Kaiser Permanente Date: 05/19/2018

Topic: Using Regression Discontinuity Designs to Measure the Impact of a KP HDHP Product Offering

Introduction

Kaiser Permanente (KP) rarely offers more than one product offering within a group account. However, due to market dynamics, KP has been feeling pressure to rethink about its position. Part of KP's reason for the scarcity of product offerings is driven by the fact that the primary product KP offers, hmo, is their bread and butter – it's a rich product (no deductibles are attached and generally low copayment), KP knows the product really well (e.g. who purchases it, how to price it, etc.), and compared to the dhmo or hdhp (other KP products offered which are deductible products), hmo is charged at a higher rate.

800 -800 -800 -400 -

hdhp

KP Product

Chart 1: Average Rate Breakdown by KP Product

Market dynamics however are impacting KP's competitive product positioning within their large commercial book of business group accounts (e.g. Google, Bank of America, Walmart). Competitors (e.g. Anthem, Cigna, United) are offering leaner and cheaper products (hdhp), whereas KP is mainly offering a more rich and expensive product, hmo.

This problem creates a couple issues:

dhmo

a. Adverse selection occurs for the healthy KP members within group accounts.

hmo

- Healthy KP members terminate and enroll in a competitor learner available product.
 This creates a reduction in market share within the group and leaves KP with a costly demographic of members who are high utilizers of the health care service.
- b. A reduction in annual enrollment growth rates.
 - KP has a hard time attracting new, low utilizer members.

The challenges stated above have been deteriorating KP's profits. Therefore, leadership suggested to develop a product strategy to match competitors' leaner product by offering KP's learner product, hdhp, alongside a second KP product, dhmo or hmo.

This analysis will explore the impact KP's hdhp product offering has on KP's membership growth. The data is observational data and therefore the typical tools for evaluating the causal effect will have to circumvent to other possible methods. Since, KP rarely prefers to offer an hdhp product offering and is driven by other market factors (e.g. Affordable Health Care Act, employer not member demands it), I have considered to use a Regression Discontinuity Design to take advantage of this setup.

Background

There are challenges and assumptions that must be considered when attempting to generate causal inference from an analysis. Particularly around the issues of bias and precision. Since causal inference is the focus of this analysis, I feel it is important to review some of the key issues and assumptions. From the outset, making causal inference is not physically possible. For the reason that we must observe and compare the outcome of each observation under two yet concurrent experimental conditions (1) Treatment, (e.g. KP offers a group an hdhp product), and (2) the Counterfactual or Control (e.g. KP doesn't offer an hdhp product). Fortunately, Rubin's Causal model provides an alternative.

Rubin's framework describes the effect of a well-defined treatment—the offering of a KP hdhp product within a group—on KP's membership growth. We use $Y_i(1)$ to represent what the value of the i^{th} group's outcome Y would be if the group were assigned to the treatment ("1") condition, and $Y_i(0)$ to represent what the value of the outcome would be for the same group if assigned to the treatment ("0") condition. The corresponding treatment tre

$$[0.0] ATE = E[Y_i(1) - Y_i(0)]$$

Rubin has shown that it is possible to estimate the ATE from experimental data provided that groups have been randomly assigned to the treatment conditions and that a critical assumption, *stable-unit-treatment-value-assumption* (*SUTVA*) holds. Randomly assigning groups to a treatment and control group leads to an unbiased estimate of the ATE. The reason why is that when the assignment of the treatment and control group have been random, all factors other than the treatment are identical.

Let us consider using regression notation on observation data to clearly identify where the bias can exist. Suppose,

$$[0.1] Y = \beta_0 + \beta_1 X + \varepsilon$$

where X is the predictor variable we are interested in estimating (e.g. KP offering an hdhp product) and Y is the outcome variable (e.g. membership growth) we would like to measure X's relationship too. Note, we do not need to do a regression to obtain β_1 , we can calculate it;

$$\beta_1^{OLS} = \frac{\sigma_{yx}}{\sigma_x^2}$$

However, let's examine how the presence of endogeneity in the question predictor X results with a bias in the OLS estimator. If we manipulate equation 0.1 by using covariance algebra (take covariance X throughout the equation), we get the following;

[0.3]
$$cov(Y, X) = cov(\beta_0 + \beta_1 X + \varepsilon, X)$$

$$= \beta_1 cov(X, X) + cov(\varepsilon, X)$$

$$= \beta_1 var(X) + cov(\varepsilon, X)$$

$$\sigma_{yx} = \beta_1 \sigma_X^2 + \sigma_{\varepsilon x}$$

Now dividing throughout equation 0.3 by the population variance of the question predictor X;

$$\frac{\sigma_{yx}^2}{\sigma_x^2} = \beta_1 + \frac{\sigma_{\varepsilon x}}{\sigma_x^2}$$

The $\frac{\sigma_{\varepsilon x}}{\sigma_x^2}$ ratio in equation 0.4 will give us the magnitude and direction of the bias in β_1 . The larger the ratio, the larger the bias.

Note that in equation 0.4, the population covariance of Y with X, σ_{yx} , divided by the population variance of X, σ_X^2 , can only be equal to the impact of β_I , when the second term on the right-hand side of the equation is zero. This in turn can only happen if the population covariance of the predictor and the residual, σ_{ex} , equal to zero.

In other words,

$$\frac{\sigma_{yx}^2}{\sigma_x^2} = \beta_1, \quad \text{when } \sigma_{\varepsilon x} = 0$$

Now that we have a good understanding of where bias enters in our setup, it is also wise to spend time on the notion of precision in our question predictor *X*.

The question predictor X has higher precision based on having a smaller standard error (se) in our β_1 estimate;

$$[0.6] se(\beta_1) = \frac{\sigma_{\varepsilon}^2}{\sum_{i=1}^n (X_i - \bar{X})^2}$$

It is clear than that the precision of β_I depends on the variance of the residuals, σ_{ε}^2 . Datasets that generate extensive scatter in the residuals lead to OLS estimates of slope that are not very precise.

Increased precision thus leads to an increase in the magnitude of the t-statistic associated with the β_1 , and a corresponding improvement in our ability to reject the null hypothesis that β_1 is equal to zero;

$$[0.7] t-statistic = \frac{\beta_1}{se(\beta_1)}$$

Let us now return back to the discussion regarding randomized experiments.

In some cases, it can be very costly to conduct a randomized experiment. Randomly offering KP groups a leaner hdhp product alongside a richer KP product (e.g. hmo) can cause a lot of problems from a financial perspective. If no new member signs up for a KP hdhp plan and all existing KP hmo members cannibalize to the hdhp product, then KP just lost potential revenue of charging KP hmo members a higher rate than the KP hdhp rate.

Regression Discontinuity Design

Participants are sometimes randomized to different product options, innovative practices, or to different incentives by exogenous mechanisms that are not under the direct control of an investigator, but still provide the equality in expectation prior to the treatment that supports causal inference.

Provided that we can argue persuasively that those participants who are then subject to the contrasting and naturally occurring experimental conditions are indeed equal in expectation prior to the treatment, we have a logical basis for making unbiased inferences about the causal impact of the treatment. Thus, data from "natural experiments" can sometimes be analyzed in the same way as data from investigator-designed experiments.

For this analysis, conducing a randomized experiment was not feasible. An argument can be made however that KPs offering of an hdhp product was driven naturally and outside the influence of the investigator. After the Affordable Health Care Act, several employers were required to offer high deductible health care products (e.g. hdhp). With these new policy changes, Kaiser was either forced to offer a hdhp products to employers or they were at risk of losing the entire business. Since hdhp product offers were mainly driven by forces based on the groups abrupt change in plan offerings, I will be using it as a "natural experiment". The method I will be using to derive the causal impact of offering an hdhp product is a Regression Discontinuity Design.

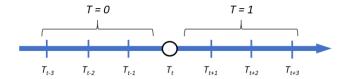
All Regression Discontinuity Designs incorporate:

- a. An underlying continuum along which participants are arrayed.
 - We refer to this continuum as the "assignment" or "forcing variable".
 - A passage of time can provide us with a credible "forcing variable".
- b. An exogenous determined cut-point on the forcing variable that divides participants explicitly into groups that experience different treatment or conditions.
 - In interpreting the implications of our findings causally, we must rely on the assumption that the timing of the "forcing variable" was determined exogenously.
 - Abrupt changes, for example in a product mix, can provide one important source of potential natural experiments.

For our analysis, the exogenous cut off point has been defined as $kp_hdhp_fv_i$, which is a dummy variable defined as 1, if the hdhp product was offered within a group, and 0, if the hdhp product did not exist within a group. The passage of time in our analysis is the enrollment periods after and prior to the abrupt offering of the KP hdhp product.

In making use of using a simple t-test to compare groups (groups offered hdhp vs groups not offered hdhp), we find ourselves having to argue that the analytic samples of groups who fall within the analytic window on either side of the cut-off on the forcing variable, $kp_hdhp_fv_{it}$, are equal in expectations prior to treatment. I believe this argument can hold since (1) the window bandwidth is small (less than +- 3 enrollment periods), (2) we are measuring the outcomes on the same groups (e.g. 2014 to 2016 KP did not offer hdhp to Cisco and MG was ~5%, 2017 to 2018, KP offered Cisco hdhp and MG was ~10%). Therefore, it can be argued there are very little difference between the control and treatment groups, since they are the same participants (e.g. the location is the same, the B2B relationship is the same, etc.), and (3) we have included covariates to control for argued differences between the two groups.

Figure 1: The Analytic Window



Where, T_t , is defined as the period of the offering of the KP hdhp product, t+n are the periods after the offering, and t-n are the periods prior to the KP hdhp offering.

The table below illustrates how the treatment variable, $kp_hdhp_fv_{it}$, was defined in the dataset. Note that the KP hdhp product offering was not based on a single year (however the majority occurred after ACA, 2015). Instead the offering varied between groups (e.g. CO_1071 first offered an KP hdhp in 2017, whereas CO_14000 offered an hdhp in 2016):

(146 unique groups were used in this analysis)

	region_account_number	effective_date_year	kp_hdhp_fv
1	CO_1071	2014	0
2	CO_1071	2015	0
3	CO_1071	2016	0
4	CO_1071	2017	1
5	CO_1071	2018	1
6	CO_1095	2014	0
7	CO_1095	2015	1
8	CO_1095	2017	1
9	CO_1095	2018	1
10	CO_14000	2014	0
11	CO_14000	2015	0
12	CO_14000	2016	1
13	CO_14000	2017	1

Model Results

As a reminder, the focus of this analysis is to aim for the causal inference of offering an hphp product. Therefore, while we review the models below, leveraging the knowledge we learned in equations 0.4, 0.5 and 0.6, will help us deal with interpreting where the bias might lie and how to increase precision as we preview each iteration of the models.

The first model I developed was a simple regression model;

[1]
$$KP_MG_{it} = \beta_0 + \beta_1 KP_hdhp_fv_{it} + \varepsilon_{it}$$

The results of this model were not promising. First of all, I expected the β_1 coefficient to have a positive sign, but instead it was negative. The β_1 coefficient was -.02. The interpretation would be that the addition of offering a KP hdhp product alongside another KP product (dhmo or hmo) would have a -%2 impact on KP's membership growth. Not only was the sign not what I expected, but it was also lacking precision resulting in an insignificant t-statistic. Which means our variable of interest KP_hdhp_fv is statistically insignificant.

I mentioned that the KP hdhp product is offered alongside a second KP product, dhmo or hmo. Therefore, it is sensible that we control for which product the second product is in our model;

[2]
$$KP_MG_{it} = \beta_0 + \beta_1 KP_hdhp_fv_{it} + \beta_2 KP_dhmo_{it} + \beta_3 KP_hmo_{it} + \varepsilon_{it}$$

In model 2, I included which second KP product was offered alongside KP's hdhp product. The results were better. Just as I had expected, the coefficient sign on β_1 was positive. The interpretation on β_1 was the addition of offering a KP hdhp product alongside another KP product (dhmo or hmo) would have a +2% impact on KP's membership growth. However, I was still lacking precision on β_1 , resulting in an insignificant t-statistic. Therefore, our variable of interest KP_hdhp_fv is still not statistically significant.

In the third model, I find myself having to argue that the analytic samples of groups who fall within the analytic window on either side of the cut-off on the forcing variable, $kp_hdhp_fv_{it}$, are equal in expectations prior to treatment. It has been shown that the new members who enroll in a KP hdhp plan are younger. The hdhp product is a high deductible product. This means that a larger proportion of the risk is bared on the member and not the health plan. Therefore, it could be argued that both sides of the forcing variable, $kp_hdhp_fv_{it}$, are not equal in expectation because the average age of the members have potentially declined.

It is also argued that the addition of offering a KP hdhp product has influenced the rates of KP's second product (dhmo or hmo). Therefore, I also controlled for rate increases/decreases;

[3]
$$KP_MG_{it} = \beta_0 + \beta_1 KP_hdhp_fv_{it} + \beta_2 KP_dhmo_{it} + \beta_3 KP_hmo_{it} + \beta_4 KP \text{ average age }_{it} + \beta_5 KP \text{ rate increase decrease}_{it} + \varepsilon_{it}$$

Model 3, wasn't exactly promising. Lack of precision continues in our variable of interest. However, the addition of both variables significantly impacted model 3's R^2 . Model 3's R^2 is two times larger than model 2's R^2 .

It can be argued that the prior year membership growth of a group can impact the current year membership growth. This can result in a bias in our variable of interest $kp_hdhp_fv_{it}$. I therefore controlled for this in model 4. As I expected, the β_1 coefficient increased from 2% to 4%. The addition of including the prior year membership growth also increased the precision of β_1 . The t-statistic increased from 1.19 to 1.58 (that is over 30%). Note, we need a t-statistic of at least 1.65 to have $kp_hdhp_fv_{it}$ statistically significant at 90% or an α -level of 10%;

[4]
$$KP_MG_{it} = \beta_0 + \beta_1 KP_hdhp_fv_{it} + \beta_2 KP_dhmo_{it} + \beta_3 KP_hmo_{it} + \beta_4 KP_average_age_{it} + \beta_5 KP_t rate_increase_the decrease_the decrease_th$$

In my final model, model 5, I included two variables that boosted the precision of our variable of interest significantly (t-statistic is 2.73 and α -level of 1%). The two variables I introduced are (1) Trend, and (2) count of years hdhp product was offered within a group. There could be the potential of heteroskedastic variances in membership growth related to the years a KP hdhp product has been offered. This violates our assumption that the errors are distributed normally with a variance of σ_{ε}^2 . I also included trend to control for secular trend;

$$\varepsilon \sim N(0, \sigma_{\varepsilon}^2)$$

[5]
$$KP_MG_{it} = \beta_0 + \beta_1 KP_hdhp_fv_{it} + \beta_2 KP_dhmo_{it} + \beta_3 KP_hmo_{it} + \beta_4 KP_average_age_{it} + \beta_5 KP_rate_increase_decrease + \beta_6 KP_prior_MG_{it} + \beta_7 trend_{it} + \beta_8 years_hdhp_offered_{it} + \varepsilon_{it}$$

The table below illustrated all model results

Table 1: Regression Discontinuity Design, Model Results

	Dependent variable: KP Membership Growth					
	(1)	(2)	(3)	(4)	(5)	
KP hdhp Forcing Variable	-0.02 (0.02)	0.02 (0.02)	0.02 (0.02)	0.04 (0.02)	0.08*** (0.03)	
KP Offers dhmo		0.04* (0.02)	0.05** (0.02)	0.08*** (0.02)	0.07*** (0.02)	
KP Offers hmo		0.09*** (0.02)	0.09*** (0.02)	0.09*** (0.02)	0.09*** (0.02)	
Average Age			-0.005*** (0.001)	-0.004*** (0.001)	-0.004*** (0.001)	
Average KP Rate Increase			0.06 (0.06)	0.06 (0.07)	0.08 (0.07)	
Prior Membership Growth				0.03 (0.04)	0.02 (0.04)	
Trend					-0.01 (0.01)	
Years KP hdhp Offered					-0.02** (0.01)	
Constant	-0.01 (0.01)	-0.07*** (0.02)	0.06 (0.04)	0.02 (0.04)	16.11 (15.15)	
Observations R2 Adjusted R2 Residual Std. Error F Statistic				568 0.05 0.04 0.23 (df = 561) 5.32*** (df = 6; 561)		

Other Considerations

In addition to evaluating the impact an offering of a KP hdhp product has on membership, I measured the effect of the change in pricing, relative to KPs competitors price, charged to the member. Specifically, I looked at *prior to* the KP hdhp product offering, what was the state of KP's product price compared to its competitor within a group. Was it favorable (KP's product offering was cheaper than its competitor), parity (both KP and competitor product prices are relatively marked at the same rate) or was it unfavorable (KP's product offering was more expensive than its competitor). Then, I looked at what was the *current* state of KP's product price when hdhp was offered compared to its competitor. This allowed me to evaluate the membership growth impact from the pricing of a parity state to an unfavorable state, or parity state to a favorable state.

To model this, we need to include interaction terms between our variable of interest, $kp_hdhp_fv_{it}$, and the change in KP's pricing state;

[6]
$$KP_MG_{it} = \beta_0 + \beta_1 KP_hdhp_fv_{it} + \beta_2 KP_dhmo_{it} + \beta_3 KP_hmo_{it} + \beta_4 KP_average_age_{it}$$
 $+ \beta_5 KP_rate_increase_decrease + \beta_6 KP_prior_MG_{it} + \beta_7 trend_{it} + \beta_8 years_hdhp_offered_{it}$
 $+ \beta_9 KP_state_parity_positive_{it} + \beta_{10} (KP_state_parity_positive_{it} * KP_hdhp_fv_{it}) + \varepsilon_{it}$

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[7] KP\_MG_{it} = \beta_0 + \beta_1 KP\_hdhp\_fv_{it} + \beta_2 KP\_dhmo_{it} + \beta_3 KP\_hmo_{it} + \beta_4 KP\_average\_age_{it}
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- + β_5 KP_rate_increase_decrease + β_6 KP_prior_MG_{it} + β_7 trend_{it} + β_8 years_hdhp_offered_{it}
- + β_9 KP_state_parity_negative_{it} + β_{10} (KP_state_parity_negative_{it} * KP_hdhp_fv_{it}) + ε_{it}

I expected to see a large positive swing on membership growth when KP's prior state was parity and moved to favorable when the hdhp product was offered within the group. I also expected a negative swing to membership growth when KP's product offering was parity prior to the hdhp product offering but then moved to a negative state after the product offering.

In Table 2, we see exactly as expected. To get the total effect of the interaction terms in model 6, we need to add the coefficients $\beta_1 + \beta_{10}$. The result is a 20% drop in membership growth when KP's change in contribution state went from parity to negative. In model 7, the result is a 16% positive impact on KP's membership growth when the change in contribution state went from parity to positive.

Dependent variable:	KP Membership	Growth
	(6)	(7)
KP hdhp fv	0.09***	
	(0.03)	(0.03)
KP Offers dhmo	0.06**	0.06***
	(0.02)	(0.02)
KP Offers hmo	0.09***	0.09***
	(0.02)	(0.02)
Average Age	-0.004***	-0.004***
	(0.001)	(0.001)
Average KP Rate Increase	0.09	0.09
•	(0.07)	(0.07)
Prior Membership Growth	0.02	0.02
The state of the s	(0.04)	(0.04)
Trend	-0.01	-0.01
	(0.01)	(0.01)
Years KP hdhp Offered	-0.02**	-0.02**
	(0.01)	(0.01)
KP parity to negative	0.17***	
	(0.06)	
KP parity to negative*KP hdhp fv	-0.29***	
	(0.08)	
KP parity to positive		-0.06
And the last the second		(0.06)
KP parity to positive*KP hdhp fv		0.09
		(0.08)
Constant	13.88	17.24
	13.88 (15.08)	(15.23)
Observations R2	568 0.09	568 0.07
Adjusted R2	0.07	0.05
Residual Std. Error (df = 557)	0.07 0.22	0.23
F Statistic (df = 10; 557)	5.26***	3.97***
Note:		0.05; ***p<0.01