to patients being given incentives to reduce their cholesterol by taking statins.

While these results do not support the notion that financial incentives increase activation and motivation, it is important to note that financial incentives have been shown to directly improve clinical outcomes in some circumstances. The main randomized trial found that patients in the shared incentives arm had significantly better LDL-C control over 12 months compared to patients in the control arm, and a subsequent analysis found that shared financial incentives were cost-effective based on cost per quality-adjusted life-year. (Asch et al., 2015; Pandya et al., 2018) Financial incentives have also been shown to improve anticoagulation control, smoking cessation, physical activity, and other health outcomes. (Kimmel et al., 2012; Halpern et al., 2015; Patel et al., 2016)

With regard to the second study issue, neither baseline nor change in activation or autonomous motivation were predictive of 12-month change in LDL-C, the primary outcome. However, autonomous motivation was predictive of 15-month change in LDL-C. Specifically, lower baseline autonomous motivation and greater increase in autonomous motivation over the intervention period were associated with greater decreases in LDL-C at 15 months. Trends for patient activation were similar to the pattern observed for autonomous motivation, in that lower baseline activation and greater increases in activation over the intervention period were correlated with greater decreases in LDL-C. Taking into consideration the high levels of activation and motivation at baseline, this would be consistent with a strong ceiling effect, in which a subset of study participants with lower baseline levels had room to experience increases in activation or motivation scores, and these participants tended to experience greater decreases in LDL-C. It is unclear why findings were only significant for 15-month change in LDL-C; similar trends were observed for 12-month change in LDL-C, and it may be that more time was needed for a substantial relationship to manifest.

Prior studies have correlated high baseline levels of autonomous...
motivation with outcomes such as medication adherence and cholesterol control. (Umeukeje et al., 2015; Williams et al., 2009) However, our study is different in that we examined both baseline and change in autonomous motivation over a one-year period, and found that greater increases in autonomous motivation, rather than high baseline levels of autonomous motivation, were associated with better long-term LDL-C control. While the results of the main trial indicate that financial incentives can produce statistically significant improvements in LDL-C control independent of changes in motivation, medication adherence rates remained low (mean 39% over 12 months in the shared incentives group). Our results raise the question of whether greater improvements could be seen with interventions that successfully increase participants’ autonomous motivation. Further studies that design interventions specifically geared toward increasing autonomous motivation or focusing on participants with low baseline levels of autonomous motivation may help answer this question.

Strengths of the present study include its cluster randomized design, the use of electronic pill bottles to objectively measure adherence, and the use of two validated psychosocial measures. However, this study has some limitations. First, the patient sample was predominantly white and well educated, which limits the generalizability of our findings. Second, our participants had extremely high levels of motivation at baseline and this limited our ability to use variability in baseline motivation to predict performance. Third, our analysis included multiple comparisons, which raises the possibility that our significant findings may have been due to chance. However, the fact that both baseline and change in autonomous motivation were associated with LDL-C control and that a similar trend was seen for baseline and change in activation, may have been due to chance alone. Finally, we did not control or measure the nature of the patient-physician interactions in which the rewards were administered. Self-determination theory postulates that the interpersonal context in which rewards are administered may modulate the effect of the rewards on intrinsic motivation.

5. Conclusions

A financial incentive intervention to improve LDL-C management among patients at high cardiovascular risk was not associated with changes in patients’ measured activation or autonomous motivation. However, greater increases in autonomous motivation over the course of the intervention were predictive of better long-term LDL-C control.

Table 1
Patient baseline psychosocial measures.

| Study group | Total (n = 1496) | Shared incentives (n = 343) | Patient incentives (n = 358) | Physician incentives (n = 430) | Control (n = 365) |
|-------------|-----------------|-----------------------------|-----------------------------|-------------------------------|-----------------|
| PAM score: mean (SD) | 79.1 (13.1) | 79.5 (13.3) | 78.3 (14.2) | 79.3 (12.9) | 79.1 (12.1) |
| PAM level: n (%) | | | | | |
| Starting to take a role | 22 (1.5) | 5 (1.5) | 8 (2.2) | 7 (1.6) | 2 (0.6) |
| Building knowledge and confidence | 36 (2.4) | 8 (2.3) | 12 (3.4) | 9 (2.1) | 7 (1.9) |
| Taking action | 178 (11.9) | 40 (11.7) | 51 (14.3) | 43 (10.0) | 44 (12.1) |
| Maintaining behaviors | 1260 (84.2) | 290 (84.6) | 287 (80.2) | 371 (86.3) | 312 (85.5) |
| TSRQ subscores: mean (SD) | | | | | |
| Autonomous | 6.5 (0.8) | 6.5 (1.0) | 6.5 (0.7) | 6.7 (0.6) | 6.5 (0.9) |
| Controlled | 3.2 (1.7) | 3.2 (1.7) | 3.1 (1.7) | 3.3 (1.8) | 3.1 (1.8) |
| Amotivation | 2.7 (1.6) | 2.8 (1.6) | 2.6 (1.6) | 2.7 (1.6) | 2.8 (1.6) |

Abbreviations: TSRQ, Treatment Self Regulation Questionnaire; PAM, patient activation measure.

a PAM score ranges from 0 to 100, and is classified into one of four levels: starting to take a role (level 1, score ≤ 47.0), building knowledge and confidence (level 2, score of 47.1 to 55.1), taking action (level 3, score of 55.2 to 67.0), and maintaining behaviors (level 4, score ≥ 67.1).

b TSRQ subscores range from 1 to 7, and are defined as: “amotivation” (the absence of motivation); “controlled regulation” (behavior performed to obtain a reward or to avoid negative consequences or feeling guilty); and “autonomous regulation” (behavior performed because it is valued by the individual, perceived as part of the larger self, or engaged for their own sake).

N = 498 for TSRQ subscores, limited to patients taking statins at baseline.

Table 2
Change in LDL-C and average adherence stratified by baseline and change in psychosocial measures.

| Study group | Mean 12-month change in LDL-C (SD) | Mean 15-month change in LDL-C (SD) | Average adherence (SD) |
|-------------|-------------------------------------|-------------------------------------|-----------------------|
| Baseline PAM level | | | |
| Starting to take a role | −35.9 (37.6) | −41.2 (31.5) | 0.31 (0.34) |
| Building knowledge and confidence | −33.8 (45.8) | −33.0 (41.9) | 0.31 (0.34) |
| Taking action | −29.5 (39.1) | −27.8 (39.1) | 0.34 (0.35) |
| Maintaining behaviors | −27.2 (37.4) | −27.5 (37.6) | 0.32 (0.34) |
| Change in PAM level | | | |
| Lower | −21.8 (37.0) | −24.9 (36.5) | 0.20 (0.33) |
| No change | −27.5 (37.7) | −27.5 (38.3) | 0.34 (0.34) |
| Higher | −34.3 (39.1) | −33.9 (38.4) | 0.36 (0.36) |
| Baseline TSRQ autonomous subscore | | | |
| < 7 | −28.2 (36.9) | −30.4 (39.2) | 0.53 (0.29) |
| 7 | −25.8 (41.9) | −21.5 (41.8) | 0.50 (0.31) |
| Change in TSRQ autonomous subscore | | | |
| Lower | −24.2 (43.3) | −18.4 (47.9) | 0.51 (0.29) |
| No change | −26.0 (37.9) | −23.7 (38.1) | 0.57 (0.29) |
| Higher | −29.5 (38.5) | −32.4 (38.5) | 0.56 (0.29) |

LDL-C low density lipoprotein cholesterol; PAM patient activation measure; TSRQ Treatment Self Regulation Questionnaire.

* PAM score ranges from 0 to 100, and is classified into one of four levels: starting to take a role (level 1, score ≤ 47.0), building knowledge and confidence (level 2, score of 47.1 to 55.1), taking action (level 3, score of 55.2 to 67.0), and maintaining behaviors (level 4, score ≥ 67.1). Individual participants were assigned to “lower”, “no change”, or “higher” categories based on whether their PAM level changed from baseline to 12 months.

† TSRQ subscores range from 1 to 7, and are defined as: “amotivation” (the absence of motivation); “controlled regulation” (behavior performed to obtain a reward or to avoid negative consequences or feeling guilty); and “autonomous regulation” (behavior performed because it is valued by the individual, perceived as part of the larger self, or engaged for their own sake). Individual participants were assigned to “lower”, “no change”, or “higher” categories based on whether their TSRQ autonomous subscore changed or remained in the same range (< 7 vs 7) from baseline to 12 months.

‡ Average adherence defined as the percentage of days that patients took their prescribed medications. Average adherence differs between patients subdivided by PAM score and by TSRQ score because TSRQ analyses only included patients who were already taking statins at baseline (N = 498).

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Table 3
Multivariable regression model of 12-month change in LDL-C, 15-month change in LDL-C, and average adherence on baseline psychosocial measures.

| Parameter                                | 12-Month change in LDL-C | 15-Month change in LDL-C | Average adherence |
|------------------------------------------|--------------------------|--------------------------|------------------|
| **Baseline PAM score**                   | 0.10 (−0.16, 0.36)       | 0.13 (−0.15, 0.41)       | 0.00 (0.00, 0.00) |
| **Baseline TSRQ autonomous subscore**     | 2.23 (−2.11, 6.57)       | 4.72 (0.41, 9.04)        | −0.01 (−0.04, 0.02) |

Abbreviations: LDL-C, low density lipoprotein cholesterol; PAM, patient activation measure; TSRQ, Treatment Self Regulation Questionnaire.

Table 4
Multivariable regression model of 12-month change in LDL-C, 15-month change in LDL-C, and average adherence on change in psychosocial measures.

| Parameter                                | 12-Month change in LDL-C | 15-Month change in LDL-C | Average adherence |
|------------------------------------------|--------------------------|--------------------------|------------------|
| **Change in PAM score**                  | −0.01 (−0.24, 0.23)      | 0.18 (−0.10, 0.47)       | 0.00 (0.00, 0.00) |
| **Change in TSRQ autonomous subscore**   | −3.02 (−7.39, 1.35)      | −9.30 (−14.59, −4.01)    | 0.01 (−0.03, 0.04) |

Abbreviations: LDL-C, low density lipoprotein cholesterol; PAM, patient activation measure; TSRQ, Treatment Self Regulation Questionnaire.

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