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Moderating Effects of Patient Characteristics on the Impact of Financial Incentives

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Abstract

While financial incentives to providers or patients are increasingly common as a quality improvement strategy, their impact on patient subgroups and healthcare disparities is unclear. To examine these patterns, we analyzed data from a randomized clinical trial of financial incentives to lower low-density lipoprotein (LDL) cholesterol levels in patients at risk for cardiovascular disease. Patients with higher baseline LDL experienced greater cholesterol reductions in the shared incentive arm (0.23 mg/dL per unit change in baseline LDL, 95% CI [−0.46, −0.00]) but were also less likely to have medication potency increases in the physician incentive arm (odds ratio = 0.98 [0.97, 0.996]). Uninsured patients and those of race other than Black or White were less likely to have potency increases in the shared incentive arm (OR = 0.15 [0.03, 0.70] and 0.09 [0.01, 0.93]), respectively. These findings suggest some differential response to incentives, particularly in the form of targeted medication changes.

Keywords

cardiovascular disease; health economics; patient engagement; physician behavior; randomized trials

INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of mortality in the United States (Heron & Anderson, 2016). While there are many risk factors for CVD, high levels of low-density lipoprotein (LDL) cholesterol significantly increase the risk of cardiovascular events and complications (National Cholesterol Education Program Expert Panel on Detection, 2002). In randomized trials, statin therapy has been shown to effectively, safely, and cost-efficiently lower LDL cholesterol, and is recommended by national guidelines for patients at risk for

cardiovascular disease (Collins et al., 2016; Pandya, Sy, Cho, Weinstein, & Gaziano, 2015; Stone et al., 2014). Despite these benefits, patient adherence to statin therapy is a challenge, with half of patients discontinuing the use of statins within the first year (Jackevicius, Mamdani, & Tu, 2002; Maningat, Gordon, & Breslow, 2013). Evidence indicates that patient and provider incentives may improve medication adherence (Asch et al., 2015; DeFulio & Silverman, 2012; Petry, Rash, Byrne, Ashraf, & White, 2012). However, the effectiveness of these approaches may vary for patient subgroups. In this paper, we examine factors that influence variability in response to incentives.

Financial incentives are increasingly common as a quality improvement strategy in healthcare. Pay for performance (P4P) programs targeting hospitals and physician medical groups have been variably effective at changing behavior and improving quality (Eijkenaar, Emmert, Scheppach, & Schoffski, 2013; Rosenthal, Frank, Li, & Epstein, 2005). P4P has shown promise for improving process measures of quality in certain diseases, but the evidence for improving clinical outcomes and reducing costs has been mixed and relatively weak (Damberg et al., 2014; Van Herck et al., 2010). While P4P programs traditionally target providers, financial incentives for patients have been effective in changing health in contexts such as smoking cessation (Halpern et al., 2015; Volpp et al., 2009). Data from randomized controlled trials of financial incentives remain scarce, and few trials have focused on outcome measures of quality such as cholesterol control, or incentive programs that include patient rewards.

Financial incentives might also affect patients differently either through their own behavior and risk factors or through their physician's behavior. Patients in racial or ethnic minority groups, or those with lower socioeconomic status, often face greater challenges to adherence and risk reduction and have poorer outcomes than majority or more resourced groups. If these groups differentially respond to financial incentives, incentive programs might either remediate or exacerbate disparities. Observational studies of P4P have suggested that these incentives do not exacerbate disparities by ethnicity or socioeconomic status, but to our knowledge no randomized intervention studies have reported equity of care results (Van Herck et al., 2010). If other observable patient characteristics, like age and sex, are associated with responsiveness to financial incentives, one might be able to target interventions to increase their population effectiveness. To date, no studies have provided consistent evidence of any behavioral phenotypes that offer such promise for financial incentives (Haff et al., 2015).

We recently reported the results of a multi-center, cluster-randomized trial demonstrating that a shared financial incentive for primary care physicians and their patients improves cholesterol control among patients with established cardiovascular disease or high cardiovascular risk (Asch et al., 2015). Based on the trial we concluded that financial incentives are a moderately effective tool for reducing cholesterol, but our mixed results may mask substantial heterogeneity in response. Here, we examine the differential impact of these financial incentives among patients as a function of their age, race, income, and education level, as well as their clinical characteristics at the start of the study, including baseline cholesterol control, Framingham Risk Score, diagnosis of cardiovascular disease, and statin medication use.

NEW CONTRIBUTION

While financial incentives to providers or patients are often used to target improved health outcomes, few randomized controlled trials have rigorously evaluated their effectiveness. Furthermore, the impact of these financial incentives on patient subgroups and healthcare disparities is largely unknown. Previous studies have examined the heterogeneous effects of financial incentive programs like P4P, as summarized in a recent review (Markovitz & Ryan, 2016). However, such analyses are generally focused on how aggregate characteristics of a patient population influence a hospital or physician group's performance in financial incentive programs, not on how individual patient characteristics affect health outcomes at the patient level. Given the increasing prevalence of financial incentive programs and the importance of equity in healthcare delivery, understanding how such programs affect individual patients with different characteristics is critical. We analyzed data from a randomized controlled trial of financial incentives to providers, patients, or both to lower low-density lipoprotein cholesterol levels, examining how the financial incentives differentially affected patients with various demographic and clinical characteristics.

CONCEPTUAL FRAMEWORK

As economic actors, both patients and providers take into account costs — including time, effort and financial costs — and benefits when making decisions that might influence cholesterol control. The basic economic framework of the demand for and supply of health behavior and medical care that we rely on to motivate our analysis can be adapted to relax the neoclassical assumption that consumers perfectly optimize over complete and transitive preferences. Indeed, the design of incentives in the trial whose results we rely on incorporated insights from behavioral economics. These decision-making biases may be differentially present in some groups of patients versus others and therefore affect behavioral responses. For example, the sense of “invincibility” often associated with youth might logically be associated with greater present bias, sometimes explained as a higher than optimal or hyperbolic discount rate for future outcomes. More frequent experience with lottery tickets might partially inoculate patients from the insensitivity to differences in small probabilities reflected in prospect theory. To the extent that lottery ticket experience may be disproportionately represented in some populations, those populations might be less influenced by probabilistic rewards, or perhaps more influenced because their purchase of lottery tickets instead reveals more of the very insensitivity that prospect theory predicts. There is no clear evidence of differential effects of various behavioral economic processes across identifiable subgroups of individuals, nor is there any clear theory predicting specific effects. Nevertheless, it is plausible that individuals vary on this or other dimensions.

With or without assumptions about decision errors, the impact of financial incentives offered through the trial will change the balance of costs and benefits to decision makers, and potentially lead either physicians, patients, or both to differentially change their behavior. These effects may or may not be symmetric across trial arms. For example, a patient's clinical risk may be more important in predicting physician behavior change than patient response to an incentive because patients might not fully understand the nature of the risk and therefore the benefit of risk reduction. There is extensive evidence suggesting that social

risk factors including race, income, and education also affect health behavior and outcomes in ways that moderate both physician and patient response to incentives (Balsa & McGuire, 2001; Markovitz & Ryan, 2016; National Academies of Sciences, 2016; Ryan, Blustein, Doran, Michelow, & Casalino, 2012; Smedley, Stith, & Nelson, 2003). Likewise sex and age enter the analysis of response heterogeneity because they are conceptually linked to real or perceived differential benefits from cholesterol control as a function of different risk profiles at baseline (D'Agostino et al., 2008; Fulcher et al., 2015; Rathore, Mehta, Wang, Radford, & Krumholz, 2003).

METHODS

Study Design

As described in more detail elsewhere, we conducted a cluster-randomized, controlled trial to compare three approaches to reducing LDL cholesterol in patients at high risk of cardiovascular disease (Asch et al., 2015). Patients were eligible for the study if they met one of two criteria: 1) A 10-year Framingham Risk Score of $\geq 20\%$ or prior coronary artery disease equivalents, combined with LDL ≥ 120 mg/dL, or 2) FRS of 10–20% with LDL ≥ 140 mg/dL. Primary care physicians were eligible for the study if at least five of their patients met the above inclusion criteria for high cardiovascular risk. Participating physicians were assigned to one of four study arms: control (CNTRL), physician incentives (PHYS), patient incentives (PAT), and shared physician-patient incentives (SHARE). Participating patients were assigned to the same arm as their primary care physician. The intervention took place over 12 months, and the primary outcome was change in LDL for each patient at the end of this 12-month period. Secondary outcomes included achievement of LDL goals, medication potency increase, and adherence rate.

In the three intervention arms, each patient was given a quarterly goal of either reducing his/her LDL level by 10 mg/dL from the previous quarter's target, or maintaining LDL level below 100 mg/dL for high risk participants or 130 mg/dL for medium risk participants. All patients were also given a wireless medication bottle (Vitality GlowCap) for their lipid-lowering medications. Every time the cap was opened, the bottle transmitted a wireless signal to *Way to Health*, a web-based study platform, creating a proxy for medication adherence (Asch & Volpp, 2012). Financial incentives depended on both adherence as measured by the wireless medication bottle, and achievement of the quarterly LDL goal.

Incentive Design

Patients and physicians in the control arm did not receive any financial incentives. In the physician incentive arm, PCPs received \$256 for each patient that met his/her quarterly goal, for a maximum payment of \$1024 over 12 months. This payment was distributed semi-annually, separate from larger streams of money, to maximize the effect of the incentive. In the patient incentive arm, patients who adhered to their medication were eligible for a daily lottery, in which they had approximately a 1 in 5 chance of winning \$10, and a 1 in 100 chance of winning \$100. Earnings from these daily lotteries accrued on patients' accounts on *Way to Health*, but were paid out at the end of the quarter only if patients met their quarterly LDL goal. A fully adherent patient who also met each quarter's goal would expect

to earn \$1022 over the year, equivalent to the payment in the physician incentive arm for each patient meeting the goal. In the shared incentive arm, physicians and patients each received the same incentive design as in their individual intervention arms, but the amounts were reduced by half. Therefore, the total potential monetary gain across both patients and physicians was equivalent for all incentive arms.

Analysis

We estimated the effects of race, sex, income, education, age, baseline LDL, Framingham Risk Score (FRS), baseline presence of coronary artery disease (CAD) equivalents, and baseline use of statin medication on 12-month change in LDL within each incentive arm of the trial. We used indicator variables for incentive arms (PAT, PHYS, and SHARE, with CNTRL as reference), high frequency visits at baseline, and payer (Medicaid, Medicare, or uninsured, with private insurer as reference). To test for heterogeneous effects of the trial's incentives on patient subgroups, we fitted a model with interactions between each patient characteristic and the incentive arm variable. We repeated these analyses for the trial's secondary outcomes of 12-month LDL goal achievement (set at the higher of 40mg/dL below the participant's baseline LDL-C value, 100mg/dL for high-risk participants or 130mg/dL for medium-risk participants), medication potency increase (measured as the initiation of a statin for a participant previously not taking statins or an increase in dose or drug potency for a participant currently taking a statin), and annual medication adherence rate (as measured by percentage of days of having opened electronic pill bottles over the one year study period). For context, we also include main effects models without interactions in the appendix; these models show which patients improved in the trial overall, holding incentive arm constant.

Approximately 9% of patients were missing LDL values at 12 months. We conducted multiple imputation using five imputations achieving 97–99% relative efficiency and ensuring in-range values. Analyses were conducted on each imputed data set; results were combined using Rubin's standard rules (Rubin, 1987). For the primary outcome of LDL change, we use mixed-effects models on change in LDL from baseline to 12 months with random effects to adjust for clustering of patients with the same primary care physician (Diggle & Diggle, 2002). Differences in likelihood of achieving LDL goal, likelihood of medication intensification, and annual adherence rates by group were estimated in GEE models with adjustment for patient clustering by primary care physician. The logit link was applied for models of LDL goal achievement and medication intensification, and no imputation was applied for outcomes of adherence rate or medication intensification.

We present our findings in two ways. First, we present the coefficients and confidence intervals from the fully interacted regression models described above. The coefficients on interaction terms allow us to examine the importance of a particular dimension of patient characteristics as a moderator of trial effects. We consider estimates with a p-value less than 0.05 to be statistically significant. Second, we use the fully interacted model of the trial's primary outcome to generate predicted population means of LDL change for each patient characteristic for each intervention arm, and report these marginal predictions and confidence intervals in forest plots. These predictions allow visual comparison of how the

incentives of each trial arm affected patients of differing characteristics, while adjusting for all other covariates. For these plots, we dichotomized all continuous variables with the following cutpoints: income at \$50,000, age at 65, baseline LDL at 160 mg/dL, and Framingham Risk score at 20%.

RESULTS

Table 1 compares patient characteristics across each arm of the trial. As expected, randomization resulted in largely similar patient characteristics across the intervention and control groups. The difference in distribution of characteristics across arms was significant only for race ($p=.02$) and coronary artery disease diagnosis at baseline ($p=.04$).

Table 2 shows output from our models including interactions between all patient characteristics and trial arms to test for heterogeneous effects of the financial incentives on patient subgroups. We fit models for the trial's primary outcome (12-month LDL change), as well as secondary outcomes of 12-month LDL goal achievement, medication potency increase, and adherence rate. For the model of LDL change, more negative numbers indicate greater reduction (better response). Since goal achievement and potency increase are binary outcomes, we present odds ratios for those models. In the LDL change model, we find that patients with higher LDL measurements at baseline experienced greater reductions in cholesterol in the shared incentive arm (estimate = -0.23 mg/dL per unit change in baseline LDL, 95% CI $[-0.46, -0.00]$). For secondary outcomes, we find that patients with higher baseline LDL were less likely to see medication potency increases in the physician incentive arm (odds ratio = 0.98 , 95% CI $[0.97, 0.996]$). Uninsured patients and those of race other than Black or White were less likely to see potency increases in the shared incentive arm (OR = 0.15 $[0.031, 0.70]$ and 0.090 $[0.009, 0.93]$, respectively).

Figure 1 presents marginal predictions of 12-month LDL change by patient characteristic and study arm, with predictions derived from the model with interactions between all patient characteristics and trial arms. Across all arms, we saw substantial improvements in LDL for nearly all subgroups. For the most part, the degree of LDL reduction tended to be similar across subgroups. A notable exception, however, is that patients with higher LDL (≥ 160 mg/dL) at baseline experienced greater reductions in LDL in the shared incentive arm over the course of the trial, compared to those with lower baseline LDL measurements.

DISCUSSION

We examined data from a randomized clinical trial of physician, patient, and shared financial incentives aimed at improving cholesterol control to identify patient characteristics associated with differential response to the incentives in the trial. We assessed differential responses for the trial's primary outcome of 12-month LDL cholesterol change, as well as secondary outcomes of 12-month LDL goal achievement, medication potency increase, and medication adherence rate. We found that patients with higher baseline LDL experienced significantly greater reductions in the shared incentive arm. For secondary outcomes, we found that patients with higher baseline LDL were less likely to experience medication potency increases in the physician incentive arm, and uninsured patients and those of other

race were less likely to experience potency increases in the shared incentive arm. Viewing the results as a whole, we find clues most suggestive of differential behavioral response on the part of physicians on behalf of specific patient subgroups. In particular, we attribute differential potency increases to physician behavior as such decisions are typically governed by physicians or other prescribers with the clinical knowledge to judge optimal medication dosage.

Our findings are subject to several key limitations. The trial was not designed to detect small subgroup differences, so a lack of statistical power remains a plausible interpretation of our null findings. Post hoc power calculations indicate that detectable differences for interaction effects were 20 mg/dL or more, depending on the proportion in the sample. Nonetheless, our analysis represents a refinement of the overall population results and helps rule out larger subgroup effects, which could theoretically be masked by smaller aggregate findings. Separate from this question of power, it is also important to bear in mind that the trial population is not a representative sample of patients in community practice both by design and due to the fact that only 6% of targeted patients enrolled in the trial. If pay for performance were introduced in the general population, we might well find strong associations between key clinical factors, such as baseline LDL, and demographic variables, such as race and education, that would yield a stronger gradient of impact along demographic lines.

Our comparison of adjusted effects for different patient subgroups within trial arm yields some directions for future research. In particular, future research should include studies designed to examine race and income as determinants of differential effects of incentives and tease apart the drivers of these differences if they do in fact exist. Currently, most intervention studies are powered to detect main effects and are not designed to assess patient heterogeneity, although some studies have examined heterogeneity in post-hoc analysis (Markovitz & Ryan, 2016). Such studies have mostly found that hospitals and physician groups with higher numbers of low-income patients face additional challenges in improving quality in response to financial incentives. The effect of patient race/ethnicity is not certain, although some studies suggest that hospital level incentive programs do not harm minority health or access (Epstein, Jha, & Orav, 2014; Ryan, 2010). Many studies, however, analyze patient characteristics and effects at the health system level, relating aggregate patient population characteristics to hospital or physician group response to incentives. Furthermore, many of the incentive programs evaluated also take place at the health system level, and do not directly incentivize individual providers or patients.

If consistent patterns of differential effect were observed based on factors such as race, we believe the response should not be to leap headlong toward different incentive strategies for different subpopulations. That kind of differential policy based on identifiable subgroups is fraught with social tension and must be justified with care. However, if incentive approaches were found to be highly effective in some populations based on clinical criteria, such as high LDL at baseline, it might make sense to consider targeting the programs accordingly. In any case, understanding heterogeneous effects prepares policy makers for the practical consequences of broad policies. As programs move toward rewarding individual physicians

and their patients, it will be important to reassess the how such programs affect patients based on their individual characteristics, and how the impact on disparities may change.

Our study was strengthened by the fact that it was built on a well-designed and successful randomized clinical trial of financial incentives for cholesterol reduction. Our study is one of the first to evaluate a randomized trial of incentives to individual providers and patients, and to assess heterogeneous effects on outcomes by patient characteristics at the individual level. As noted above, an important limitation of our study was the lack of power to detect small- to moderate-sized subgroup effects. Confidence intervals for our regression estimates are wide, which may evidence limited power or underlying variability in the effects of the treatment in the population. In addition we perform multiple statistical tests in our analysis, raising the possibility of Type I error inflation. For dichotomous outcomes, we would ideally report marginal effects of interaction terms instead of odds ratios; however, this was not feasible due to additional complexity of estimation as a result of clustering and multiple imputation. As with any trial, ours was limited in both the scope of patients and practices enrolled and the duration of follow-up.

The science of incentive design for health care improvement continues to advance through trials and natural experiments that leverage insights from psychology and economics. As these programs are deployed, we need to better understand how they affect different populations, both in terms of understanding the implications and possibly for better targeting based on clinical criteria. Understanding these gradients of responsiveness to incentives is critical to maximizing the effectiveness of such programs and the likelihood of achieving intended consequences, while minimizing unintended exacerbation of existing inequities.

Appendix: Main effect models of trial outcomes on patient characteristics and trial arms

	LDL ^I change at 12 months (mg/dL)	Achievement of 12-month LDL goal	Potency Increase	Annual Adherence Rate
	Estimate (95% CI)	Odds Ratio (95% CI)	Odds Ratio (95% CI)	Estimate (95% CI)
Age	0.08 (−0.18, 0.34)	0.99 (0.97, 1.00)	0.98 (0.96, 0.99)**	−0.001 (−0.003, 0.001)
Female gender	6.0 (1.5, 11)**	0.74 (0.56, 0.97)*	1.01 (0.77, 1.31)	−0.024 (−0.062, 0.014)
Black race	−1.1 (−7.0, 4.7)	0.95 (0.65, 1.39)	1.06 (0.75, 1.51)	−0.048 (−0.098, 0.003)
Other race	−1.2 (−12, 9.2)	0.72 (0.35, 1.46)	0.72 (0.37, 1.42)	−0.072 (−0.15, 0.008)
Income <50,000	1.4 (−3.2, 6.1)	1.02 (0.78, 1.33)	1.12 (0.87, 1.43)	0.010 (−0.025, 0.045)
Education less than college	−5.7 (−10, −1.4)*	1.41 (1.06, 1.88)*	1.03 (0.77, 1.36)	0.051 (0.016, 0.086)**
LDL at baseline	−0.61 (−0.68, −0.53)**	1.02 (1.01, 1.03)**	1.01 (1.001, 1.01)*	0.002 (0.001, 0.002)**
FRS at baseline	20 (−4.7, 45)	0.36 (0.075, 1.71)	2.16 (0.47, 9.9)	0.16 (−0.057, 0.37)
CAD diagnosis	−1.5 (−5.9, 2.9)	0.85 (0.64, 1.13)	0.94 (0.70, 1.27)	−0.028 (−0.066, 0.009)
No baseline medication	2.8 (−1.6, 7.1)	0.68 (0.52, 0.89)**	0.78 (0.59, 1.02)	−0.29 (−0.33, −0.25)**
Patient incentive arm	−2.5 (−8.2, 3.1)	1.37 (0.93, 2.00)	0.99 (0.65, 1.51)	0.092 (0.044, 0.14)**
Physician incentive arm	−4.8 (−10, 0.65)	1.27 (0.89, 1.82)	1.33 (0.92, 1.93)	0.055 (0.002, 0.11)*

	LDL ¹ change at 12 months (mg/dL)	Achievement of 12-month LDL goal	Potency Increase	Annual Adherence Rate
	Estimate (95% CI)	Odds Ratio (95% CI)	Odds Ratio (95% CI)	Estimate (95% CI)
Shared incentive arm	−9.8 (−15, −4.1) **	1.81 (1.29, 2.55) **	1.68 (1.14, 2.46) **	0.12 (0.072, 0.18) **
Medicaid	12 (−0.77, 25)	0.37 (0.15, 0.93) *	1.21 (0.61, 2.40)	0.001 (−0.10, 0.10)
Medicare	−0.60 (−5.6, 4.4)	1.00 (0.74, 1.34)	0.97 (0.72, 1.32)	−0.015 (−0.055, 0.025)
Uninsured	12 (3.3, 20) **	0.47 (0.26, 0.84) *	1.38 (0.83, 2.29)	−0.054 (−0.12, 0.011)
Visits per year	−0.15 (−0.64, 0.34)	1.00 (0.97, 1.04)	1.04 (1.01, 1.07) **	−0.001 (−0.005, 0.004)

*
p<0.05;

**
p<0.01

SI conversion factor: To convert cholesterol to mmol/L, multiply values by 0.0259

Abbreviations:

¹ low-density lipoprotein

² Framingham Risk Score

³ coronary artery disease

Notes: Models of trial outcomes on patient characteristics and incentive arms, with interactions between all patient characteristics and incentive arms. Estimates and 95% confidence intervals are reported for the models of LDL change and adherence rate, while odds ratios and 95% confidence intervals are reported for the models of LDL goal achievement and medication potency increase. The goal achievement model does not include interactions between payor and incentive arms, because the model would not converge when including these terms.

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Figure 1: Predictions of LDL change for patient subgroups, by trial arm

Legend: Marginal predictions of LDL reduction (measured in mg/dL) for different patient subgroups, by trial arm, derived from the model with interactions between all patient characteristics and trial arms. Point estimates are presented along with 95% confidence intervals.

Abbreviations: CAD, coronary artery disease; FRS, Framingham Risk Score; LDL, low-density lipoprotein

SI conversion factor: To convert cholesterol to mmol/L, multiply values by 0.0259

Table 1:

Characteristics of enrolled patients, by trial arm

Patient Characteristics	Control (N=366)	Patient incentive (N=358)	Physician incentive (N=433)	Combined incentive (N=346)	p value
<i>Demographic Characteristics</i>					
Age	62	62	62	62	0.72
Female gender	44%	46%	42%	39%	0.26
<i>Race</i>					
White	81%	75%	84%	84%	0.02
Black	17%	20%	13%	12%	
Other	2%	5%	3%	4%	
<i>Income</i>					
Less than \$50,000	46%	41%	46%	41%	0.36
Greater than \$50,000	54%	59%	54%	59%	
<i>Education</i>					
Less than college	64%	59%	62%	61%	0.50
College graduate and above	36%	41%	38%	39%	
<i>Clinical Characteristics</i>					
Framingham Risk Score	0.20	0.20	0.20	0.19	0.36
Pre-existing CAD ¹	40%	31%	33%	33%	0.04
LDL ² at baseline	162	160	160	160	0.77
More than 4 office visits per year	40%	43%	45%	39%	0.28
Taking cholesterol-reducing medications at baseline	32%	32%	34%	34%	0.91
<i>Payor</i>					
Medicaid	3%	3%	3%	3%	0.47
Medicare	24%	27%	29%	26%	
Private	68%	61%	63%	65%	
Uninsured	5%	9%	6%	6%	

Abbreviations:

¹ coronary artery disease² low-density lipoprotein

Table 2:

Fully interacted models of trial outcomes on patient characteristics and incentive arms

	LDL ¹ change at 12 months (mg/dL)	Achievement of 12-month LDL goal	Potency Increase	Annual Adherence Rate
	Estimate (95% CI)	Odds Ratio (95% CI)	Odds Ratio (95% CI)	Estimate (95% CI)
Age	0.07 (−0.47, 0.61)	1.01 (0.98, 1.04)	0.99 (0.96, 1.03)	−0.000 (−0.004, 0.003)
Female gender	6.0 (1.4, 11) [*]	1.37 (1.04, 1.81) [*]	0.98 (0.75, 1.30)	−0.028 (−0.066, 0.010)
Black race	−2.20 (−15, 11.0)	0.97 (0.34, 2.77)	0.88 (0.45, 1.73)	−0.057 (−0.141, 0.027)
Other race	14.5 (−13, 42.2)	2.98 (0.22, 40)	1.54 (0.43, 5.5)	−0.14 (−0.29, 0.018)
Income <50,000	5.25 (−4.0, 14.5)	1.42 (0.73, 2.76)	1.19 (0.69, 2.05)	−0.002 (−0.069, 0.064)
Education less than college	−8.11 (−18, 1.64)	0.61 (0.33, 1.14)	0.97 (0.42, 2.21)	0.021 (−0.043, 0.085)
LDL ¹ at baseline	−0.56 (−0.71, −0.40) ^{**}	0.98 (0.97, 0.99) ^{**}	1.02 (1.01, 1.03) ^{**}	0.002 (0.001, 0.003) ^{**}
FRS ² at baseline	47 (−7.8, 101)	3.60 (0.14, 95)	3.39 (0.12, 97)	0.089 (−0.28, 0.46)
CAD ³ diagnosis	2.1 (−7.0, 11)	1.29 (0.69, 2.41)	1.14 (0.77, 1.70)	−0.010 (−0.063, 0.043)
No baseline medication	0.31 (−8.5, 9.2)	1.51 (0.87, 2.62)	1.02 (0.62, 1.68)	−0.27 (−0.33, −0.21) ^{**}
Patient incentive arm	3.1 (−62, 68)	0.82 (0.027, 25)	93 (1.64, 5318) [*]	0.14 (−0.33, 0.61)
Physician incentive arm	−6.6 (−68, 55)	0.97 (0.049, 19)	69 (2.03, 2310) [*]	0.12 (−0.35, 0.59)
Shared incentive arm	50 (−15, 116)	2.96 (0.068, 129)	449 (9.5, 21000) ^{**}	0.057 (−0.41, 0.52)
Medicaid	−0.20 (−27, 27)	2.82 (1.21, 6.6) [*]	2.42 (0.55, 11)	−0.088 (−0.24, 0.062)
Medicare	−4.9 (−16, 5.7)	0.99 (0.73, 1.36)	1.04 (0.57, 1.9)	−0.007 (−0.086, 0.071)
Uninsured	14 (−4.0, 32)	2.14 (1.18, 3.90) [*]	3.68 (1.13, 12) [*]	−0.040 (−0.17, 0.091)
Visits per year	0.22 (−0.80, 1.2)	1.03 (0.95, 1.11)	1.08 (1.01, 1.16) [*]	−0.002 (−0.010, 0.006)
Black race X PAT ⁴	0.05 (−17, 17)	0.75 (0.24, 2.36)	1.75 (0.64, 4.8)	0.031 (−0.089, 0.15)
Black race X PHYS ⁵	−2.4 (−21, 17)	1.10 (0.33, 3.74)	0.78 (0.27, 2.24)	−0.017 (−0.16, 0.13)
Black race X SHARE ⁶	7.7 (−11, 26)	1.74 (0.51, 5.9)	1.45 (0.55, 3.86)	0.015 (−0.11, 0.14)
Other race X PAT	−27 (−60, 6.0)	0.26 (0.016, 4.3)	0.76 (0.15, 3.91)	0.10 (−0.12, 0.32)
Other race X PHYS	−2.4 (−36, 32)	0.89 (0.040, 19)	0.27 (0.041, 1.75)	0.091 (−0.14, 0.32)
Other race X SHARE	−25 (−60, 11)	0.41 (0.023, 7.4)	0.090 (0.009, 0.93) [*]	0.051 (−0.20, 0.30)
Income <50,000 X PAT	−6.0 (−19, 6.6)	0.69 (0.30, 1.59)	0.75 (0.36, 1.57)	−0.008 (−0.095, 0.080)
Income <50,000 X PHYS	−3.9 (−16, 8.3)	0.66 (0.31, 1.44)	0.81 (0.41, 1.61)	0.026 (−0.065, 0.12)
Income <50,000 X SHARE	−3.8 (−17, 9.0)	0.55 (0.23, 1.33)	1.44 (0.66, 3.11)	0.015 (−0.093, 0.12)
Education <college X PAT	1.6 (−11, 14)	0.96 (0.40, 2.28)	1.07 (0.40, 2.88)	0.037 (−0.056, 0.13)
Education <college X PHYS	5.0 (−8.0, 18)	1.41 (0.62, 3.18)	1.25 (0.49, 3.15)	0.007 (−0.089, 0.10)
Edu <college X SHARE	−0.74 (−14, 13)	1.18 (0.49, 2.86)	0.86 (0.32, 2.34)	0.077 (−0.021, 0.18)
Age X PAT	−0.01 (−0.79, 0.76)	1.01 (0.97, 1.06)	0.96 (0.92, 1.01)	−0.003 (−0.009, 0.003)
Age X PHYS	0.11 (−0.63, 0.86)	1.002 (0.97, 1.04)	0.99 (0.96, 1.03)	0.001 (−0.004, 0.005)
Age X SHARE	−0.21 (−0.97, 0.55)	1.004 (0.97, 1.04)	0.96 (0.91, 1.00)	0.000 (−0.005, 0.005)
LDL at baseline X PAT	0.02 (−0.21, 0.24)	0.999 (0.98, 1.01)	0.99 (0.98, 1.01)	0.000 (−0.001, 0.002)
LDL at baseline X PHYS	0.02 (−0.19, 0.23)	0.998 (0.99, 1.01)	0.98 (0.97, 0.996) ^{**}	−0.001 (−0.002, 0.001)

	LDL ¹ change at 12 months (mg/dL)	Achievement of 12-month LDL goal	Potency Increase	Annual Adherence Rate
	Estimate (95% CI)	Odds Ratio (95% CI)	Odds Ratio (95% CI)	Estimate (95% CI)
LDL at baseline X SHARE	-0.23 (-0.46, -0.00) *	0.99 (0.98, 1.01)	0.99 (0.98, 1.00)	0.000 (-0.002, 0.002)
FRS at baseline X PAT	-44 (-115, 28)	0.18 (0.002, 17)	0.36 (0.004, 30)	0.30 (-0.35, 0.95)
FRS at baseline X PHYS	-26 (-97, 44)	3.27 (0.055, 196)	0.51 (0.007, 36)	-0.033 (-0.52, 0.45)
FRS at baseline X SHARE	-38 (-113, 38)	0.46 (0.006, 36)	0.72 (0.009, 59)	-0.007 (-0.56, 0.55)
CAD diagnosis X PAT	-1.1 (-14, 11)	0.95 (0.47, 1.95)	0.81 (0.37, 1.76)	-0.004 (-0.10, 0.095)
CAD diagnosis X PHYS	-5.5 (-18, 7.2)	1.05 (0.45, 2.46)	0.69 (0.35, 1.35)	-0.004 (-0.10, 0.096)
CAD diagnosis X SHARE	-7.1 (-20, 6.1)	0.68 (0.29, 1.58)	1.06 (0.52, 2.16)	-0.061 (-0.15, 0.027)
No baseline medication X PAT	7.7 (-4.9, 20)	1.15 (0.54, 2.43)	0.46 (0.20, 1.07)	-0.071 (-0.17, 0.032)
No baseline medication X PHYS	-2.0 (-14, 10)	0.91 (0.43, 1.95)	0.95 (0.49, 1.83)	0.008 (-0.076, 0.091)
No baseline medication X SHARE	3.9 (-8.6, 16)	0.92 (0.43, 1.98)	0.64 (0.29, 1.38)	-0.024 (-0.14, 0.088)
Visits per year X PAT	-0.34 (-1.7, 0.99)	0.97 (0.88, 1.07)	0.97 (0.89, 1.05)	0.004 (-0.009, 0.018)
Visits per year X PHYS	-0.81 (-2.1, 0.47)	0.93 (0.84, 1.03)	0.96 (0.88, 1.04)	0.003 (-0.007, 0.013)
Visits per year X SHARE	-0.33 (-1.9, 1.3)	0.97 (0.86, 1.09)	0.91 (0.83, 1.00)	-0.002 (-0.016, 0.012)
Medicaid X PAT	11 (-25, 48)		0.66 (0.097, 4.4)	0.13 (-0.067, 0.33)
Medicaid X PHYS	32 (-4.6, 68)		0.51 (0.052, 5.0)	0.21 (-0.086, 0.50)
Medicaid X SHARE	3.3 (-35, 42)		0.20 (0.032, 1.24)	0.026 (-0.20, 0.25)
Medicare X PAT	4.2 (-11, 19)		0.94 (0.39, 2.26)	0.040 (-0.068, 0.15)
Medicare X PHYS	6.7 (-7.4, 21)		0.82 (0.37, 1.78)	-0.019 (-0.12, 0.086)
Medicare X SHARE	6.5 (-8.8, 22)		1.04 (0.40, 2.72)	-0.048 (-0.17, 0.075)
Uninsured X PAT	-4.7 (-28, 18)		0.49 (0.12, 1.98)	0.047 (-0.14, 0.23)
Uninsured X PHYS	4.7 (-22, 31)		0.33 (0.065, 1.64)	-0.093 (-0.26, 0.073)
Uninsured X SHARE	-5.8 (-30, 19)		0.15 (0.031, 0.70) *	-0.024 (-0.20, 0.15)

* $p < 0.05$;

** $p < 0.01$

SI conversion factor: To convert cholesterol to mmol/L, multiply values by 0.0259

Abbreviations:

¹ low-density lipoprotein

² Framingham Risk Score

³ coronary artery disease

⁴ patient incentive arm

⁵ physician incentive arm

⁶ shared incentive arm