Essays on Health Care Provider Behavior

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Abstract

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Using innovative econometric methods that condition on observed and unobserved characteristics, I estimate patient-specific treatment effects (in particular, Person-Centered Treatment (PeT) effects) for schizophrenia patients taking atypical antipsychotic drugs. Using these treatment effect estimates, I determine how well physicians matched patients to the appropriate drug. By looking at trends in matching over time, I look for evidence of physician learning. Additionally, using patient-specific treatment effects, I look at geographic variation in a number of measures to better understand variation in quality of care. Lastly, I build a theoretical model to study how the threat of waiving copayments affects the optimal insurance contract between the insurer and the patient and payment contract between the insurer and the provider. I find that the threat of waiving copayments may result in reduced patient welfare.

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Chapter 1: Understanding Physician Uptake of Technologies:

Using Person-Centered Treatment Effects to Study Physician Uptake of New Therapies Among Medicaid Patients with Schizophrenia

I. Introduction

The decision-making process that physicians undergo when deciding about treatment choice is complex. In addition to uncertainty in treatment effectiveness, response to treatment is heterogeneous for many diseases. Furthermore, physicians often have more experience using some therapies than others. Still, in this environment, physicians must decide which treatment to prescribe for each patient.

To improve decision-making in this complex environment, physicians can engage in a learning process about which treatments to choose for which patients. However, when considering the possibility of learning, physicians face a tension between two competing goals (Crawford and Shum, 2005). If physicians are risk averse concerning patient outcomes and desire to maximize current patient health, they will be reluctant to try newer drugs where there is a higher degree of uncertainty about outcomes. However, if physicians are forward looking, using newer drugs generates information about treatment effectiveness, which can lead to better outcomes for future patients.

Although clinical trials provide some information about the average effectiveness of treatments, they often do not provide the nuanced information about treatment effects that physicians need to optimally prescribe to a heterogeneous population of patients. In this environment, are physicians willing to trade off current patient health in order to learn about the newer drug and reduce uncertainty? Or is learning too costly?

A number of studies have been done trying to understand physician learning. Most of these analyses have used a Bayesian learning-by-doing framework. Under this approach, the physician's (or patient's) utility is a function of a parameter that represents the value of a given treatment. As quality signals about the drug are observed over time, the evolution of the value parameter is studied. One concern with using this framework to study learning is that the analyst-observed quality signal may differ from the actual signal to which the physician is responding. Also, many of these models study how physicians learn as they take patients through a series of treatments (known as "within-patient learning"). While this is important to understand, it would be better if physicians could successfully match patients to the appropriate first-line therapy.

In this paper, I study whether physicians are learning to match patients to the optimal first-line therapy without needing to make any assumptions about quality signals the physician may or may not be receiving. In order to do this, I model counterfactual outcomes and look to see if physicians are making optimal decisions. This is a challenging task in a model of heterogeneous returns with endogeneity concerns. In such a setting, issues of sorting on the level and sorting on the gain arise. Heckman (1997) demonstrated that, in this setting, typical instrumental variables (IV) methods do not estimate treatment effect parameters of interest. Since this discovery, a series of papers (Heckman and Vytlacil (1999); Heckman, Urzua, and Vytlacil (2006); Basu (2014)) have followed that have developed local IV (LIV) methods that can be used to estimate common treatment effect parameters (e.g., the average treatment effect (ATE), the effect of the treatment on the treated (TT), and the effect of the treatment on the untreated (TUT)) in these circumstances. While these parameters can be used to model

counterfactual outcomes on subgroups of the population, they are not personalized for each individual in the population.

Building on this research, a recent paper by Basu (2014) developed a new treatment effect parameter, the Person-centered Treatment (PeT) effect, which uses information about the individual's treatment decision and the circumstances under which they made the decision to estimate a personalized treatment effect for each individual. Using these PeT effects, we can analyze the treatment decision made for each patient to determine if it is optimal. By examining trends over time, we can look for evidence of learning.

In this paper, I use the methods described above to evaluate if physicians are learning. I look at a large data set of Medicaid patients who have been diagnosed with schizophrenia. Schizophrenia is a useful syndrome to study because response to the treatment is known to be heterogeneous (Meltzer, 1986).

I study patients who have been treated with atypical antipsychotic drugs (AADs). Each patient can either take an older drug (the treatment) or a newer drug to treat their symptoms. The outcome of interest is hospitalizations in the year following initiation of the therapy. Using LIV methods, I estimate a PeT effect for each patient. From this estimate, I can determine whether the optimal treatment for the patient was an older drug (PeT effect less than zero) or a newer drug (PeT effect greater than zero). By looking at the match rate (proportion of patients who received their optimal treatment) over time, I can check to see if physicians are learning.

When looking at the match rate, I find some evidence of physicians being able to distinguish which treatment is optimal for a particular patient. In particular, the proportion of patients who should have received an older drug is higher in the group of patients who actually

received an older drug than in the group of patients who actually received a newer drug. However, the match rate is decreasing over the two-and-a-half year time period I consider. This is driven almost entirely by physicians switching to a higher use of newer drugs over time. The match rate among patients receiving newer drugs and the match rate among patients receiving older drugs is roughly constant over time. However, the match rate for patients receiving newer drugs is much lower than the match rate among patients receiving older drugs.

To determine if the physicians are being selective in the types of patients that they are switching to newer drugs over time, I study trends in the ATE, TT, and TUT, which can be easily estimated using the PeT effects. I find evidence that physicians are switching patients with smaller (but still negative) PeT effects to newer drugs and keeping patients with very large PeT effects on older drugs. This suggests that, in the short term, physicians may be able to determine which patients would have large benefits or harms from a given treatment, but they may not be able to determine smaller effects as easily.

Lastly, using the PeT effects, I examine trends in different counterfactual outcomes: all patients receiving newer drugs, all patients receiving older drugs, and all patients receiving the optimal predicted treatment. These strategies can be compared to the actual outcomes to determine the benefits from personalization compared to alternative treatment strategies.

A number of other papers study physician learning. Crawford and Shum (2005) use a Bayesian learning model to study within-patient learning for anti-ulcer drugs. Chan and Hamilton (2006) use a Bayesian learning model to study within-patient learning among AIDS patients. In addition to using clinical information (CD4 counts), the authors also use revealed patient preferences (in particular, decision to drop out of a trial) to understand patient

decisions. Narayanan and Manchanda (2009) use a Bayesian learning model to study heterogeneous learning among physicians prescribing erectile dysfunction drugs. Chintagunta, Jiang, and Jin (2009) use a Bayesian learning model to study within-patient and across-patient learning among physicians prescribing Cox-2 inhibitors. Ching (2010) uses a Bayesian learning model to study how physicians aggregate information about product attributes among drugs whose patents had recently expired. Chan, Narasimhan, and Xie (2010) use a Bayesian learning model to study how physicians prescribing erectile dysfunction drugs learn about multiple treatment attributes using patient-reported information. The frameworks used in these papers all differ from the framework used here in that they rely on analyst-observed signals to which the physicians (or patients) are assumed to be responding.

A related paper by Basu et al. (2014) uses PeT effects to study treatment effects in a population of schizophrenia patients similar to that studied here. However, that analysis focuses on measuring the benefits of treatments in smaller subgroups than the broad subgroups typically used in studies. It does not study learning or examine trends in the match rate or PeT effects over time.

The rest of the paper is organized as follows. In Section II, I discuss the data set used in this analysis. Section III lays out the econometric methods used in this paper. I discuss the results in Section IV. In Section V, I draw some conclusions.

II. Data

The data in this study come from a large Medicaid claims data set. The following criteria were used to determine which patients were included in the analysis: (i) at least one diagnosis of schizophrenia between 2002 and 2006, (ii) at least one prescription for an AAD between January 1, 2003 and June 30, 2005, (iii) age 19-64 on the date that the patient received their first AAD prescription (index date), and (iv) continuously enrolled during the full year preceding and following the index date. All patients considered were clean starters, defined as patients who had not received an AAD in the six months preceding their index date.

A. Outcome Variable

I measure physician productivity by the number of schizophrenia-related hospitalizations each patient had in the year following their index date. Schizophrenia is a syndrome that can manifest itself with positive symptoms and negative symptoms. The most common positive symptoms are delusions and hallucinations. Common negative symptoms include suicidal thoughts and effects on cognition. AADs can help in managing symptoms, especially positive symptoms. Since positive symptoms often result in hospitalizations, using this as a measure of performance for physicians captures a broad and important set of results from treatment.

B. Treatment and Control Variables

In this study, I consider the five most common AADs in use during the time period under consideration: risperidone, olanzapine, quetiapine, ziprasidone, and aripiprazole. I refer to risperidone and olanzapine as older drugs and quetiapine, ziprasidone, and aripiprazole as newer drugs. While risperidone and olanzapine entered the market first, all five drugs were

branded during the study time period. Therefore, I assume that costs did not factor into the physician's treatment decision. Although physicians can switch AADs during a course of treatment, I am interested in measuring the effect of the choice of AAD at the index date on schizophrenia-related hospitalizations. The treatment variable is therefore an indicator for whether the index AAD was an older drug. The older AADs are the ones that physicians had more experience with leading up to 2003, whereas the newer AADs are the ones physicians had less experience using.

I control for demographic factors (age and gender) that could be correlated with schizophrenia-related hospitalizations. To ensure that state-level variations are taken into account, I control for state-level fixed effects. Many comorbidities are controlled for, including congestive heart failure, obesity, and alcohol abuse. I also control for several area-level factors. Table 1 summarizes the patient characteristics.

C. Instrumental Variable

Although there are a number of controls, it is likely that there are unobserved factors (e.g., symptom severity) that influence the treatment decision and also are correlated with the number of schizophrenia-related hospitalizations. Therefore, I use the following instrumental variable (which is similar to those considered by Brookhart and Schneeweiss (2007)): the proportion of older drug prescriptions among all AAD prescriptions made in the six month prior to a patients index date by all physicians in the patient's three-digit zip code, apart from the patient's own physician. This variable is likely to be correlated with the likelihood of receiving an older drug prescription. However, this variable is unlikely to influence the number of

schizophrenia-related hospitalizations the patient would incur under receipt of either treatment.

Table 1: Patient Characteristics

	Older AADs (N = 24,491)	Newer AADs (N = 17,764)	
Variable	Mean	Mean	p-value
Average age, year (SD)	40.7 (10.9)	40.8 (10.6)	0.28
Female, %	54.6	62.7	<0.001
Health care utilization prior to index date, %			
Any neurological medicine	2.9	3.1	0.40
Any AAD	6.3	11.3	<0.001
Any non-AAD	75.4	81.0	<0.001
Any non-psychiatric office visit	59.8	64.6	<0.001
Any psychiatric office visit	22.8	25.7	<0.001
Any non-psychiatric hospitalization	9.8	10.4	0.06
Any schizophrenia hospitalization	17.0	13.8	<0.001
Comorbidities, %			
Average number of comorbidity indicators (SD)	1.24 (1.6)	1.34 (1.7)	<0.001
Congestive heart failure	1.2	1.5	0.02
Valvular disease	1.1	1.1	0.92
Other neurological disorders	6.1	6.3	0.44
Chronic pulmonary disease	11.3	13.4	<0.001
Diabetes without chronic complications	7.0	9.3	<0.001
Diabetes with chronic complications	1.3	1.5	0.01
Hypothyroidism	2.2	2.9	<0.001
Liver disease	2.7	2.8	0.56
Acquired immune deficiency syndrome	1.8	1.4	<0.001
Obesity	3.5	5.2	<0.001
Weight loss	1.3	1.2	0.48
Fluid and electrolyte disorders	5.1	5.6	0.02
Deficiency anemias	5.1	5.2	0.78
Alcohol abuse	10.2	10.0	0.36
Drug abuse	15.0	15.0	0.99
Psychoses	21.4	21.0	0.28
Depression	11.6	13.2	<0.001
Hypertension	15.5	17.8	<0.001
Number of schizophrenia hospitalizations (SD)	0.26 (1.06)	0.25 (0.92)	0.35

AAD, atypical antipsychotic drug

SD, standard deviation

III. Estimation Approach

A. PeT Effects

In order to estimate which treatment is most effective in each patient, we need an estimate of the treatment effect for each individual. There are several options that we can use for this purpose, each with deferent levels of specificity. We could simply use the TT for patients who were treated and the TUT for patients who were untreated. However, this would not offer a very fine estimate, since the treatment effect is known to vary across many other characteristics. A commonly used and more nuanced treatment effect parameter that we could use for this purpose is the conditional average treatment effect (CATE). The CATE measures the ATE conditional upon observable covariates, which the treatment effect is known to vary across. For example, an older drug may be known to work differently in a 55-year-old male than in a 22-year-old female. By conditioning on age and gender, the CATE is able to capture this difference in the treatment effect, and, hence, can provide a better patient-specific estimate of the treatment effect.

However, it is often the case that there are other factors that are unobserved to the analyst, but moderate the treatment effect. If these factors are known to the physician and influence the treatment decision, this can generate essential heterogeneity (Heckman, Urzua, and Vytlacil, 2006). Heckman (1997) has shown that, in the presence of essential heterogeneity, standard IV techniques do not identify treatment effect parameters of interest. In particular, the local average treatment effect (LATE) (introduced by Imbens and Angrist (1994)) will not be equal to the ATE.

Building on the marginal gain parameter introduced by Björklund and Moffitt (1987), Heckman and Vytlacil (1999) showed how the marginal treatment effect (MTE) can be used to build treatment effect parameters of interest. The MTE is an even more nuanced treatment effect parameter than the CATE. It is defined as the treatment effect conditional on observed covariates for an individual with unobserved characteristics such that they are indifferent between being treated or untreated. By integrating over appropriate regions of unobserved characteristics, the MTE can be used to obtain the ATE, TT, TUT, CATE, and other treatment effect parameters. Due to its importance in estimating treatment effect parameters in models with essential heterogeneity, I now provide background about the MTE.

As in Heckman and Vytlacil (1999), consider the Neyman-Fisher-Cox-Rubin (1990; 1935; 1958; 1978) model of potential outcomes:

$$Y_i = D_i Y_{1i} + (1 - D_i) Y_{0i} \qquad (1)$$

where $D_i = 0$ represents the untreated state, $D_i = 1$ represents the treated state, Y_{0i} is the outcome in the untreated state, and Y_{1i} is the outcome in the treated state. As in Basu et al. (2014), we assume

$$Y_{ji} = \mu_j(X_{Oi}, X_{Ui}, \nu_i) \text{ for } j = 0, 1 \qquad (2)$$

where X_{Oi} is a vector of observed characteristics, X_{Ui} is a vector of unobserved characteristics that are assumed to be correlated with the treatment decision, and ν_i is captures all other unobserved random variables. We assume $(X_O, X_U) \coprod \nu$ and $X_O \coprod X_U$ where $\coprod$ denotes statistical independence.

Assume the following latent model characterizes the treatment decision:

$$D_i^* = \mu_D(X_{Oi}, Z_i) - U_{Di} \qquad (3)$$

where $D_i = 1$ if $D_i^* \geq 0$, $D_i = 0$ if $D_i^* < 0$, μ_D is an unknown function, Z_i is a vector of observed characteristics that affect the treatment decision but do not affect the outcomes (i.e., they are the instruments), and U_{Di} captures unobserved characteristics that affect the treatment decision. As in Basu et al. (2014), we assume $U_D \coprod v$.

To use the notation used in related work (Heckman and Vytlacil, 1999; Basu et al., 2014), we denote the propensity score $Pr(D_i = 1|X_{Oi} = x_{Oi}, Z_i = z_i) = F_{U_D}\big(\mu_D(x_{Oi}, z_i)\big)$ as $P(x_{Oi}, z_i)$, where F_{U_D} is the cumulative distribution function for the random variable U_D. Denote the probability transform of U_{Di} by V_i so that $V_i = F_{U_D}(U_{Di})$. Therefore, we can write $D_i = 1$ if $P(x_{Oi}, z_i) \geq V_i$ and $D_i = 0$ if $P(x_{Oi}, z_i) < V_i$.

Using the above notation, the MTE is equal to $E_v(Y_1 - Y_0|X_O = x_O, V = P(x_O, z))$, which we denote by $MTE(x_O, v)$ where $v = P(x_O, z)$. Heckman and Vytlacil (1999) define the LIV as

$$\frac{\partial E_v\big(Y\big|X_O = x_O, P(X_O, Z) = P(x_O, z)\big)}{\partial P(x_O, z)}$$

which we denote by $LIV(x_O, v)$. Heckman and Vytlacil (1999) demonstrate $LIV(x_O, v) = MTE(x_O, v)$ so that LIV can be used to identify the MTE.

Basu (2014) uses the MTE to identify a new treatment effect parameter, the PeT effect:

$$PeT(x_O, z|D = 1) = \frac{1}{P(x_O, z)} \int_0^{P(x_O, z)} MTE(x_O, v)dv \qquad (4)$$

$$PeT(x_O, z|D = 0) = \frac{1}{1 - P(x_O, z)} \int_{P(x_O, z)}^1 MTE(x_O, v)dv \qquad (5)$$

For individuals receiving the treatment, the PeT effect is the TT conditional on x_O and z. For untreated individuals, the PeT effect is the TUT conditional on x_O and z. Basu (2014) shows

that PeT effects can explain more individual-level variability in treatment effects than CATEs. This is because PeT effects use more information that CATEs. PeT effects incorporate information about the decision that was made (D) and the circumstances under which the decision was made (Z).

PeT effects can be averaged to obtained common treatment effect parameters. Averaging all the PeT effects yields the ATE, averaging the PeT effects for all treated individuals yields the TT, and averaging the PeT effects for all untreated individuals yields the TUT.

B. Estimation Technique

As in Basu et al. (2014), I estimate the PeT effects in a two-stage process. In the first stage, I regress an indicator for receipt of an older drug on the IV and control variables using a probit model. The estimates from this regression are used to determine the fitted values. These fitted values are the propensity scores: the predicted probability of each patient's likelihood of receiving an older index AAD.

After obtaining the propensity scores, the MTEs are estimated using a control-function approach. The control function uses the propensity scores and controls to approximate the observed and unobserved components of the outcomes equation. The control function is represented by a generalized linear model with ¼-power link and negative binomial variance and is estimated using iterated, reweighted least squares (IRLS). Several goodness-of-fit tests were used to refine the specification of the model. After obtaining estimates of the control function, the first derivative of this function with respect to the propensity score was taken. This gives an estimate of $MTE(x_O, v)$.

For each patient, the MTEs are averaged over the appropriate region based on the patient's values of x_O, z, and d (see (4) and (5)). This process is carried out using numerical integration and provides an estimate of the PeT effect for each patient. Ninety-five percent confidence intervals were obtained by repeating the two-stage process on 1,000 bootstrapped samples.

C. Match Rate

For each observation, the PeT effect provides information about how index AAD choice affects the expected number of schizophrenia-related hospitalizations. A negative PeT effect implies that the patient's schizophrenia-related hospitalizations would be reduced by taking an older index AAD rather than a newer index AAD. A positive PeT effect implies that the patient's schizophrenia-related hospitalizations would be increased by taking an older index AAD rather than a newer index AAD. Since we have information about the actual index AAD for each patient, we can check if each patient took the index AAD predicted by the model to reduce schizophrenia-related hospitalizations. I create an indicator, m_i, that equals one when a patient took the index AAD predicted by the model to reduce schizophrenia-related hospitalizations and that equals zero when the patient took the index AAD predicted by the model to increase schizophrenia-related hospitalizations.

I break the data into 10 quarters, based on the index date for each patient (which falls between January 1, 2003 and June 30, 2005). For each quarter, I calculate the average value of m_i, which I denote by mr_q. This provides the estimated proportion of patients who were given the appropriate index AAD (defined as the index AAD that would result in fewer schizophrenia-

related hospitalizations for the patient) during each quarter. An upward trend in mr_q would suggest that physicians are learning.

Additionally, for each quarter, I calculate the average value of m_i for patients who were given an older index AAD, denoted by mr_o_q, and for patients who were given a newer index AAD, denoted by mr_n_q. These trends can provide additional useful information about possible physician learning.

To provide further information about physicians' ability to correctly match patients, I also examine related trends. In particular, I consider the proportion of patients who should be given an older index AAD among patients who actually received an older index AAD, denoted by $opt\_o\_rec\_o_q$, and among patients who received a newer index AAD, denoted by $opt\_o\_rec\_n_q$. Notice $opt\_o\_rec\_o_q = mr\_o_q$. If physicians are unable to determine which treatment works best for a given patient, then we would expect to see

$$opt\_o\_rec\_o_q = opt\_o\_rec\_n_q. \qquad (6)$$

However, if physicians are able to distinguish the optimal index AAD for at least some patients, we would expect

$$opt\_o\_rec\_o_q > opt\_o\_rec\_n_q. \qquad (7)$$

Furthermore, if physicians are learning we would expect $opt\_o\_rec\_o_q - opt\_o\_rec\_n_q$ to be increasing as q increases from 1 to 10.

D. Trends in Treatment Effects

To further look for evidence of learning, I examine trends in treatment effect parameters. Let ATE_q denote the ATE for each quarter, let TT_q denote the TT for each quarter, and let TUT_q denote the TUT for each quarter. We expect ATE_q to remain roughly constant

over the 10 quarters. If physicians are able to match at least some patients to the optimal index AAD, we would expect to see

$$TT_q < TUT_q. \qquad (8)$$

In other words, we would expect an older index AAD to be better at reducing schizophrenia-related hospitalizations among patients who were initiated on an older AAD than among patients who were initiated on a newer AAD. Furthermore, if physicians are learning, we would expect to see $|TT_q - ATE_q|$ and $|TUT_q - ATE_q|$ increasing over time.

E. Actual and Counterfactual Outcomes

As in Basu et al. (2014), I use PeT effects to examine counterfactual outcomes. For each patient, we know the actual number of schizophrenia-related hospitalizations that they had, $Hosp_i$. We can also calculate the expected number of schizophrenia-related hospitalizations they would have experienced if they took an index AAD from the alternative category. For patients who took an older index AAD, we can calculate their expected number of schizophrenia-related hospitalizations from choosing a newer index AAD as $Hosp_i - PeT_i$, where PeT_i is the estimated PeT effect for patient i. For patients who took a newer index AAD, we can calculate their expected number of schizophrenia-related hospitalizations from choosing an older index AAD as $Hosp_i + PeT_i$. Using these counterfactual outcomes, I calculate the average number of schizophrenia-related hospitalizations by quarter of index date for three different counterfactual scenarios: (i) all patients receive an older index AAD, (ii) all patients receive a newer index AAD, (iii) and all patients receive the index AAD that is predicted (based on their PeT effect) to minimize schizophrenia-related hospitalizations. These trends are

compared to the actual trend in schizophrenia-related hospitalizations to provide information about how different treatment strategies would be expected to modify outcomes.

IV. Results

In the 21 states included in the analysis, 24,491 patients were initiated on an older AAD and 17,764 patients were initiated on a newer AAD between January 1, 2003 and June 30, 2005. There are several differences in patient-level characteristics across the two groups of patients (Table 1). Patients initiated on an older AAD were more likely to be male and to have had a schizophrenia related-hospitalization in the year prior to their index date. Patients initiated on a newer AAD were more likely to have taken an AAD in the year prior to the index date, to have had a psychiatric office visit, and to have had a non-psychiatric office visit. More comorbidities in the year prior to the index date were found among patients initiated on a newer AAD. The average number of schizophrenia related-hospitalizations in the year following the index date is not significantly different across the two groups (p-value = 0.35).

In the first-stage probit regression, the IV was found to be significant (p-value < 0.001). An IV-only propensity-score model was fitted to the data, and patients were grouped dichotomously: above and below the median of the IV-only propensity score. Patient characteristics were well-balanced across the two groups.

A. Matching

Over the two-and-a-half-years of index dates, physicians matched 57.8% of patients to the index AAD category predicted to minimize schizophrenia related-hospitalizations. Interestingly, there is a downward trend in the match rate from a high of 67.7% in the first quarter of 2003 to a low of 48.5% in the second quarter of 2005 (Table 2; Figure 1). Among patients initiated on an older AAD, the match rate is quite high (match rate of 96.0% over two-and-a-half years) and relatively constant over the 10 quarters. Among patients initiated on a

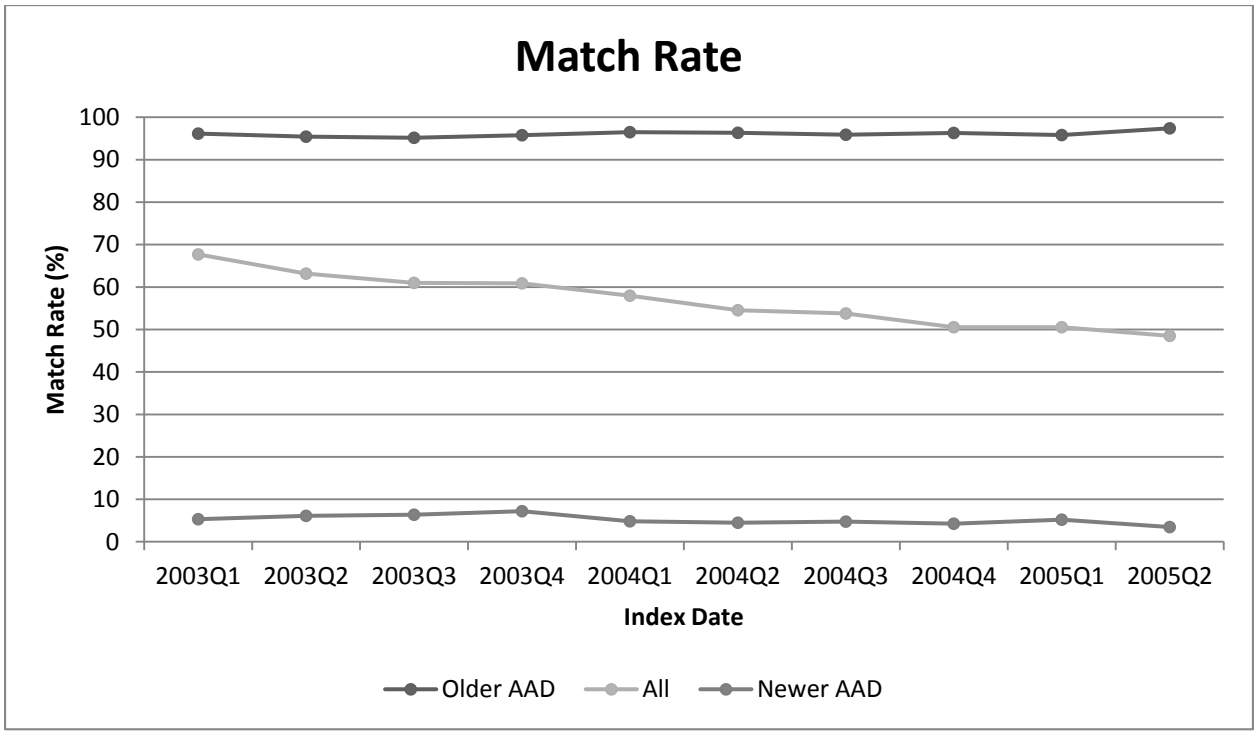
Table 2: Match Rate

	Match Rate (%)		
Index Date	Older AAD	All	Newer AAD
2003Q1	96.2	67.7	5.3
	(83.8, 97.1)	(63, 68.3)	(3.6, 18.5)
2003Q2	95.4	63.2	6.1
	(83.9, 96.5)	(59.7, 64.1)	(4.2, 18.4)
2003Q3	95.2	61.0	6.4
	(83.2, 96.6)	(57.6, 62.2)	(3.9, 19.8)
2003Q4	95.8	60.9	7.2
	(83.7, 96.9)	(58, 61.9)	(4.6, 20.3)
2004Q1	96.5	58.0	4.8
	(85.9, 97)	(55.7, 59.2)	(3.8, 16.9)
2004Q2	96.3	54.5	4.5
	(85.4, 97)	(52.5, 56.1)	(3.5, 15.9)
2004Q3	95.9	53.8	4.8
	(84.9, 97)	(52.1, 55.7)	(3.9, 16.5)
2004Q4	96.3	50.5	4.3
	(85.3, 97.3)	(49.1, 52.8)	(3.3, 16.6)
2005Q1	95.8	50.5	5.2
	(84.6, 97.1)	(48.8, 52.7)	(3.6, 16.9)
2005Q2	97.4	48.5	3.5
	(86.9, 97.6)	(46.8, 50.5)	(2.9, 13.8)
All Quarters	96.0	57.8	5.2
	(85.1, 96.7)	(56.2, 58.2)	(4, 16.9)

95% CIs in parentheses

newer AAD, the match rate is quite low (match rate of 5.2% over the two-and-a-half years) and relatively constant, but with a slight downward trend. Between the first quarter of 2003 and the second quarter of 2005, the percentage of patients initiated on a newer AAD steadily rose from 31.3% to 52.0%. This suggests that the decreasing match rate is driven by physicians initiating more patients on newer AADs, where they are less successful at matching patients to the appropriate index AAD.

Figure 1: Match Rate



It is also useful to consider the percentage of patients who are predicted to have the fewest schizophrenia-related hospitalizations from initiating with an older AAD. Among all patients, 95.5% would minimize schizophrenia-related hospitalizations by initiating with an older index AAD. This rate is relatively constant over the two-and-a-half years of data. Among patients initiated on a newer AAD, 94.8% would minimize schizophrenia-related hospitalizations by initiating with an older index AAD. Whereas 96.0% of patients initiated on an older AAD would minimize schizophrenia-related hospitalizations by initiating with an older index AAD. In all 10 quarters, physicians are showing some discrimination in their ability to correctly match patients to the appropriate index AAD category (Table 3 and Figure 2). That is, equation (7) is true for all 10 quarters of data. However, the difference $opt\_o\_rec\_o_q - opt\_o\_rec\_n_q$ is relatively constant over the 10 quarters (with a low of 0.6% and a high of 3.0%). If this difference was increasing over time, this would have provided some

Table 3: Percentage of Patients with Negative PeT Effects

	Patients with Negative PeT Effects (%)		
Index Date	Older AAD	All	Newer AAD
2003Q1	96.2	95.7	94.7
	(83.8, 97.1)	(83.1, 96.8)	(81.5, 96.4)
2003Q2	95.4	94.9	93.9
	(83.9, 96.5)	(83.3, 96.3)	(81.6, 95.8)
2003Q3	95.2	94.6	93.6
	(83.2, 96.6)	(82.5, 96.2)	(80.2, 96.1)
2003Q4	95.8	94.6	92.8
	(83.7, 96.9)	(82.4, 96.2)	(79.7, 95.4)
2004Q1	96.5	95.9	95.2
	(85.9, 97)	(84.7, 96.5)	(83.1, 96.2)
2004Q2	96.3	96.0	95.5
	(85.4, 97)	(85, 96.5)	(84.1, 96.5)
2004Q3	95.9	95.6	95.2
	(84.9, 97)	(84.4, 96.7)	(83.5, 96.1)
2004Q4	96.3	96.0	95.7
	(85.3, 97.3)	(84.5, 96.9)	(83.4, 96.7)
2005Q1	95.8	95.3	94.8
	(84.6, 97.1)	(83.8, 96.6)	(83.1, 96.4)
2005Q2	97.4	96.9	96.5
	(86.9, 97.6)	(86.7, 97.3)	(86.2, 97.1)
All quarters	96.0	95.5	94.8
	(85.1, 96.7)	(84.5, 96.2)	(83.1, 96)

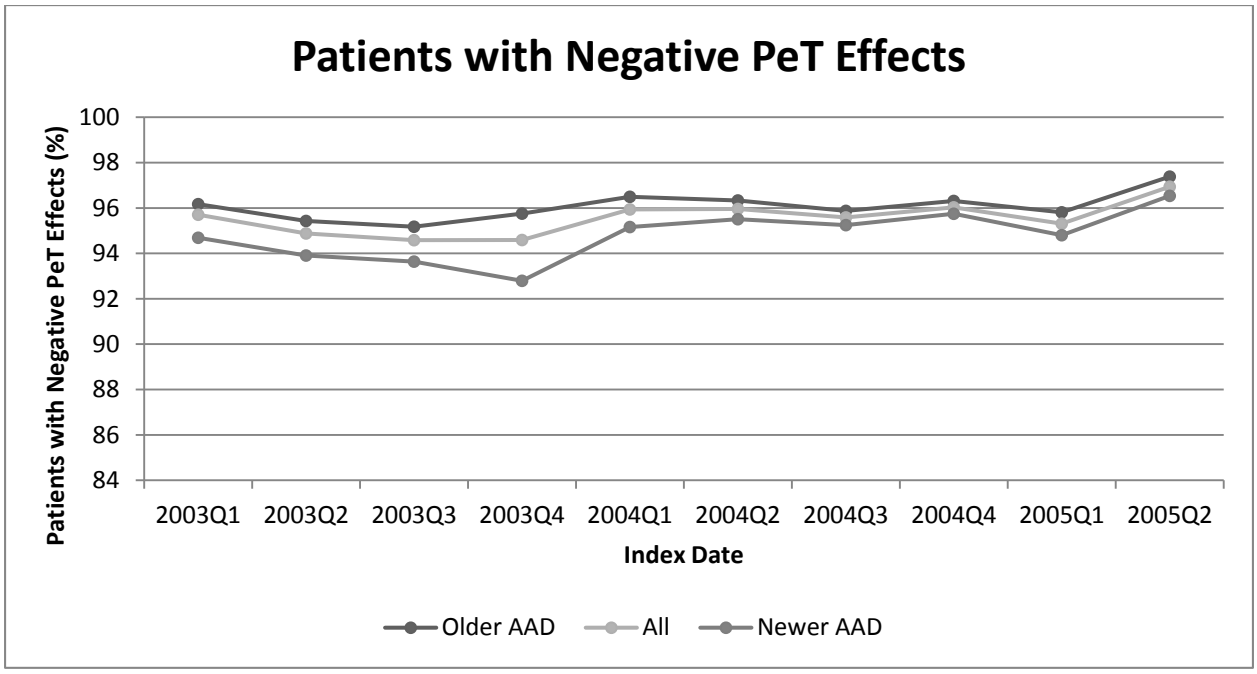
95% CIs in parentheses

evidence of learning. To further investigate whether physicians are learning, we now examine trends in treatment effects.

B. Trends in Treatment Effects

The ATE is -0.88 over the two-and-a-half year time period. This indicates that initiating treatment with an older index AAD is expected to result in almost 1 fewer schizophrenia-related hospitalization in the year following initiation of treatment compared to using a

Figure 2: Patients with Negative PeT Effects



newer AAD. Over the same period, the TT is -1.22 and the TUT is -0.41. This shows that the patients that physicians are initiating on an older index AAD are receiving a much larger benefit from treatment that the patients initiated on a newer AAD would receive. However, the negative TUT shows that physicians are not matching these patients well, which is consistent with the low match rate among these patients. There is a downward trend in all three parameters over time (Table 4; Figure 3). The trend is stronger with the TT than with the other parameters, which we now explore further.

Table 5 and Figure 4 show the trends in $TT - ATE$ and $TUT - ATE$. The difference in the TUT and ATE is very stable over the 10 quarters with a mean of 0.47. However, the difference in the TT and ATE is growing over time from -0.22 in the first quarter of 2003 to -0.48

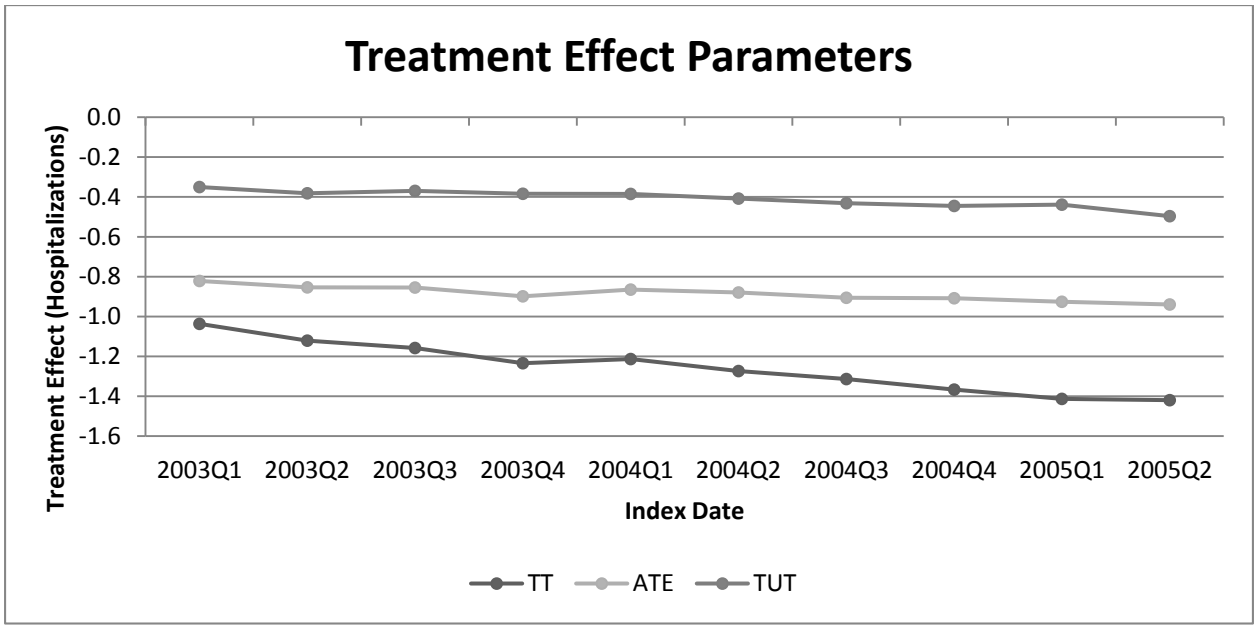
Table 4: Treatment Effect Parameters

	Treatment Effect Parameters		
Index Date	TT	ATE	TUT
2003Q1	-1.04	-0.82	-0.35
	(-1.47, -0.81)	(-1.12, -0.64)	(-0.4, -0.23)
2003Q2	-1.12	-0.85	-0.38
	(-1.61, -0.87)	(-1.18, -0.65)	(-0.46, -0.25)
2003Q3	-1.16	-0.85	-0.37
	(-1.67, -0.9)	(-1.19, -0.65)	(-0.44, -0.25)
2003Q4	-1.23	-0.90	-0.38
	(-1.78, -0.96)	(-1.24, -0.69)	(-0.46, -0.26)
2004Q1	-1.21	-0.86	-0.38
	(-1.74, -0.94)	(-1.19, -0.67)	(-0.45, -0.26)
2004Q2	-1.27	-0.88	-0.41
	(-1.84, -1.01)	(-1.21, -0.68)	(-0.48, -0.29)
2004Q3	-1.31	-0.91	-0.43
	(-1.88, -1.03)	(-1.24, -0.71)	(-0.5, -0.3)
2004Q4	-1.37	-0.91	-0.44
	(-2, -1.07)	(-1.25, -0.7)	(-0.52, -0.32)
2005Q1	-1.41	-0.93	-0.44
	(-2.04, -1.11)	(-1.27, -0.71)	(-0.53, -0.3)
2005Q2	-1.42	-0.94	-0.50
	(-2.08, -1.11)	(-1.3, -0.73)	(-0.59, -0.36)
All quarters	-1.22	-0.88	-0.41
	(-1.74, -0.96)	(-1.2, -0.69)	(-0.48, -0.29)

95% CIs in parentheses

in the second quarter of 2005. If both differences were growing over time, this would provide strong evidence of learning. However, the trend we see suggests a different story. When $|TUT - ATE|$ remains constant over time while $|TT - ATE|$ increases, this suggests that the patients who physicians are choosing to initiate on a newer AAD rather than an older AAD (there is a 20% growth in the proportion of patients taking a newer AAD over the two-and-a-

Figure 3: Treatment Effect Parameters

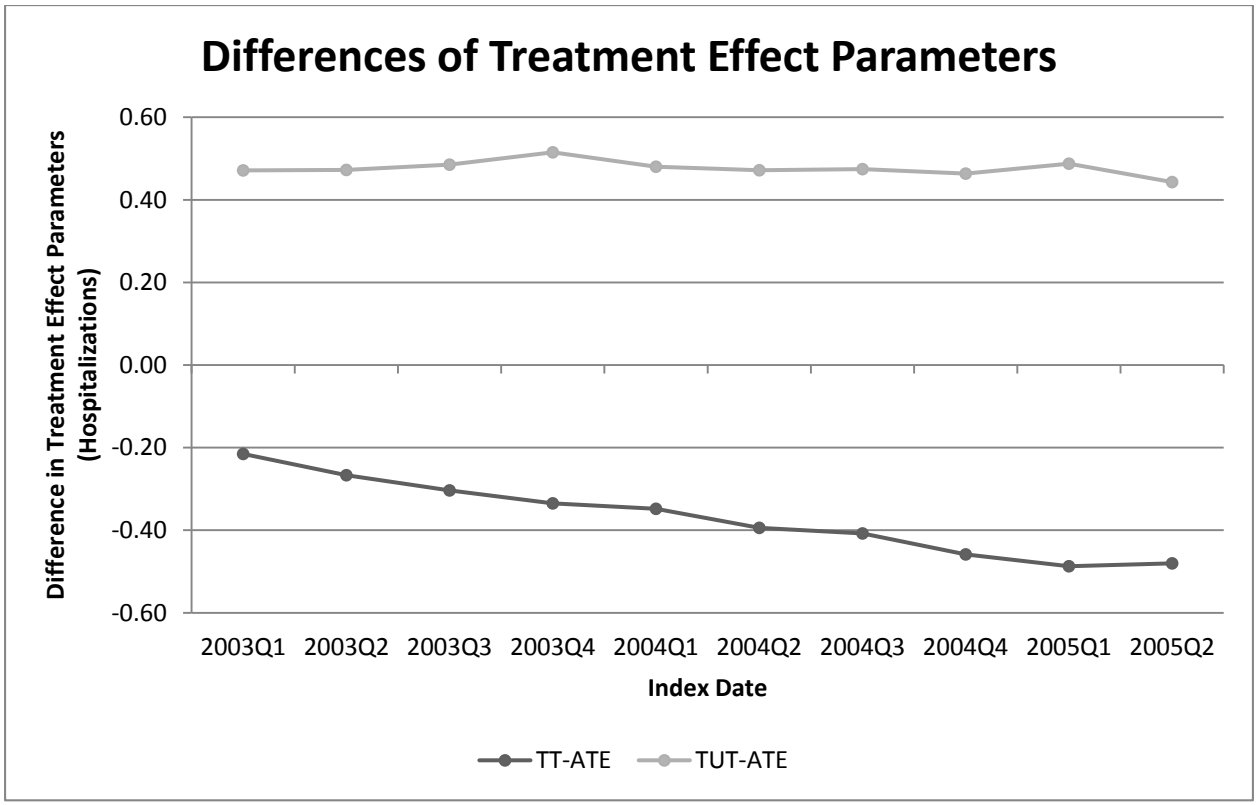


half years) are not ones who would be significantly harmed by initiating with the wrong AAD. In fact, the average PeT effect for these new patients beginning treatment on a newer AAD

Table 5: Differences of Treatment Effect Parameters

	Differences of Treatment Effect Parameters	
Index Date	TT-ATE	TUT-ATE
2003Q1	-0.22	0.47
2003Q2	-0.27	0.47
2003Q3	-0.30	0.49
2003Q4	-0.34	0.52
2004Q1	-0.35	0.48
2004Q2	-0.39	0.47
2004Q3	-0.41	0.47
2004Q4	-0.46	0.46
2005Q1	-0.49	0.49
2005Q2	-0.48	0.44
All quarters	-0.34	0.47

Figure 4: Differences of Treatment Effect Parameters



appears to be very close to the overall TUT value of -0.41. Moving these patients from an older AAD to a newer AAD necessarily increases the absolute value of the TT over time. Taken together, this suggests at least two possible stories. One explanation is that physicians are trying to learn by initiating more patients on a newer AAD over time, but the two-and-a-half year time period is not long enough to see the benefits of learning. Despite the lack of improved outcomes, physicians are leaving the patients who have the larger benefits from an older AAD over a newer AAD on an older AAD. An alternative explanation is that physicians are trying newer drugs because they are receiving non-pecuniary benefits from pharmaceutical companies trying to promote their new products. In this case, physicians are not necessarily trying to learn. However, due perhaps to liability concerns, physicians only initiate patients on

a newer AAD who are not expected to be significantly harmed, in terms of schizophrenia-related hospitalizations, relative to initiating on an older AAD.

C. Actual and Counterfactual Outcomes

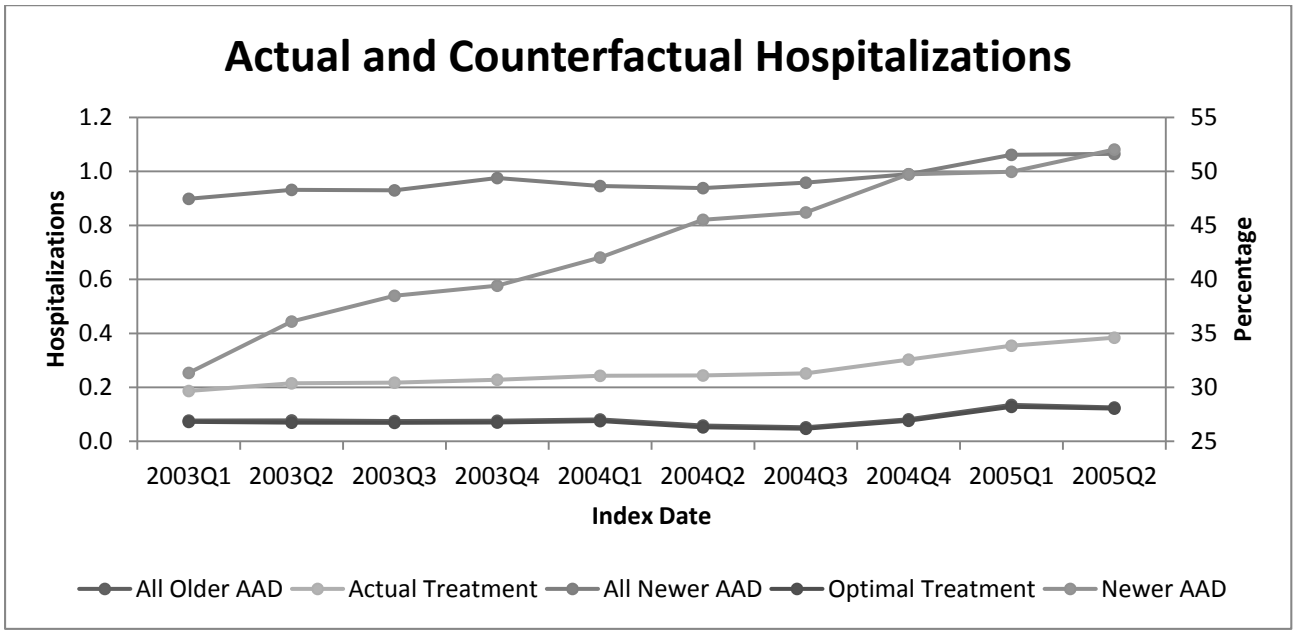
Over the 10 quarters of index dates, schizophrenia-related hospitalizations steadily increase from 0.187 in the first quarter of 2003 to 0.384 in the second quarter of 2005 (Table 6; Figure 5). This represents the growth in schizophrenia-related hospitalizations when physicians are free to choose whichever treatment they want for a given patient. If physicians instead

Table 6: Actual and Counterfactual Hospitalizations

	Hospitalizations				Percentage
Index Date	All Older AAD	Actual Treatment	All Newer AAD	Optimal Treatment	Newer AAD
2003Q1	0.077	0.187	0.899	0.072	31.3
	(0.051, 0.125)		(0.745, 1.184)	(0.042, 0.095)	
2003Q2	0.077	0.215	0.931	0.070	36.1
	(0.04, 0.145)		(0.779, 1.241)	(0.026, 0.105)	
2003Q3	0.075	0.217	0.930	0.069	38.5
	(0.035, 0.136)		(0.768, 1.239)	(0.027, 0.1)	
2003Q4	0.077	0.228	0.976	0.070	39.4
	(0.03, 0.146)		(0.822, 1.3)	(0.02, 0.108)	
2004Q1	0.081	0.243	0.946	0.075	42.0
	(0.046, 0.14)		(0.794, 1.253)	(0.035, 0.107)	
2004Q2	0.059	0.244	0.938	0.052	45.5
	(0.02, 0.122)		(0.794, 1.252)	(0.009, 0.09)	
2004Q3	0.052	0.252	0.958	0.047	46.2
	(0.014, 0.115)		(0.806, 1.285)	(0.005, 0.08)	
2004Q4	0.082	0.303	0.990	0.077	49.7
	(0.035, 0.152)		(0.827, 1.303)	(0.026, 0.12)	
2005Q1	0.135	0.354	1.061	0.128	50.0
	(0.089, 0.207)		(0.893, 1.388)	(0.076, 0.173)	
2005Q2	0.126	0.384	1.065	0.122	52.0
	(0.078, 0.193)		(0.9, 1.416)	(0.069, 0.169)	
All quarters	0.082	0.253	0.961	0.076	42.0
	(0.052, 0.133)		(0.813, 1.259)	(0.045, 0.097)	

95% CIs in parentheses

Figure 5: Actual and Counterfactual Hospitalizations



were to initiate all patients on a newer AAD, schizophrenia-related hospitalizations would be expected to increase from 0.899 in the first quarter of 2003 to 1.065 in the second quarter of 2005. This represents a significant increase in schizophrenia-related hospitalizations compared to the outcomes we see when physicians are allowed to personalize treatment. Alternatively, if physicians were to initiate all patients on an older AAD, schizophrenia-related hospitalizations would be expected to increase from 0.077 in the first quarter of 2003 to 0.126 in the second quarter of 2005. The represents a significant reduction compared to outcomes achieved when physicians are personalizing care.

If physicians chose the index AAD predicted to minimize schizophrenia-related hospitalizations, they would do even better than the outcomes achieved from initiating all patients on an older AAD. However, the improvement in outcomes would be small. Over all 10 quarters, the average number of schizophrenia-related hospitalizations is expected to be 0.082

when all patients are initiated on an older AAD and 0.076 when patients are initiated on the AAD predicted to minimize schizophrenia-related hospitalizations. The improvement is small due to the high proportion of patients (95.5%) that are expected to have fewer schizophrenia-related hospitalizations by initiating with an older AAD.

It is interesting to note that although an older index AAD appears to be superior to a newer index AAD, there is a growth in patients initiated with a newer AADs (31.3% to 52.0%) over the 10 quarters of index dates.

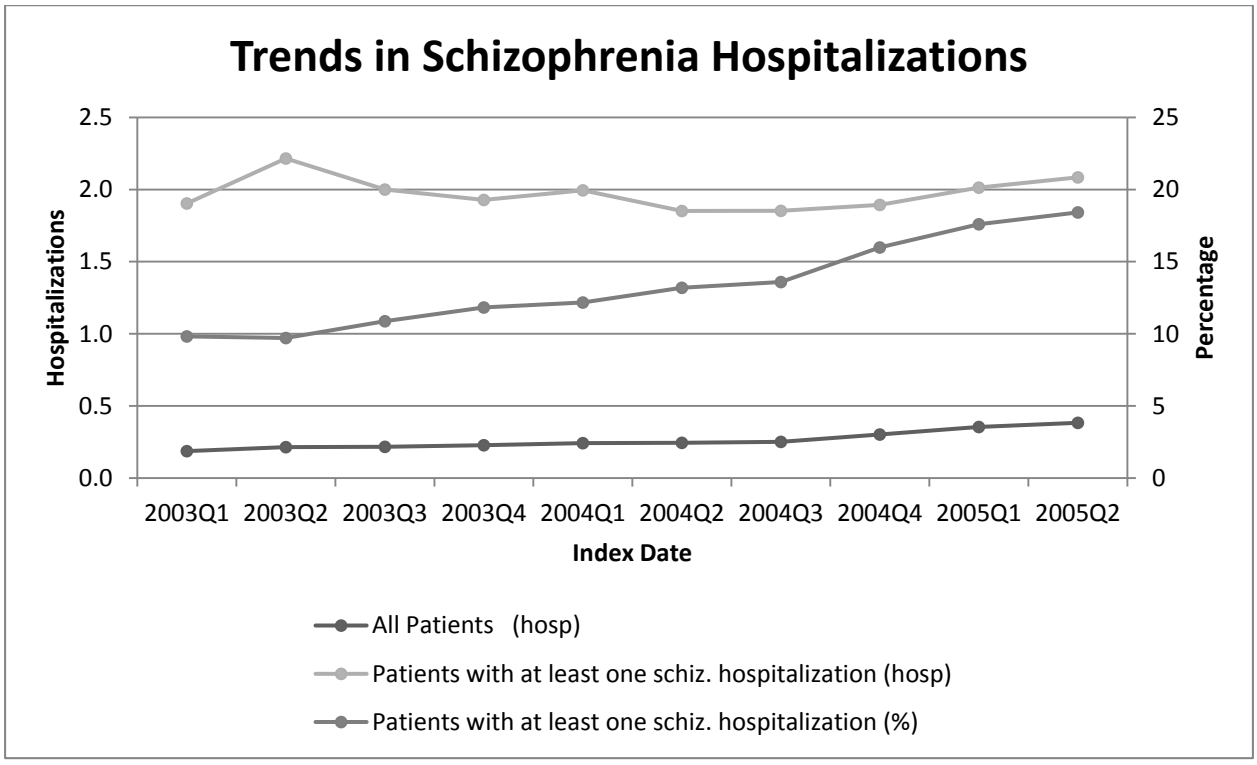
D. Potential Costs Associated with Learning

Table 7 and Figure 6 show three trends in schizophrenia-related hospitalizations. We see that among all patients, average schizophrenia-related hospitalizations steadily increases from 0.19 in the first quarter of 2003 to 0.38 in the second quarter of 2005. However, among patients who experienced at least one schizophrenia-related hospitalizations, schizophrenia-related hospitalizations stayed roughly constant over the 10 quarters of index dates. Over the

Table 7: Trends in Schizophrenia Hospitalizations

	Hospitalizations		Percentage
Index Date	All Patients (hosp)	Patients with at least one schiz. hospitalization (hosp)	Patients with at least one schiz. hospitalization (%)
2003Q1	0.19	1.90	9.8
2003Q2	0.22	2.22	9.7
2003Q3	0.22	2.00	10.9
2003Q4	0.23	1.93	11.8
2004Q1	0.24	1.99	12.2
2004Q2	0.24	1.85	13.2
2004Q3	0.25	1.85	13.6
2004Q4	0.30	1.89	16.0
2005Q1	0.35	2.01	17.6
2005Q2	0.38	2.08	18.4
All quarters	0.25	1.97	12.8

Figure 6: Trends in Schizophrenia Hospitalizations



same time period, we see that the proportion of patients with at least one schizophrenia-related hospitalizations steadily increases from 9.8% in the first quarter of 2003 to 18.4% in the second quarter of 2005. This suggests that growth in schizophrenia-related hospitalizations is not driven by patients being very poorly matched to the initial AAD category and, thus, experiencing a large number of schizophrenia-related hospitalizations. Instead, this suggest that patients who otherwise would have experienced no schizophrenia-related hospitalizations are now experiencing schizophrenia-related hospitalizations. As mentioned above, there is a 0.21 increase in the proportion of patients who are initiated on newer AADs over the two-and-a-half years of index dates. We also saw that the average PeT effects for these new types of patients that are initiated on a newer AAD is near the overall TUT value of -0.41. Taken

together, $(0.21 \times 0.41) = 0.09$ of the 0.19 increase in schizophrenia-related hospitalizations may be due to trying new patients on newer AADs. Whether the reason for this growth in use of newer index AADs is due to physicians trying to learn or to non-pecuniary benefits that physicians receive from pharmaceutical companies, these results suggest that physicians are willing to trade off current patient health (for future benefits to patients, for their own personal benefits, or for both).

V. Conclusions

This paper uses recently developed econometric methods to study the existence of physician learning. I find that while physicians demonstrate some ability to match patients to the appropriate index AAD category, matching is not improving over time, at least during the short time horizon considered here. However, I do not find that physicians are holding initial AAD choice constant over time. In fact, there is large growth in the proportion of patients initiated on a newer AAD. This may suggest that physicians may be trying to learn about how to prescribe to a heterogeneous population of patients, but that the time horizon considered in this study is not long enough to see improved matching. Alternatively, this could suggest that physicians are increasing newer AAD use due to non-pecuniary benefits that they receive from pharmaceutical companies of the new drugs. In either case, the increase in use of newer AADs over the two-and-a-half year time period is leading to more schizophrenia-related hospitalizations. This reveals that physicians are willing to trade off current patient health.

An important implication of this work is that it was able to use observational data to find similar results to a large clinical trial. In particular, the Clinical Antipsychotic Trials of Intervention Effectiveness (CATIE) investigators found that olanzapine had the longest time to discontinuation, among the antipsychotics studied (Lieberman et al., 2005). In the above, I found that initiation of therapy with an older AAD (i.e., olanzapine or risperidone) resulted in -0.88 (-1.2, -0.69) fewer hospitalizations compared to initiation with a newer AAD. This suggests that methods such as those used in this paper may be useful for arriving at important results in a more inexpensive manner than a clinical trial.

One limitation of this study is the length of the time horizon considered. A data set that provides an additional five years of index dates would potentially be very useful, as it may provide a more definitive conclusion about whether physicians are actually learning. It is plausible that physicians experiment with newer-AAD use for several years before they begin to learn enough to improve matching. However, due to the results of the CATIE trial being released in late 2005 (Lieberman et al., 2005), physicians treatment decisions may have been strongly influenced by this information in the period following the time period studied here.

The use of PeT effects in this paper provided a unique way to study learning by enabling us to examine counterfactual outcomes that are specific to each patient. This nuanced treatment effect parameter is better able to capture heterogeneity across patients (Basu, 2014). Therefore, this methodology would be valuable for studying treatment effects in other diseases where heterogeneity in treatment response is known to be important.

As opportunities for personalization continue to grow (Gautschi et al., 2008), physicians will continue to be faced with the challenge of learning how to discern the optimal treatment for patients. Due to limited resources, answers to these questions will not always be able to be studied using clinical trials. Therefore, physicians will need to engage in learning on their own to improve patient outcomes. Using methods like the one used in this paper allow us to understand how well physicians are able to learn. Knowledge of areas where physicians are successfully learning can be very valuable, as successful learning techniques may be able to be transferred to other areas where learning is slow or nonexistent.

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Chapter 2: Geographic Variations in Physician Matching and Treatment Effects Among Medicaid Patients with Schizophrenia

I. Introduction

There is a large health economics literature studying geographic variation in health care costs, utilization, and outcomes. Understanding regional variation in these measures can be useful for uncovering important features of the workings of the U.S. health care system. For example, this research has found that high costs areas do not necessarily correspond to areas that have the best outcomes (IOM, 2013).

Several hotly debated questions revolve around the quality of care in the U.S. Is there variation in quality of care? If so, how can this be measured? What factors can explain differences in quality of care? In this paper, I attempt to address some of these questions in a new way, using novel econometric techniques. In particular, I study regional variation in how well physicians match patients to the appropriate therapy (for a particular class of drugs).

Determining an appropriate drug match requires modeling counterfactual patient outcomes. This is a challenging task in a model of heterogeneous returns with endogeneity concerns. In such a setting, issues of sorting on the level and sorting on the gain arise. Heckman (1997) demonstrated that, in this setting, typical instrumental variables (IV) methods do not estimate treatment effect parameters of interest. Since this discovery, a series of papers (Heckman and Vytlacil (1999); Heckman, Urzua, and Vytlacil (2006); Basu (2014)) have followed that have developed local IV (LIV) methods that can be used to estimate common treatment effect parameters (e.g., the average treatment effect (ATE), the effect of the treatment on the treated (TT), and the effect of the treatment on the untreated (TUT)) in these

circumstances. While these parameters can be used to model counterfactual outcomes on subgroups of the population, they are not personalized for each individual in the population.

Building on this research, a recent paper by Basu (2014) developed a new treatment effect parameter, the Person-centered Treatment (PeT) effect, which uses information about the individual's treatment decision and the circumstances under which they made the decision to estimate a personalized treatment effect for each individual. Using these PeT effects, we can analyze the treatment decision made for each patient to determine if it is optimal. By looking at regional variation in matching, we can seek to better understand how quality of care varies across the country.

In this paper, I use the methods described above to study geographic variation in the quality of care. I examine a large data set of Medicaid patients who have been diagnosed with schizophrenia and have been treated with atypical antipsychotic drugs (AADs). Each patient can either take an older drug (the treatment) or a newer drug to treat their symptoms. The outcome of interest is hospitalizations in the year following initiation of the therapy. Using LIV methods, I estimate a PeT effect for each patient. From this estimate, I can determine whether the optimal treatment for the patient was an older drug (PeT effect less than zero) or a newer drug (PeT effect greater than zero). By looking at the match rate (proportion of patients who received their optimal treatment) in each region, I am able to study geographic variation in the quality of care. Furthermore, by breaking down the match rates across time, I am able to see if this variation changes over time, as physicians gain more experience with the newer therapies.

I find that there is large variation across the country in the ability of physicians to match patients to the appropriate therapy. Furthermore, this variation is stable over the two-and-a-

half year time period studied. To look for further evidence of variation in quality of care, I looked at variation in region-specific treatment effects on the treated and the untreated. There is especially large regional variation in the effect of the treatment on the treated, providing further evidence for important geographic differences in the quality of care. I also study trends in region-specific treatment effects on the treated and the untreated.

The rest of the paper is organized as follows. In Section II, I consider the data used in this study. Section III lays out the estimation approach. The results are presented in Section IV. Section V draws together some conclusions.

II. Data

The data in this study come from a large Medicaid claims data set. The following criteria were used to determine which patients were included in the analysis: (i) at least one diagnosis of schizophrenia between 2002 and 2006, (ii) at least one prescription for an AAD between January 1, 2003 and June 30, 2005, (iii) age 19-64 on the date that the patient received their first AAD prescription (index date), and (iv) continuously enrolled during the full year preceding and following the index date. All patients considered were clean starters, defined as patients who had not received an AAD in the six months preceding their index date.

A. Outcome Variable

I measure physician productivity by the number of schizophrenia-related hospitalizations each patient had in the year following their index date. Schizophrenia is a syndrome that can manifest itself with positive symptoms and negative symptoms. The most common positive symptoms are delusions and hallucinations. Common negative symptoms include suicidal thoughts and effects on cognition. AADs can help in managing symptoms, especially positive symptoms. Since positive symptoms often result in hospitalizations, using this as a measure of performance for physicians captures a broad and important set of results from treatment.

B. Treatment and Control Variables

In this study, I consider the five most common AADs in use during the time period under consideration: risperidone, olanzapine, quetiapine, ziprasidone, and aripiprazole. I refer to risperidone and olanzapine as older drugs and quetiapine, ziprasidone, and aripiprazole as newer drugs. While risperidone and olanzapine entered the market first, all five drugs were

branded during the study time period. Therefore, I assume that costs did not factor into the physician's treatment decision. Although physicians can switch AADs during a course of treatment, I am interested in measuring the effect of the choice of AAD at the index date on schizophrenia-related hospitalizations. The treatment variable is therefore an indicator for whether the index AAD was an older drug. The older AADs are the ones that physicians had more experience with leading up to 2003, whereas the newer AADs are the ones physicians had less experience using.

I control for demographic factors (age and gender) that could be correlated with schizophrenia-related hospitalizations. To ensure that state-level variations are taken into account, I control for state-level fixed effects. Many comorbidities are controlled for, including congestive heart failure, obesity, and alcohol abuse. I also control for several area-level factors. Table 1 summarizes the patient characteristics.

C. Instrumental Variable

Although there are a number of controls, it is likely that there are unobserved factors (e.g., symptom severity) that influence the treatment decision and also are correlated with the number of schizophrenia-related hospitalizations. Therefore, I use the following instrumental variable (which is similar to those considered by Brookhart and Schneeweiss (2007)): the proportion of older drug prescriptions among all AAD prescriptions made in the six month prior to a patient's index date by all physicians in the patient's three-digit zip code, apart from the patient's own physician. This variable is likely to be correlated with the likelihood of receiving an older drug prescription. However, this variable is unlikely to influence the number of

schizophrenia-related hospitalizations the patient would receive under receipt of either treatment.

Table 1: Patient Characteristics

	Older AADs (N = 22,208)	Newer AADs (N = 15,716)	
Variable	Mean	Mean	p-value
Average age, year (SD)	40.7 (10.9)	40.9 (10.6)	0.29
Female, %	54.3	62.3	<0.001
Health care utilization prior to index date, %			
Any neurological medicine	2.9	3.0	0.48
Any AAD	6.3	11.3	<0.001
Any non-AAD	75.4	81.2	<0.001
Any non-psychiatric office visit	59.6	64.3	<0.001
Any psychiatric office visit	22.6	25.2	<0.001
Any non-psychiatric hospitalization	10.0	10.5	0.10
Any schizophrenia hospitalization	17.3	14.1	<0.001
Comorbidities, %			
Average number of comorbidity indicators (SD)	1.25 (1.6)	1.36 (1.7)	<0.001
Congestive heart failure	1.3	1.5	0.07
Valvular disease	1.1	1.1	0.79
Other neurological disorders	6.2	6.4	0.54
Chronic pulmonary disease	11.4	13.7	<0.001
Diabetes without chronic complications	7.0	9.4	<0.001
Diabetes with chronic complications	1.3	1.6	0.02
Hypothyroidism	2.1	2.9	<0.001
Liver disease	2.7	2.9	0.29
Acquired immune deficiency syndrome	2.0	1.6	<0.01
Obesity	3.5	5.2	<0.001
Weight loss	1.4	1.3	0.52
Fluid and electrolyte disorders	5.0	5.4	0.08
Deficiency anemias	5.3	5.3	0.73
Alcohol abuse	10.5	10.3	0.53
Drug abuse	15.5	15.5	0.87
Psychoses	21.6	21.1	0.23
Depression	11.7	13.1	<0.001
Hypertension	15.7	18.0	<0.001
Number of schizophrenia hospitalizations (SD)	0.27 (1.09)	0.25 (0.95)	0.18

AAD, atypical antipsychotic drug

SD, standard deviation

III. Estimation Approach

A. PeT Effects

In order to estimate which treatment is most effective in each patient, we need an estimate of the treatment effect for each individual. There are several options that we can use for this purpose, each with deferent levels of specificity. We could simply use the TT for patients who were treated and the TUT for patients who were untreated. However, this would not offer a very fine estimate, since the treatment effect is known to vary across many other characteristics. A commonly used and more nuanced treatment effect parameter that we could use for this purpose is the conditional average treatment effect (CATE). The CATE measures the ATE conditional upon observable covariates, which the treatment effect is known to vary across. For example, an older drug may be known to work differently in a 55-year-old male than in a 22-year-old female. By conditioning on age and gender, the CATE is able to capture this difference in the treatment effect, and, hence, can provide a better patient-specific estimate of the treatment effect.

However, it is often the case that there are other factors that are unobserved to the analyst, but moderate the treatment effect. If these factors are known to the physician and influence the treatment decision, this can generate essential heterogeneity (Heckman, Urzua, and Vytlacil, 2006). Heckman (1997) has shown that, in the presence of essential heterogeneity, standard IV techniques do not identify treatment effect parameters of interest. In particular, the local average treatment effect (LATE) (introduced by Imbens and Angrist (1994)) will not be equal to the ATE.

Building on the marginal gain parameter introduced by Björklund and Moffitt (1987), Heckman and Vytlacil (1999) showed how the marginal treatment effect (MTE) can be used to build treatment effect parameters of interest. The MTE is an even more nuanced treatment effect parameter than the CATE. It is defined as the treatment effect conditional on observed covariates for an individual with unobserved characteristics such that they are indifferent between being treated or untreated. By integrating over appropriate regions of unobserved characteristics, the MTE can be used to obtain the ATE, TT, TUT, CATE, and other treatment effect parameters. Due to its importance in estimating treatment effect parameters in models with essential heterogeneity, I now provide background about the MTE.

As in Heckman and Vytlacil (1999), consider the Neyman-Fisher-Cox-Rubin (1990; 1935; 1958; 1978) model of potential outcomes:

$$Y_i = D_i Y_{1i} + (1 - D_i) Y_{0i} \qquad (1)$$

where $D_i = 0$ represents the untreated state, $D_i = 1$ represents the treated state, Y_{0i} is the outcome in the untreated state, and Y_{1i} is the outcome in the treated state. As in Basu et al. (2014), we assume

$$Y_{ji} = \mu_j(X_{Oi}, X_{Ui}, \nu_i) \text{ for } j = 0, 1 \qquad (2)$$

where X_{Oi} is a vector of observed characteristics, X_{Ui} is a vector of unobserved characteristics that are assumed to be correlated with the treatment decision, and ν_i is captures all other unobserved random variables. We assume $(X_O, X_U) \coprod \nu$ and $X_O \coprod X_U$ where $\coprod$ denotes statistical independence.

Assume the following latent model characterizes the treatment decision:

$$D_i^* = \mu_D(X_{Oi}, Z_i) - U_{Di} \qquad (3)$$

where $D_i = 1$ if $D_i^* \geq 0$, $D_i = 0$ if $D_i^* < 0$, μ_D is an unknown function, Z_i is a vector of observed characteristics that affect the treatment decision but do not affect the outcomes (i.e., they are the instruments), and U_{Di} captures unobserved characteristics that affect the treatment decision. As in Basu et al. (2014), we assume $U_D \coprod v$.

To use the notation used in related work (Heckman and Vytlacil, 1999; Basu et al., 2014), we denote the propensity score $Pr(D_i = 1|X_{Oi} = x_{Oi}, Z_i = z_i) = F_{U_D}\big(\mu_D(x_{Oi}, z_i)\big)$ as $P(x_{Oi}, z_i)$, where F_{U_D} is the cumulative distribution function for the random variable U_D. Denote the probability transform of U_{Di} by V_i so that $V_i = F_{U_D}(U_{Di})$. Therefore, we can write $D_i = 1$ if $P(x_{Oi}, z_i) \geq V_i$ and $D_i = 0$ if $P(x_{Oi}, z_i) < V_i$.

Using the above notation, the MTE is equal to $E_v(Y_1 - Y_0|X_O = x_O, V = P(x_O, z))$, which we denote by $MTE(x_O, v)$ where $v = P(x_O, z)$. Heckman and Vytlacil (1999) define the LIV as

$$\frac{\partial E_v\big(Y\big|X_O = x_O, P(X_O, Z) = P(x_O, z)\big)}{\partial P(x_O, z)}$$

which we denote by $LIV(x_O, v)$. Heckman and Vytlacil (1999) demonstrate $LIV(x_O, v) = MTE(x_O, v)$ so that LIV can be used to identify the MTE.

Basu (2014) uses the MTE to identify a new treatment effect parameter, the PeT effect:

$$PeT(x_O, z|D = 1) = \frac{1}{P(x_O, z)} \int_0^{P(x_O, z)} MTE(x_O, v)dv \qquad (4)$$

$$PeT(x_O, z|D = 0) = \frac{1}{1 - P(x_O, z)} \int_{P(x_O, z)}^1 MTE(x_O, v)dv \qquad (5)$$

For individuals receiving the treatment, the PeT effect is the TT conditional on x_O and z. For untreated individuals, the PeT effect is the TUT conditional on x_O and z. Basu (2014) shows

that PeT effects can explain more individual-level variability in treatment effects than CATEs. This is because PeT effects use more information that CATEs. PeT effects incorporate information about the decision that was made (D) and the circumstances under which the decision was made (Z).

PeT effects can be averaged to obtained common treatment effect parameters. Averaging all the PeT effects yields the ATE, averaging the PeT effects for all treated individuals yields the TT, and averaging the PeT effects for all untreated individuals yields the TUT.

B. Estimation Technique

As in Basu et al. (2014), I estimate the PeT effects in a two-stage process. In the first stage, I regress an indicator for receipt of an older drug on the IV and control variables using a probit model. The estimates from this regression are used to determine the fitted values. These fitted values are the propensity scores: the predicted probability of each patient's likelihood of receiving an older index AAD.

After obtaining the propensity scores, the MTEs are estimated using a control-function approach. The control function uses the propensity scores and controls to approximate the observed and unobserved components of the outcomes equation. The control function is represented by a generalized linear model with ¼-power link and negative binomial variance and is estimated using iterated, reweighted least squares (IRLS). Several goodness-of-fit tests were used to refine the specification of the model. After obtaining estimates of the control function, the first derivative of this function with respect to the propensity score was taken. This gives an estimate of $MTE(x_O, v)$.

For each patient, the MTEs are averaged over the appropriate region based on the patient's values of x_O, z, and d (see (4) and (5)). This process is carried out using numerical integration and provides an estimate of the PeT effect for each patient.

C. Match Rate

For each observation, the PeT effect provides information about how index AAD choice affects the expected number of schizophrenia-related hospitalizations. A negative PeT effect implies that the patient's schizophrenia-related hospitalizations would be reduced by taking an older index AAD rather than a newer index AAD. A positive PeT effect implies that the patient's schizophrenia-related hospitalizations would be increased by taking an older index AAD rather than a newer index AAD. Since we have information about the actual index AAD for each patient, we can check if each patient took the index AAD predicted by the model to reduce schizophrenia-related hospitalizations. I create an indicator, m_i, that equals one when a patient took the index AAD predicted by the model to reduce schizophrenia-related hospitalizations and that equals zero when the patient took the index AAD predicted by the model to increase schizophrenia-related hospitalizations.

I break the data into 10 quarters, based on the index date for each patient (which falls between January 1, 2003 and June 30, 2005). For each quarter, I calculate the average value of m_i for each hospital referral region (HRR), which I denote by mr_{qh}. This provides the estimated proportion of patients who were given the appropriate index AAD (defined as the index AAD that would result in fewer schizophrenia-related hospitalizations for the patient) in each HRR during each quarter. Only HRRs with at least 100 patients were included in the analysis so that the denominator of mr_{qh} is not too small.

D. Treatment Effect Parameters

Using the PeT effects, I calculate the TT and TUT for each HRR each quarter. Large negative values of the TT suggest that physicians are matching patients well. Similarly, large positive values of the TUT suggest that the physicians are matching patients well.

E. Variation across Hospital Referral Regions

To understand how physicians' ability to correctly match patients varies across the country, I consider several different measures. For each quarter, I determine the 10th percentile and the 90th percentile of the average HRR match rates, mr_{qh}. I take the ratio, denoted $ratio\_mr_q$, of the 90th percentile to the 10th percentile. A large ratio indicates that there is a significant regional disparity in physicians' ability to correctly match patients to the appropriate index AAD. The trend in $ratio\_mr_q$ provides information on whether this disparity is increasing or decreasing over time.

To further understand variation in the quarterly match rate, I also consider the quarterly standard deviation in HRR match rates, denoted SD_mr_q. Larger values of SD_mr_q indicate wider geographic dispersion in the ability of physicians to correctly match patients to the appropriate index AAD. By considering the trend in SD_mr_q over time, we can study how this dispersion varies over time.

The TT and TUT are alternative measures of the how well physicians are matching patients to the appropriate therapy. They provide information on the magnitude of hospitalizations being prevented. For each quarter, I determine the 10th percentile and 90th percentile of the HRR-specific TTs and TUTs. I also calculate the quarterly standard deviation in

HRR-specific TTs and TUTs, denoted SD_TT_q and SD_TUT_q, which provide other metrics to study dispersion in matching.

To consider the how well the instrumental variable is working, I also consider the dispersion in two additional measures. I define $prop\_neg_{qh}$ as the proportion of patients in a given quarter and HRR with negative PeT effects. This measure represents the proportion of patients for whom the index AAD predicted by the model to reduce schizophrenia-related hospitalizations is an older AAD. The quarterly standard deviation in this measure, denoted by $SD\_prop\_neg_q$, would be expected to be smaller than SD_mr_q and stable over time, if the instrumental variable is valid. Additionally, I define ATE_{qh} to be the average treatment effect in a given quarter and HRR. I denote the quarterly standard deviation in this measure by SD_ATE_q. If the instrument is valid, we would expect SD_ATE_q to be smaller than SD_TT_q and stable over time.

IV. Results

In the 21 states included in the analysis, 22,208 patients were initiated on an older AAD and 15,716 patients were initiated on a newer AAD between January 1, 2003 and June 30, 2005. There are several differences in patient-level characteristics across the two groups of patients (Table 1). Patients initiated on an older AAD were more likely to be male and to have had a schizophrenia related-hospitalization in the year prior to their index date. Patients initiated on a newer AAD were more likely to have taken an AAD in the year prior to the index date, to have had a psychiatric office visit, and to have had a non-psychiatric office visit. More comorbidities in the year prior to the index date were found among patients initiated on a newer AAD. The average number of schizophrenia related-hospitalizations in the year following the index date is not significantly different across the two groups (p-value = 0.18).

In the first-stage probit regression, the IV was found to be significant (p-value < 0.001). An IV only propensity-score model was fitted to the data, and patients were grouped dichotomously: above and below the median of the IV-only propensity score. Patient characteristics were well-balanced across the two groups.

A. Geographic Variation in Mate Rate

The ratio $ratio\_mr_q$ falls between 1.5 and 2.1 for all 10 quarters (Table 2; Figure 1). This indicates that there is significant regional disparity in the ability of physicians to match patients to the appropriate index AAD. While the ratio remains high for all ten quarters, there is not a strong trend in either direction (Figure 1). By considering the 10th and 90th percentiles, we see that there is a downward trend in both over time. This is likely due to the large increase in the percentage of newer AAD prescriptions during this time period (see Robertson (2014)). The

Table 2: Match Rate Variation by HRR

	Match Rate (%) Variation by HRR			
Index Date	10th Percentile	90th Percentile	Ratio (90th/10th)	Standard Deviation
2003Q1	46.4	81.0	1.74	0.144
2003Q2	41.4	75.0	1.81	0.128
2003Q3	41.7	75.0	1.80	0.133
2003Q4	40.8	74.5	1.82	0.156
2004Q1	40.9	73.3	1.79	0.126
2004Q2	40.2	63.7	1.59	0.121
2004Q3	34.5	63.3	1.84	0.126
2004Q4	33.3	64.7	1.94	0.135
2005Q1	33.3	66.7	2.00	0.154
2005Q2	30.8	62.5	2.03	0.113

90th percentile always remains above 50%, while the 10th percentile always remains below 50%. The standard deviation SD_mr_q tells a similar story (Table 2; Figure 2). There is not a

Figure 1: Match Rate Variation

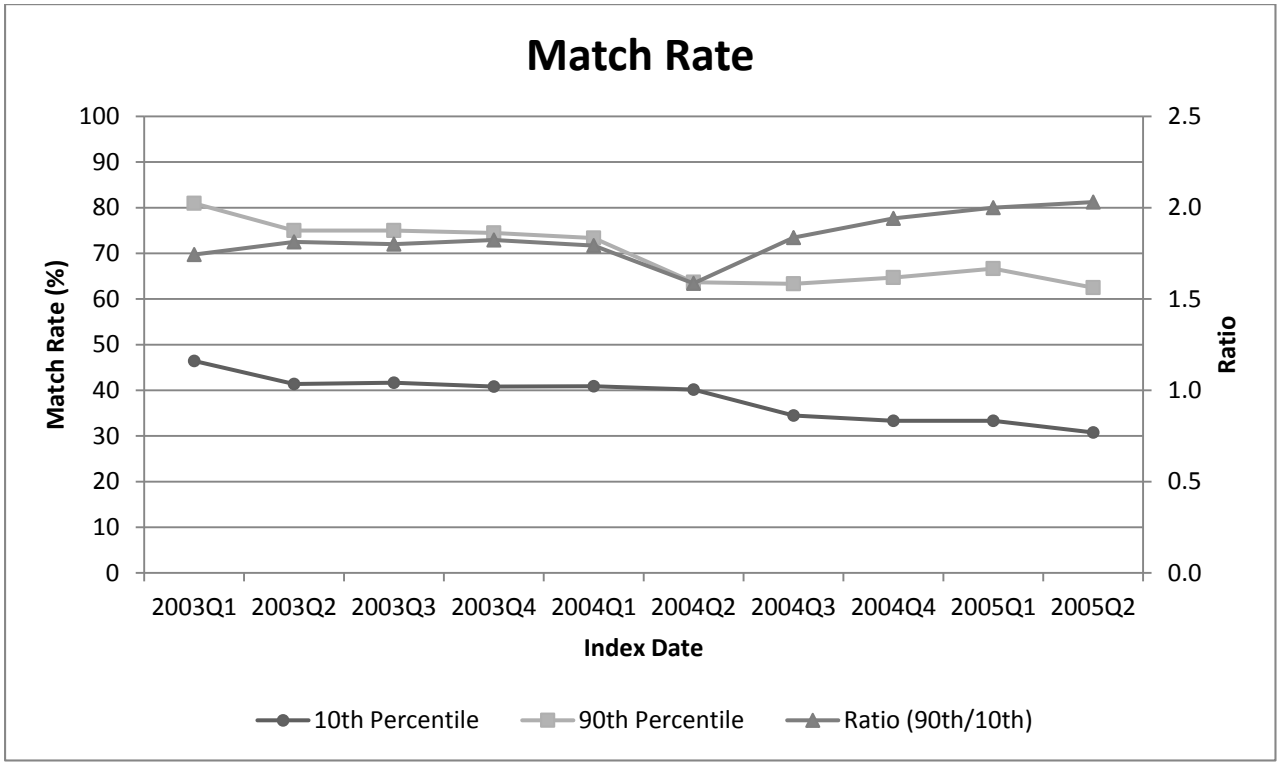
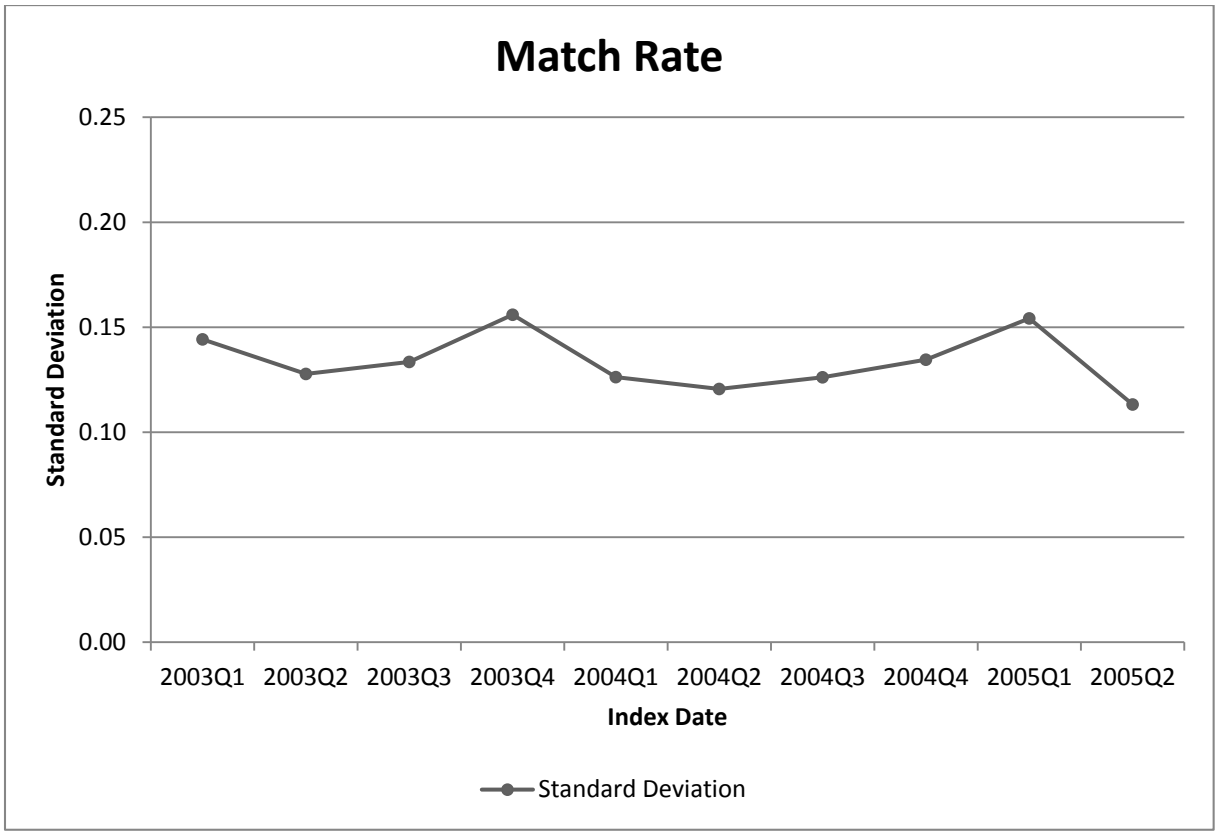


Figure 2: Standard Deviation in HRR-Specific Match Rate



strong trend in either direction over time. Additionally, $SD\_prop\_neg_q$ is less than SD_mr_q and remains stable over time (ranging from 0.07 and 0.09). This gives some evidence in favor of the validity of the instrumental variable. To further investigate regional variation in physicians' ability to match patients to the appropriate index AAD, we now look at treatment effect parameters.

B. Geographic Variation in the Effect of the Treatment on the Treated

For the TT, the 10th percentile is much larger in absolute value than the 90th percentile for all 10 quarters (Table 3; Figure 3). This indicates that among patients prescribed older AADs, there were very large regional differences in the response to treatment. We see that the 90th percentile is relatively stable over time, but the 10th percentile is decreasing over time.

Table 3: TT Variation by HRR

	TT Variation by HRR		
Index Date	10th Percentile	90th Percentile	Standard Deviation
2003Q1	-2.02	-0.31	0.703
2003Q2	-1.88	-0.31	0.625
2003Q3	-2.23	-0.33	0.719
2003Q4	-2.12	-0.37	0.718
2004Q1	-2.15	-0.42	0.783
2004Q2	-2.17	-0.43	0.812
2004Q3	-2.17	-0.41	0.779
2004Q4	-2.74	-0.33	0.914
2005Q1	-2.47	-0.39	0.897
2005Q2	-2.45	-0.35	0.966

This suggests that the HRRs that are match patients poorly continue to do so, whereas the HRRs that are matching patients well continue to improve. The magnitudes of the standard

Figure 3: Effect of the Treatment on the Treated Variation

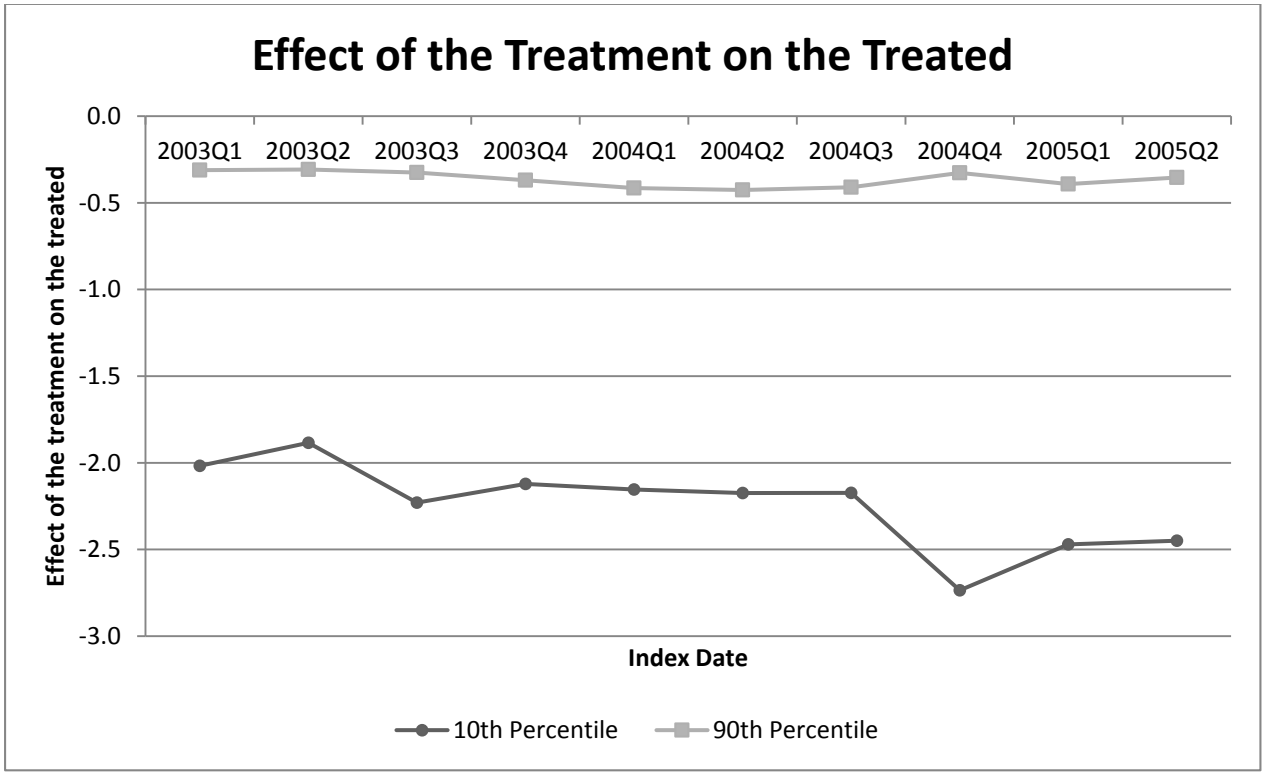
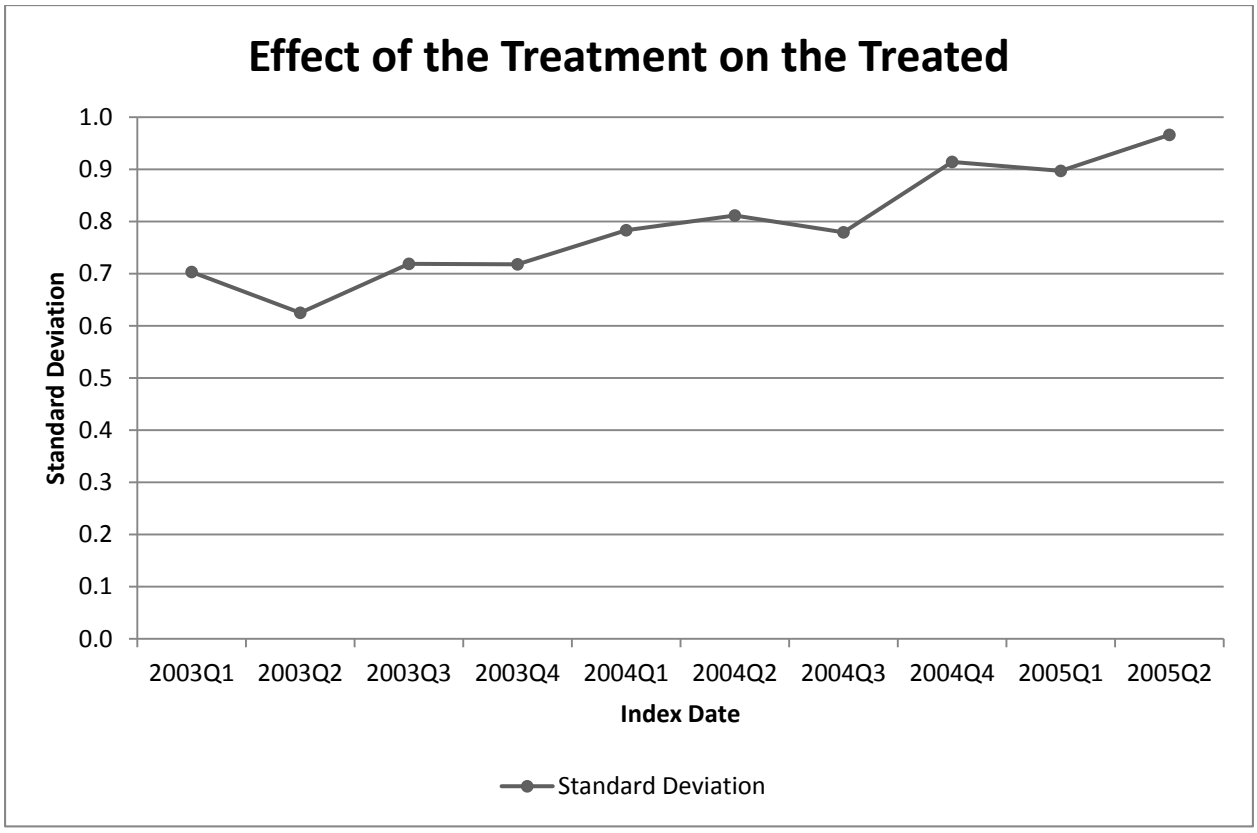


Figure 4: Standard Deviation in HRR-Specific Effect of the Treatment on the Treated



deviations SD_TT_q fall between 0.6 and 1.0 (Table 3). Additionally, there is an upward trend in SD_TT_q, indicating that the dispersion is growing over time (Figure 4). This may reflect the growth in patients taking newer AADs over time. In a related paper, I have found evidence that these patients have treatment effects that are small, but negative (Robertson, 2014). The ATE_q is less than TT_q and is relatively stable over time (ranging from 0.4 to 0.6). This gives some evidence in favor of the validity of the instrumental variable.

C. Geographic Variation in the Effect of the Treatment on the Untreated

For the TUT, the 10th percentile is larger in absolute value than the 90th percentile for all 10 quarters (Table 4; Figure 5). This suggests that among patients prescribed newer AADs, there were sizable regional disparities in response to these therapies. We see that the 90th

Table 4: TUT Variation by HRR

	TUT Variation by HRR		
Index Date	10th Percentile	90th Percentile	Standard Deviation
2003Q1	-0.59	-0.12	0.225
2003Q2	-0.61	-0.08	0.233
2003Q3	-0.79	-0.11	0.312
2003Q4	-0.84	-0.12	0.262
2004Q1	-0.71	-0.10	0.251
2004Q2	-0.63	-0.12	0.201
2004Q3	-0.64	-0.12	0.244
2004Q4	-0.82	-0.13	0.253
2005Q1	-0.68	-0.14	0.222
2005Q2	-0.70	-0.16	0.218

percentile remains quite stable over time. This suggests that HRRs that are doing the best at matching patients do not see much change over time. However, the 10th percentile has a slight

Figure 5: Effect of the Treatment on the Untreated Variation

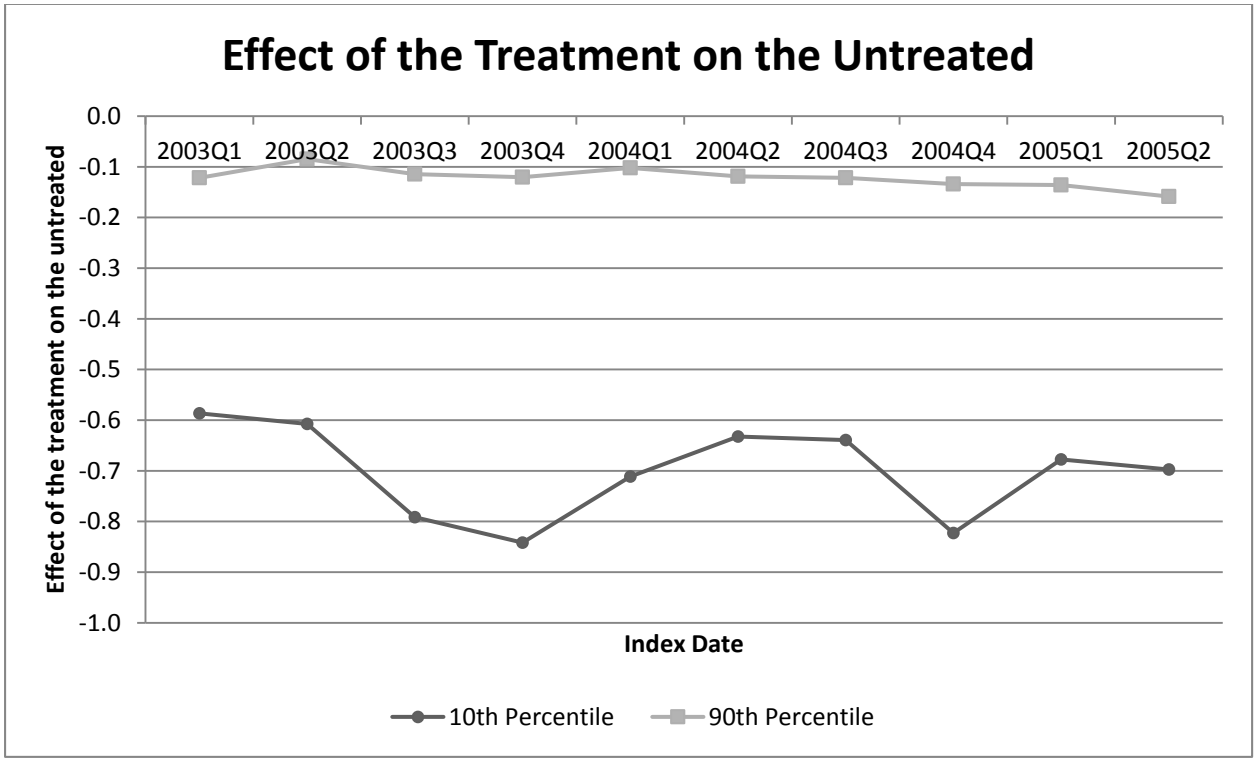
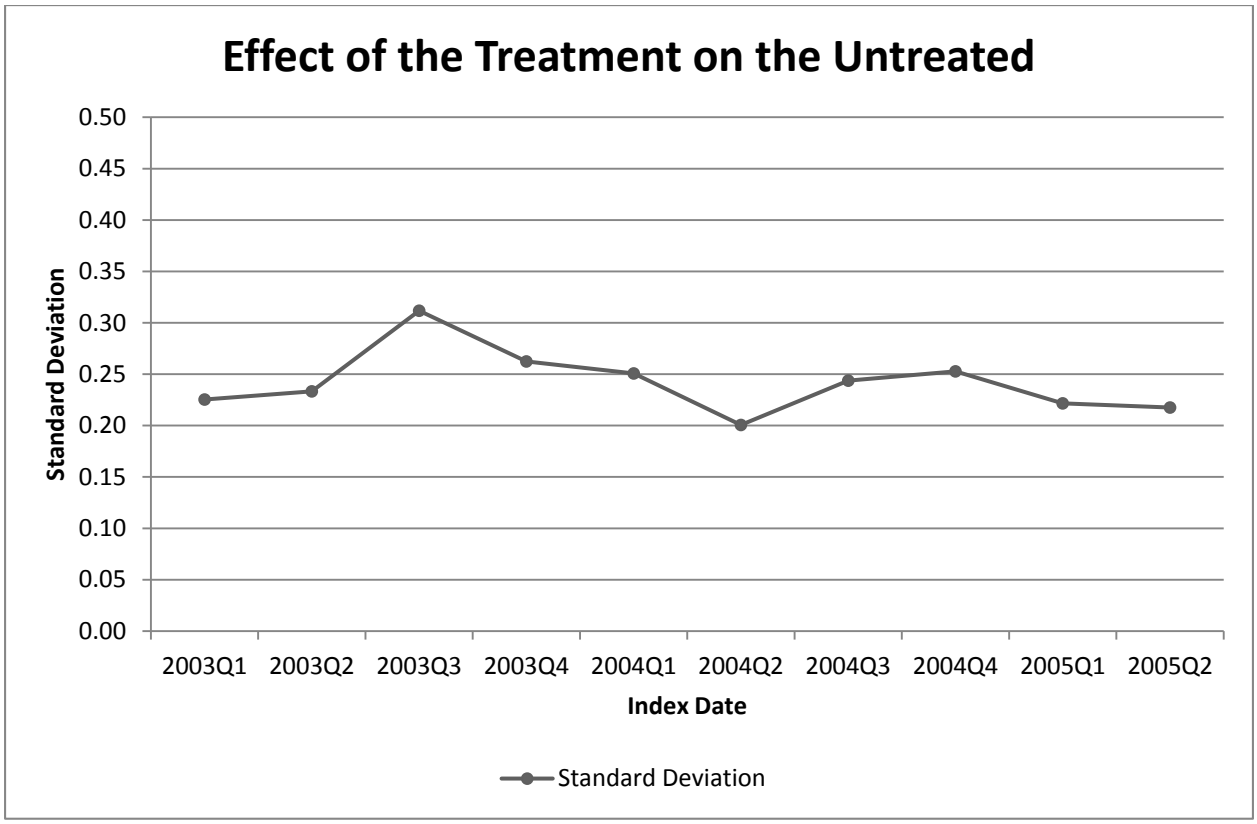


Figure 6: Standard Deviation in HRR-Specific Effect of the Treatment on the Untreated



downward trend. This indicates that HRRs that are doing poorly at matching patient are worsening over time. It is interesting that HRRs that are doing the best at matching patients have a more stable pattern over time. The magnitude of the standard deviations SD_TUT_q are smaller than the standard deviations SD_TT_q (Table 3; Table 4). The standard deviations SD_TUT_q are relatively stable over time, indicating that the dispersion is roughly constant over time (Figure 6).

V. Conclusions

This paper uses a novel econometric technique to study regional variation in the quality of care among Medicaid patients with schizophrenia. I find that there is large variation in the match rate for index AAD in regions across the country. Furthermore, this match rate remains stable over the two-and-a-half year time period considered in this study. To explore variation in other measures of quality of care, I studied HRR-specific effects of the treatment on the treated and the untreated. I found variations in both of these measures across regions. The variation in HRR-specific TT had an upward trend over time, while variation in the HRR-specific TUT was relatively stable over time.

These results indicate that quality of care varies greatly across the country. It would be useful to conduct further studies that look for characteristics that are typical of higher quality regions. This could provide insight into why some regions have physicians who are better able to match patients to the appropriate therapy.

This paper demonstrated how PeT effects can be used to define a new measure of quality of care: the match rate. This technique can be applied to many other disease areas where there is uncertainty over treatment choice, such as cancer, rheumatoid arthritis, and diabetes. A better understanding of variation in quality of care can be used to inform policies that can improve quality in lower performing areas.

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Chapter 3: Optimal Insurance Contracts with Patients and Payment Contracts with Medical Providers when Threat of Waiving Copayments Exists

I. Introduction

In this paper, I consider the relationship between a medical provider, a patient, and an insurance company. In the standard health insurance model, first-best insurance is not achievable (Pauly, 1968; Zeckhauser 1970). A typical (non-health) insurance contract specifies the payment to the insured in the event that the individual experiences a loss. The probability of event (assumed to be fixed) and the magnitude of the loss are known ex ante. The individual pays the actuarially fair price for insurance and is paid a known amount for the loss in the case that the event occurs. However, with health insurance, the probability of the event (illness) and the magnitude of the loss (the cost of total health care sought) are endogenous. The former situation often leads to what is known as ex ante moral hazard and the latter situation often leads to ex post moral hazard (Zweifel and Manning, 2000). In this paper, we only consider ex post moral hazard. (This approach has been taken in elsewhere (e.g., Ma and McGuire, 1997) and isolates the problem that we are studying.)

Ex post moral hazard arises due to the fact that treatment quantity is not contractible ex ante. Illness severity is unknown to the insurer, and the insurer allows the patient to choose treatment quantity. If the patient faces zero out-of-pocket costs, they will consume excessive health care (they will consume treatment past the point where marginal benefit equal marginal cost). Therefore, the insurance company introduces copayments as an instrument to control the quantity of health care consumed by the patient. Raising the copayment results in a tradeoff between overuse and risk-reduction benefit. As the copayment is raised, the patient

reduces quantity of treatment, which reduces overuse of medical care. However, the increased copayment also reduces the risk-reduction benefit that the patient gets from insurance, which was the reason that the patient chose to purchase insurance in the first place. The second-best contract chooses the copayment that balances this tradeoff.

Ma and McGuire (1997) add a new element to the typical health insurance model. They acknowledge that the provider does not play a passive role in health care delivery. (Footnote: Ma and McGuire (1997) focus exclusively on physicians, but I broaden the definition to any health care provider.) In particular, they introduce provider effort into their model. Effort is observable to the patient, but not to the insurance company. The patient's choice of treatment quantity is chosen after observing provider effort. Additionally, they assume that treatment quantity is not contractible ex post and that the insurance company must rely on reports of treatment quantity from providers. In this setting, Ma and McGuire (1997) find that the possibility of false reporting leads to a truth-telling constraint. When this constraint binds, the outcome becomes third-best. In this third-best regime, effort is suboptimal and treatment quantity is excessive.

In this paper, I generalize Ma and McGuire's (1997) model to allow for the possibility that the provider may waive the copayment. To prevent waiving of copayment, a no-waiving constraint is added to the model. When this constraint binds, the outcome becomes third-best. This third-best outcome is characterized by suboptimal effort and suboptimal treatment quantity.

When I use the same assumption about false reporting used by Ma and McGuire (1997), this model nests their model in the case of provider effort and treatment quantity being

substitutes. However, I find the assumption about false reporting to be a strong assumption (that may be unrealistic). It is unlikely that a provider and patient would agree to submit a fraudulent false report. It is more likely that the provider would be willing to waive copayments. This model shows that a third-best outcomes is possible, even in the absence of false reporting.

II. Model

I set up the model similar to Ma and McGuire (1997), but assume treatment quantity is observable ex post. An insurance company designs an insurance contract with the patient and a payment contract with a health provider. There are five stages to the game. In stage 1, the insurer agrees to an insurance contract with the patient and a payment contract with the provider. The patient pays the insurer the premium R. In stage 2, the patient experiences an illness with probability p. If there is no illness, the game ends. If the patient suffers an illness, then we move to stage 3. In stage 3, the provider chooses effort, ε, and tells the patient if they will be waiving the copayment, β. In stage 4, the patient chooses the treatment quantity, q. In the final stage, stage 5, the insurer observes q, and the terms of the insurance contract and the payment contract are executed. It is assumed that the copayment is collected by the provider rather than the insurer. (Footnote: The assumption could be changed to have the copayment collected by the insurer rather than the provider. The results would still be this same. However, the mechanism of waiving would be a transfer from the provider to the patient (rather than a simple waiving of copayment).)

The patient's treatment quantity is a demand response to the provider's effort. The patient's utility depends on their wealth, the insurance premium, the out-of-pocket costs of treatment, the loss they experience from illness, and the restoration in health from visiting the provider. I assume that the loss due to illness has a monetary equivalent of s and that the provider's productivity in restoring health is represented by the function $F(q, \varepsilon)$, where $F_q > 0$, $F_{qq} < 0$, $F_\varepsilon > 0$, $F_{\varepsilon\varepsilon} < 0$, and $0 \leq F(q, \varepsilon) \leq s$. The patient is risk averse and his expected utility is given by

$$EU_{pa} = pU\big(w - R - \beta q - s + F(q, \varepsilon)\big) + (1 - p)U(w - R), \qquad (1)$$

where w is the patient's wealth.

The provider is assumed to be risk neutral. She experiences two costs from providing care to a patient: a constant marginal cost, c, for each unit of care provided and a convex increasing cost associated with effort. The cost associated with effort is represented by $G(\varepsilon)$, where $G(0) = 0$, $G' > 0$, and $G'' > 0$.

The payment contract between the insurer and provider has two parts. The provider is paid a prospective payment (which can be negative) for each patient, ρ. She also receives a payment $\delta + c$ for each unit of care provided. The parameter δ represents the margin that the provider receives for each unit of treatment, and this parameter can be positive or negative. The provider's utility is given by

$$EU_{pr} = p\big(\rho + (\delta + c)q - cq - G(\varepsilon)\big) = p\big(\rho + \delta q - G(\varepsilon)\big). \qquad (2)$$

I assume that the insurance market is competitive and that the insurer seeks to maximize the patient's expected utility subject to a balanced budget. This gives the following budget constraint

$$R = p(\rho + (\delta + c - \beta)q). \qquad (3)$$

In equilibrium, the insurer will ensure that the provider gets their reservation utility, which is normalized to zero.

Now consider the implications of the ability of the provider to waive the copayment. If the provider waives the copayment, then the patient will pay zero out-of-pocket for each unit of treatment. Since $F_q > 0$ for all q, the patient will consume the maximum feasible quanitity of treatment, $\bar{q}$. It will never be optimal for the insurer to allow waiving. Therefore, the insurer

must design the insurance contract and payment contract to prevent waiving of the copayment. This imposes the following constraint on the payment and insurance parameters:

$$\text{(no-waiving constraint)} \quad \delta \leq \beta. \qquad (4)$$

As long as (4) holds, the provider will not waive the copayment (and set $\beta = 0$), since doing so would result in the provider receiving $\delta + c - \beta \leq c$ for each unit of treatment provided.

III. Provider Effort, Patient Treatment Quantity, and Implementable Effort Levels

In the third-best problem, the objective of the insurer is to choose (R, β, ρ, δ) to maximize the patient's expected utility. We begin by considering the patient's treatment quantity decision in stage 4. The patient maximizes (1) with respect to q. This results in the following first-order condition

$$\beta = F_q(q, \varepsilon). \qquad (5)$$

The left-hand side of equation (5) is the marginal out-of-pocket cost of treatment and the right-hand side is the marginal benefit of treatment.

We now consider the provider's effort decision. The provider cannot directly choose the treatment quantity. However, from (5), the provider can influence the treatment quantity by their choice of effort. Taking the total differential of (5) and solving, we find

$$\frac{dq}{d\varepsilon} = -\frac{F_{q\varepsilon}}{F_{qq}}. \qquad (6)$$

Since $F_{qq} < 0$, the sign of $\frac{dq}{d\varepsilon}$ depends on whether treatment quantity and effort are complements ($F_{q\varepsilon} > 0$) or substitutes ($F_{q\varepsilon} < 0$). When treatment quantity and effort are complements, then $\frac{dq}{d\varepsilon} > 0$ and the provider can increase the quantity of treatment by increasing effort. When treatment quantity and effort are substitutes, then $\frac{dq}{d\varepsilon} < 0$ and the provider can increase the quantity of treatment by decreasing effort.

The provider chooses to maximize her expected utility subject to the constraint given by (5). We know that $0 \leq \beta \leq c$, since the copayment will not be set higher than the cost of a unit of treatment (see appendix for proof). Therefore, in any equilibrium, the provider's choice of

effort and the patient's choice of treatment quantity will be given by the solution to the following Program 1: for $0 \leq \beta \leq c$ and $\delta \leq \beta$, ε and q are chosen to maximize

$$\rho + \delta q - G(\varepsilon) \qquad (7)$$

subject to

$$\beta = F_q(q, \varepsilon).$$

As in Ma and McGuire (1997), I characterize the implementable set: the set of all (q, ε) pairs that can arise as an equilibrium. The implementable set, Γ, is defined as follows

$$\Gamma = \{(q, \varepsilon): there\ exists\ (\beta, \delta), with\ 0 \leq \beta \leq c\ and\ \delta \leq \beta,$$

$$for\ which\ (q, \varepsilon)\ solves\ Program\ 1\ given\ (\beta, \delta)\}.$$

Maximizing (7) subject to the constraint (5) and substituting (6), we see that the derivative of the provider's objective function is given by

$$\delta \frac{dq}{d\varepsilon} - G'(\varepsilon) = -\delta \frac{F_{q\varepsilon}(q, \varepsilon)}{F_{qq}(q, \varepsilon)} - G'(\varepsilon). \qquad (8)$$

When treatment quantity and effort are complements and $\delta \leq 0$ or when treatment quantity and effort are substitutes and $\delta \geq 0$, this derivative is negative. In other words, increasing effort will decrease the objective function of the provider in these two situations. Therefore, when treatment quantity and effort are complements, δ must be set to be greater than zero in order to induce the provider to exert costly effort. Similarly, when treatment quantity and effort are substitutes, δ must be set to be less than zero in order to induce the provider to exert costly effort.

The above argument can be summarized with the following proposition:

Proposition 1: Consider a (q, ε) pair in the implementable set, Γ. Suppose that treatment quantity and effort are complements. Then $\varepsilon > 0$ only if the margin parameter, δ, is greater

than zero. Suppose that treatment quantity and effort are substitutes. Then $\varepsilon > 0$ only if the margin parameter, δ, is less than zero.

By considering Proposition 1, we can see the incentive problem that arises due to the threat of waiving coinsurance. When treatment quantity and effort are substitutes, reducing δ below zero makes each visit costly to the provider, since they receive less than the marginal cost of each visit, c. Therefore, the provider will exert costly effort to reduce the number of visits the patient chooses. As δ is reduced, the provider will continue to increase effort. When treatment quantity and effort are complements, the situation is more complicated. In this case, increasing δ above zero makes each visit profitable to the provider, since they receive more than the marginal cost, c, for each visit. Therefore, the provider will exert costly effort to increase the number of visits the patient chooses. However, the insurer is limited in how high they can set δ by the no-waiving constraint. In particular, δ cannot exceed β. Therefore, high levels of effort cannot be implemented because the constraint $\delta \leq \beta$ binds.

I illustrate the situation for the case of complements. Ma and McGuire (1997) have shown that under the assumption that the necessary first-order conditions for Program 1 are sufficient, the follow is true:

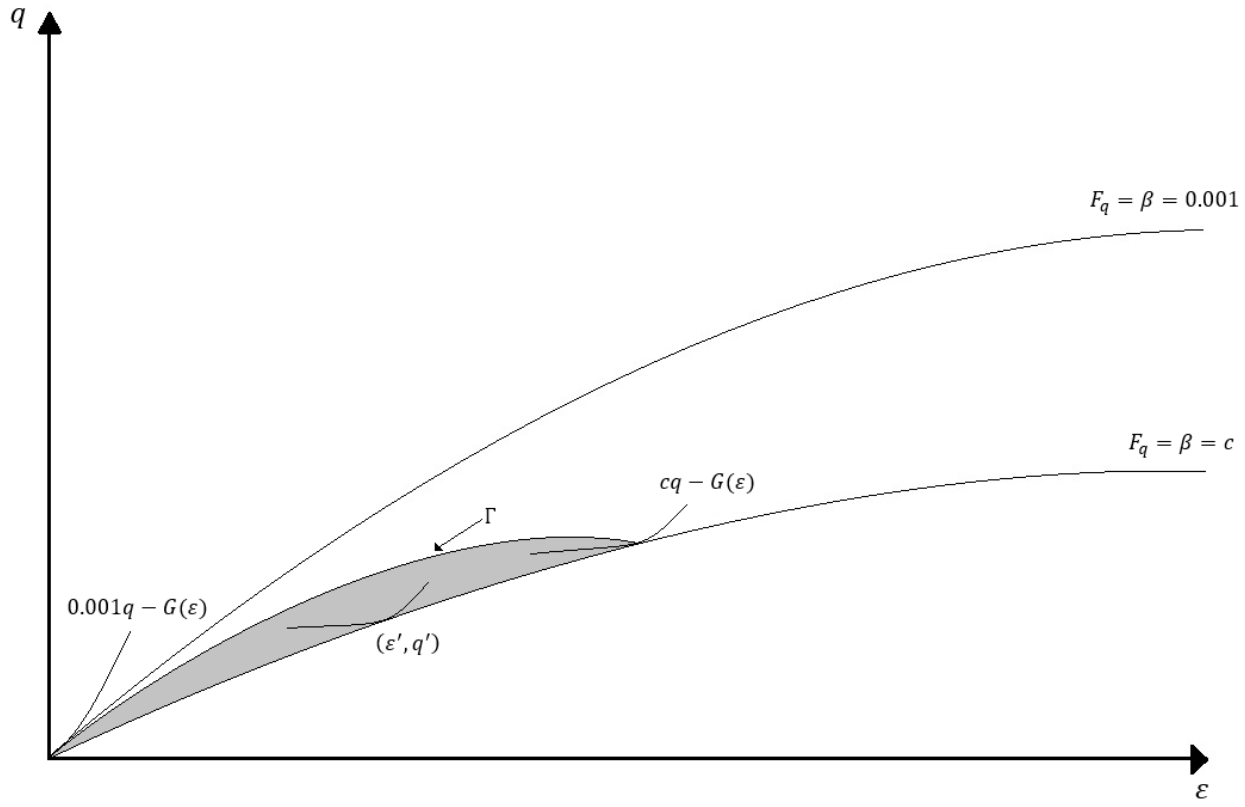
$$\frac{\partial q}{\partial \delta} = -\frac{F_{q\varepsilon}(q,\varepsilon)^2}{H}$$

$$\frac{\partial \varepsilon}{\partial \delta} = \frac{F_{q\varepsilon}(q,\varepsilon)F_{qq}(q,\varepsilon)}{H}, \qquad (9)$$

where $H < 0$ is the bordered Hessian. Thus, in the case of complements, increasing δ will increase the provider's level of effort (since $\frac{\partial \varepsilon}{\partial \delta} > 0$). Consider Figure 1, which shows the implementable set. The patient's positively sloped best-response functions are shown for two

different copayment levels. The provider's indifference curves are convex and have slopes of $G'(\varepsilon)/\delta$. I show three indifference curves for different values of δ and ρ. Equilibria are tangencies between the patient's best-response curves and the provider's indifference curves (see (ε', q') for an example). The shaded region is the implementable set. The boundary of this set is determined by the no-waiving constraint: $\delta \leq \beta$. As can be seen from the figure, the constraint becomes more restrictive as the copayment, β, is reduced.

Figure 1: The Set of Implementable Effort and Treatment Quantity



IV. Optimal Contract

In this section, I follow an argument similar to Ma and McGuire (1997) to characterize the optimal insurance contract between the insurer and the patient and payment contract between the insurer and the provider. As stated earlier, the provider will only receive her

reservation utility, since rent can be extracted without distortions with the prospective payment, ρ. Setting the provider's utility equal to zero and solving for ρ, we can substitute this expression into the budget constraint (3) to obtain

$$pcq + pG(\varepsilon) = R + p\beta q. \qquad (10)$$

Therefore, ρ can be ignored when solving for the optimal contract, since it can always be chosen to be $\rho = G(\varepsilon) - \delta q$ (which comes from setting the provider's utility equal to zero). Thus, the insurer solves the following program to obtain the optimal contract

$$\max_{R,\beta,\delta,q,\varepsilon} \quad pU\big(w - R - \beta q - s + F(q,\varepsilon)\big) + (1-p)U(w-R)$$

$$subject\ to \qquad (10)\ and\ (q,\varepsilon) \in \Gamma.$$

The solution to the above problem determines the third-best equilibrium. I denote the parameters that solve this program by $\{R^{TB}, \beta^{TB}, \delta^{*}, q^{TB}, \varepsilon^{TB}\}$ and the corresponding expected utility by EU_{pa}^{TB}.

The third-best equilibrium can be compared to the following second-best regime, which is similar to the case examined by Zeckhauser (1970). In this regime, effort is observable. Therefore, there is no threat of the provider waiving copayment, since doing so would result in extra (costly) visits from the patient and would not change the provider's payment (which is based on effort, not quantity). Furthermore, the margin parameter, δ, can be removed because the insurer can observe effort. In this situation, the insurer solves the following program to obtain the second-best contract:

$$\max_{R,\beta,q,\varepsilon} \quad pU\big(w - R - \beta q - s + F(q,\varepsilon)\big) + (1-p)U(w-R)$$

$$subject\ to \qquad (10)\ and\ (5).$$

The solution to the above problem determines the second-best equilibrium. I denote the parameters that solve this program by $\{R^{SB}, \beta^{SB}, q^{SB}, \varepsilon^{SB}\}$ and the corresponding expected utility by EU_{pa}^{SB}.

Notice that the difference between the two programs is that the third-best program requires $(q, \varepsilon) \in \Gamma$ while the second-best program only requires that (5) hold. Since (5) follows from $(q, \varepsilon) \in \Gamma$, but $(q, \varepsilon) \in \Gamma$ does not follow from (5), we see that the second-best program is a more relaxed problem than the third-best program. I now state the following proposition which relates the third-best equilibrium to the second-best equilibrium:

Proposition 2: Suppose that there exists a δ^* such that $\delta^* \leq \beta^{SB}$ and $(q^{SB}, \varepsilon^{SB}) \in \Gamma$. Then, $\{R^{SB}, \beta^{SB}, \delta^*, q^{SB}, \varepsilon^{SB}\}$ is a solution to the third-best program and $EU_{pa}^{TB} = EU_{pa}^{SB}$. In other words, the second-best solution can be achieved in the third-best regime.

Proof: Assume that there exists a δ^* such that $\delta^* \leq \beta^{SB}$ and $(q^{SB}, \varepsilon^{SB}) \in \Gamma$. Then, q^{SB} and ε^{SB} can be implemented in the third-best regime. Since, $\beta = F_q(q, \varepsilon)$ in the third-best regime, then $\beta^{SB} = F_q(q^{SB}, \varepsilon^{SB})$ can be chosen in the third-best regime. Furthermore, if $\{\beta^{SB}, q^{SB}, \varepsilon^{SB}\}$ is chosen, then, from (10), R will be given by $pcq^{SB} + pG(\varepsilon^{SB}) - p\beta^{SB}q^{SB}$, which is equal to R^{SB}. Thus, $\{R^{SB}, \beta^{SB}, \delta^*, q^{SB}, \varepsilon^{SB}\}$ is a solution to the third-best program. Therefore, the patient's expected utility will be given by $EU_{pa}^{TB} = pU\big(w - R^{SB} - \beta^{SB}q^{SB} - s + F(q^{SB}, \varepsilon^{SB})\big) + (1-p)U(w - R^{SB}) = EU_{pa}^{SB}$.

Proposition 2 says that if effort is unobservable and a δ can be chosen such that the second-best levels of treatment quantity and effort can be implemented, then the program can be solved as though effort is observable. The unobservability of effort does not distort the equilibrium. However, if the δ necessary to implement the second-best levels of treatment

quantity and effort exceeds β^{SB}, so that the no-waiving constraint binds, then the second-best cannot be achieved and the patient is worse off. In the next section, I consider a situation in which the no-waiving constraint binds and the patient's expected utility is below the second-best level.

V. A Third-Best Equilibrium

First, observe that the outcome will always be second best when treatment quantity and effort are substitutes. In this case, the insurance company will always set $\delta \leq \beta$ to get the provider to exert effort (see Proposition 1). Therefore, the no-waiving constraint never binds, and the second-best can always be implemented when effort is unobservable (see Proposition 2).

Therefore, a genuine third-best regime can only exist in the case of complements. I consider complements for the remainder of the paper.

To simplify the expression of the problem, I introduce some new notation. Define m by the solution to the following equation: $m + \beta - c = \delta$. In other words, m is the payment from the insurance company to the provider for each unit of care. This allows a simple decomposition of the provider payment into two parts, m and β. For the remainder of the paper, I exclusively use m and no longer use δ. The no-waiving constraint can now be written as $m + \beta - c \leq \beta$ or simply $m \leq c$. This constraint states that the insurance company cannot directly pay the provider more than their unit cost, c.

The provider's choice of effort and the patient's choice of treatment quantity is given by the solution to Program 1. The first-order conditions from solving this program yields the following result

$$\frac{m + \beta - c}{-G'(\varepsilon)} = \frac{F_{qq}}{F_{q\varepsilon}}. \qquad (11)$$

Solving (11) for m yields the following equation

$$m^*(\varepsilon) = c - F_q(q(\varepsilon), \varepsilon) - \frac{F_{qq}(q(\varepsilon), \varepsilon)}{F_{q\varepsilon}(q(\varepsilon), \varepsilon)} G'(\varepsilon).$$

where $q(\varepsilon)$ is the solution to (5). The expression $m^*(\varepsilon)$ characterizes the m that the insurance company must choose to implement the desired effort. If there are no restrictions on m, the insurance company can implement the second-best effort. By keeping β at its second-best level, the patient will choose the second-best level of q. Therefore, the insurance company can implement the second-best equilibrium when effort is not observable, as long as there are no restrictions on m.

However, m is restricted by the no-waiving constraint: $m \leq c$. To check if there are cases where this constraint binds, I consider an example with specific functional forms for $F(q, \varepsilon)$ and for $G(\varepsilon)$. In particular, I study the case of $F(q, \varepsilon) = d\sqrt{q\varepsilon}$, where $d > 0$, and $G(\varepsilon) = a\varepsilon^2$, where $a > 0$. I now check under what parameter configurations $m^*(\varepsilon^{SB}) < c$ so that the second best can be implemented.

$$m^*(\varepsilon^{SB}) < c$$

$$c - F_q(q(\varepsilon^{SB}), \varepsilon^{SB}) - \frac{F_{qq}(q(\varepsilon^{SB}), \varepsilon^{SB})}{F_{q\varepsilon}(q(\varepsilon^{SB}), \varepsilon^{SB})} G'(\varepsilon^{SB}) < c$$

$$-F_q(q(\varepsilon^{SB}), \varepsilon^{SB}) < \frac{F_{qq}(q(\varepsilon^{SB}), \varepsilon^{SB})}{F_{q\varepsilon}(q(\varepsilon^{SB}), \varepsilon^{SB})} G'(\varepsilon^{SB})$$

$$-\frac{1}{2} d (q^{SB})^{-\frac{1}{2}} (\varepsilon^{SB})^{\frac{1}{2}} < \frac{-\frac{1}{4}(q^{SB})^{-\frac{3}{2}}(\varepsilon^{SB})^{\frac{1}{2}}}{\frac{1}{4}(q^{SB})^{-\frac{1}{2}}(\varepsilon^{SB})^{-\frac{1}{2}}} a\varepsilon^{SB}$$

$$-\frac{1}{2} d < -a(q^{SB})^{-\frac{1}{2}}(\varepsilon^{SB})^{\frac{3}{2}}$$

$$-\frac{1}{2} d < -a\left(\frac{d^4 c}{16a(\beta^{SB})^4}\right)^{-\frac{1}{2}}\left(\frac{d^2 c}{4a(\beta^{SB})^2}\right)^{\frac{3}{2}}$$

$$-\frac{1}{2} d < -\frac{dc}{2\beta^{SB}}$$

$$c < \beta^{SB} \qquad (12)$$

From (12), we see that the second best can be implemented (i.e., $m^*(\varepsilon^{SB}) < c$) whenever $c < \beta^{SB}$. Since β^{SB} is always less than c, we see that, for this example, in the case of treatment quantity and effort being complements, the no-waiving constraint always binds and the outcome is third best (strictly inferior to the case where effort is observable).

VI. First Best, Second Best, and Third Best: Comparing Equilibria

In this section, I compare the equilibria across three different problems. I begin with the first-best problem. In this problem, q and ε are both observable and contractible. The insurance company solves the following program (Footnote: Note that β equals zero in the first best. See appendix for proof.):

$$\max_{q,\varepsilon,R} \quad pU\big(w - R - s + F(q,\varepsilon)\big) + (1-p)U(w-R)$$

$$subject\ to \qquad R = p\big(cq + G(\varepsilon)\big) \qquad (13)$$

From the necessary first-order conditions, I obtain the following condition

$$\frac{F_q(q,\varepsilon)}{F_\varepsilon(q,\varepsilon)} = \frac{c}{G'(\varepsilon)}. \qquad (14)$$

Solving for the full equilibrium requires specifying functions for $F(\cdot,\cdot)$, $G(\cdot)$, and $U(\cdot)$. In the appendix, I solve for the equilibrium using $F(q,\varepsilon) = d\sqrt{q\varepsilon}$, $G(\varepsilon) = a\varepsilon^2$, and $U(x) = \ln(1+x)$.

I next discuss the second-best problem. In this problem, ε is observable and contractible, but q is not contractible ex ante and is chosen by the patient. As discussed earlier in the paper, the patient's choice of q is determined by the solution to (5): $\beta = F_q(q,\varepsilon)$.

Therefore, the insurance company solves the following program, which characterizes the second-best equilibrium:

$$\max_{q,\varepsilon,R} \quad pU\Big(w - R - F_q(q,\varepsilon)q - s + F(q,\varepsilon)\Big) + (1-p)U(w-R)$$

$$subject\ to \qquad p\big(cq + G(\varepsilon)\big) = R + pqF_q(q,\varepsilon) \qquad (15)$$

From the necessary first-order conditions, I obtain the following condition

$$\frac{-F_{qq}(q,\varepsilon)q}{-F_{q\varepsilon}(q,\varepsilon)q + F_{\varepsilon}(q,\varepsilon)} = \frac{F_q(q,\varepsilon) + F_{qq}(q,\varepsilon)q - c}{F_{q\varepsilon}(q,\varepsilon)q - G'(\varepsilon)}. \qquad (16)$$

Solving for the full equilibrium requires specifying functions for $F(\cdot,\cdot)$, $G(\cdot)$, and $U(\cdot)$. In the appendix, I again solve for the equilibrium using $F(q,\varepsilon) = d\sqrt{q\varepsilon}$, $G(\varepsilon) = a\varepsilon^2$, and $U(x) = \ln(1+x)$.

Now, consider the third-best problem. In this problem, q is again noncontractible ex ante and is chosen by the patient, and now ε is noncontractible and chosen by the provider. As demonstrated in the previous section, the no-waiving constraint always binds for the functional forms being used ($F(q,\varepsilon) = d\sqrt{q\varepsilon}$ and $G(\varepsilon) = a\varepsilon^2$). Therefore, I impose $m = c$. In this setting, the provider's maximization problem can be simplified from

$$\max_{\varepsilon} \quad \rho + (m + \beta - c)q(\varepsilon) - G(\varepsilon)$$

to

$$\max_{\varepsilon} \quad \rho + \beta q(\varepsilon) - G(\varepsilon).$$

From the necessary first-order condition, I obtain the following condition

$$\beta = \frac{G'(\varepsilon)}{q'(\varepsilon)}. \qquad (17)$$

Using (5) and (17), I can solve for $q(\beta)$ and $\varepsilon(\beta)$. Therefore, the insurance company solves the following program, which characterizes the third-best equilibrium:

$$\max_{\beta} \quad pU\left(w - pcq(\beta) - pG\big(\varepsilon(\beta)\big) + pq(\beta)\beta - \beta q(\beta) - s + F(q(\beta), \varepsilon(\beta))\right) + (1-p)U\big(w - pcq(\beta) - pG\big(\varepsilon(\beta)\big) + pq(\beta)\beta\big)$$

From the necessary first-order condition, I obtain the following condition

$$\frac{(1-p)U'\big(w - pcq(\beta) - pG\big(\varepsilon(\beta)\big) + pq(\beta)\beta\big)}{-pcq'(\beta) - pG'\big(\varepsilon(\beta)\big)\varepsilon'(\beta) + pq'(\beta)\beta + pq(\beta) - q(\beta) - \beta q'(\beta) + F_q\big(q(\beta), \varepsilon(\beta)\big)q'(\beta) + F_\varepsilon(q(\beta), \varepsilon(\beta))\varepsilon'(\beta)}$$

$$= \frac{pU'\left(w - pcq(\beta) - pG\big(\varepsilon(\beta)\big) + pq(\beta)\beta - \beta q(\beta) - s + F(q(\beta), \varepsilon(\beta))\right)}{pcq'(\beta) + pG'\big(\varepsilon(\beta)\big)\varepsilon'(\beta) - pq'(\beta)\beta - pq(\beta)}. \qquad (18)$$

Solving for the full equilibrium requires specifying functions for $F(\cdot,\cdot)$, $G(\cdot)$, and $U(\cdot)$. In the appendix, I again solve for the equilibrium using $F(q,\varepsilon) = d\sqrt{q\varepsilon}$, $G(\varepsilon) = a\varepsilon^2$, and $U(x) = \ln(1+x)$.

Due to the complexity of the first-order conditions for each of the three problems, closed-form solutions are not obtainable for $\{q^{FB}, \varepsilon^{FB}, EU^{FB}\}$, $\{q^{SB}, \varepsilon^{SB}, EU^{SB}\}$, and $\{q^{TB}, \varepsilon^{TB}, EU^{TB}\}$. However, these solutions can be determine using simulations (solving for the solutions numerically using mathematical software). In the appendix, I show how the three sets of equilibria differ when specific parameters are chosen. Here, I discuss the general relationship between the equilibria (across a wide variety of parameter values). In particular, the relationship between treatment quantity across equilibria is characterized by

$$q^{FB} > q^{SB} > q^{TB}. \qquad (19)$$

The relationship between effort across equilibria is characterized by

$$\varepsilon^{FB} > \varepsilon^{SB} > \varepsilon^{TB}. \qquad (20)$$

The relationship between patient expected utility across equilibria is characterized by

$$EU^{FB} > EU^{SB} > EU^{TB}. \qquad (21)$$

Lastly, the second-best copayment level is greater than the third-best copayment level: $\beta^{SB} > \beta^{TB}$.

It follows that the threat of waiving copayment results in a loss of welfare to the patient by reducing the effort that the physician exerts and, therefore, the marginal productivity of health restoration, $F_q(q, \varepsilon)$ (since $F_{q\varepsilon} > 0$).

I now briefly discuss the difference between the second-best equilibrium and the third-best equilibrium. Suppose that we are in the third-best world. The second-best (q, ε) pair is no longer implementable due to the threat of waiving copayment (see Section V). To see why the third-best treatment quantity and effort levels are below the second-best levels, consider the following. Once the no-waiving constraint ($m = c$) is imposed, the provider can no longer be costlessly give an incentive for effort by increasing the payment level, m, and extracting the rent through ρ. The only way to give the provider incentive for effort is to increase the copayment, β, which comprises the provider's margin. However, raising β is not costless, since doing so reduces the risk-sharing benefit that the patient receives from insurance. By considering (A8) and (A7), we see that $q'(\beta) < 0$ and $\varepsilon'(\beta) < 0$. The downward sloping $q(\beta)$ function follows from the fact that lowering the patient's out-of-pocket cost per unit of care results in an increase in the treatment quantity he chooses. The downward sloping $\varepsilon(\beta)$ function indicates that an increase in the copayment level reduces the provider's effort. This implies that the benefit of an increased margin to the provider (represented by an increase in β) is outweighed by the loss of visits that the patient will choose as a result of having a higher out-of-pocket cost. Since effort is more difficult to implement in the third-best regime (compared to the second-best regime), then $\varepsilon(\beta^{SB}) < \varepsilon^{SB}$ (where $\varepsilon(\beta^{SB})$ represents effort in the third-best regime if β were set at its second-best level). Therefore, in the third-best regime, the insurer reduces β below β^{SB} to increase effort and treatment quantity. Once β is reduced

to β^{TB}, further reduction in β makes the patient worse off because q would become too high for the current level of effort.

VII. Conclusions

In this paper, I have considered how the threat of waiving copayments affects the optimal insurance contract between the insurer and the patient and payment contract between the insurer and health provider. When treatment quantity and effort are substitutes, the threat of waiving copayments does not affect the optimal contract. In this case, despite the unobservability of effort, the second-best equilibrium can be achieved. However, when treatment quantity and effort are complements, the threat of waiving copayments becomes an important consideration. I have shown that the no-waiving constraint may bind, which results in a third-best equilibrium. The third-best outcome is characterized by suboptimal treatment quantity, suboptimal effort, and the patient being worse off.

This paper extends the work carried out by Ma and McGuire (1997). These authors demonstrated that a third-best outcome was possible in the case of treatment quantity and effort being substitutes when false reporting is a concern. However, in the case of treatment quantity and effort being complements, Ma and McGuire (1997) show that a second-best outcome can be achieved, even when false reporting is a concern. In the above, I have demonstrated that when treatment quantity and effort are complements, the outcome can become third-best, due to the threat of waiving copayments, even when false reporting is not a concern.

This paper considers how the design of insurance contracts can lead to perverse incentives, which ultimately may make individuals who purchase insurance worse off than they otherwise would be. New types of complex insurance policies are always appearing in society.

Exploring unintended incentives that may arise in these contracts could be useful for gaining a better understanding about the welfare implications of such contracts.

Appendix

Second-Best β

In the second best, β is less than or equal to c.

Proof: In the second-best setting, the insurance company solves the following program:

$$\max_{q,\varepsilon,R,\beta} \quad pU\big(w - R - \beta q - s + F(q,\varepsilon)\big) + (1-p)U(w-R)$$

$$subject\ to \qquad pcq + pG(\varepsilon) = R + pq\beta \qquad (1^*)$$

$$\beta = F_q(q,\varepsilon) \qquad (2^*)$$

The Lagrangian for this problem is

$$L = pU(D_1) + (1-p)U(D_2) + \lambda_1\big(R + pq(\beta - c) - pG(\varepsilon)\big) + \lambda_2\left(\beta - F_q(q,\varepsilon)\right),$$

where $D_1 = w - R - \beta q - s + F(q,\varepsilon)$ and $D_2 = w - R$. The necessary first-order conditions are

$$\frac{\partial L}{\partial q} = pU'(D_1)\left(-\beta + F_q(q,\varepsilon)\right) + \lambda_1\big(p(\beta - c)\big) - \lambda_2 F_{qq}(q,\varepsilon) = 0 \qquad (3^*)$$

$$\frac{\partial L}{\partial \varepsilon} = pU'(D_1)F_\varepsilon(q,\varepsilon) - \lambda_1 pG'(\varepsilon) - \lambda_2 F_{q\varepsilon}(q,\varepsilon) = 0 \qquad (4^*)$$

$$\frac{\partial L}{\partial \beta} = -pU'(D_1)q + \lambda_1 pq + \lambda_2 = 0 \qquad (5^*)$$

$$\frac{\partial L}{\partial R} = -pU'(D_1) - (1-p)U'(D_2) + \lambda_1 = 0. \qquad (6^*)$$

From (2*) and (3*), we get

$$\lambda_1 p(\beta - c) = \lambda_2 F_{qq}(q,\varepsilon)$$

$$\beta - c = \frac{\lambda_2}{\lambda_1}\frac{F_{qq}(q,\varepsilon)}{p}. \qquad (7^*)$$

From (6*), we get

$$\lambda_1 = pU'(D_1) + (1-p)U'(D_2) > 0. \qquad (8^*)$$

We can rewrite (5*) as

$$\lambda_2 = pU'(D_1)q - \lambda_1 pq = pq(U'(D_1) - \lambda_1). \qquad (9^*)$$

Notice that

$$U'(D_1) \geq U'(D_2) > 0, \qquad (10^*)$$

since $U(\cdot)$ is concave and $D_1 \leq D_2$ (because health cannot be fully restored (i.e., $F(q, \varepsilon) \leq s$)).

Using $\lambda_1 = pU'(D_1) + (1-p)U'(D_2)$ (from (8*)) and (10*), we see

$$\lambda_1 \leq U'(D_1). \qquad (11^*)$$

Using (9*) and (11*), we get

$$\lambda_2 = pq(U'(D_1) - \lambda_1) \geq 0. \qquad (12^*)$$

Since $\lambda_1 > 0$ and $\lambda_2 \geq 0$, from (7*), we get

$$\beta - c = \frac{\lambda_2}{\lambda_1}\frac{F_{qq}(q, \varepsilon)}{p} \leq 0$$

$$\beta \leq c,$$

as desired.

First-Best β

In the first best, β will be set equal to zero.

Proof: In the first-best setting, the insurance company solves the same program as in the second-best setting, except that the constraint (2*) is eliminated, since q is contractible. We denote the other constraint, (1*), by (1**).

The Lagrangian for this problem is the same as for the second-best setting, except that $\lambda_2 = 0$, since (2*) has been eliminated. The necessary first-order conditions are

$$\frac{\partial L}{\partial q} = pU'(D_1)\Big(-\beta + F_q(q, \varepsilon)\Big) + \lambda_1 p(\beta - c) = 0 \qquad (2^{**})$$

$$\frac{\partial L}{\partial \varepsilon} = pU'(D_1)F_\varepsilon(q,\varepsilon) - \lambda_1 pG'(\varepsilon) = 0 \qquad (3^{**})$$

$$\frac{\partial L}{\partial \beta} = -pU'(D_1)q + \lambda_1 pq = 0 \qquad (4^{**})$$

$$\frac{\partial L}{\partial R} = -pU'(D_1) - (1-p)U'(D_2) + \lambda_1 = 0. \qquad (5^{**})$$

From (4**), we get

$$\lambda_1 = \frac{U'(D_1)q}{q} = U'(D_1). \qquad (6^{**})$$

Substituting (6**) into (5**), we get

$$-U'(D_1) - (1-p)U'(D_2) + U'(D_1) = 0$$

$$(1-p)U'(D_1) - (1-p)U'(D_2) = 0$$

$$U'(D_1) = U'(D_2)$$

$$D_1 = D_2$$

$$w - R - \beta q - s + F(q,\varepsilon) = w - R$$

$$\beta = \frac{F(q,\varepsilon) - s}{q}. \qquad (7^{**})$$

Since health cannot be fully restored (i.e., $F(q,\varepsilon) \leq s$), then, from (7**), we get

$$\beta \leq 0. \qquad (8^{**})$$

(Note: For any finite q and ε, $F(q,\varepsilon) < s$. Therefore, (8**) will become strict.) Since the copayment is nonnegative ($\beta \geq 0$), then we get a corner solution and must impose $\beta = 0$.

Note: This results in the patient being unable to be fully insured.

First-Best Problem

Using the specified functions, (14) becomes

$$qc = a\varepsilon^2. \qquad (A1)$$

Substituting (A1) into the constraint (13), we obtain

$$2R = 3pcq. \qquad (A2)$$

Using the remaining first-order condition, substituting (A1) and (A2), and simplifying, we obtain the following expression

$$\frac{1}{2}d\left(\frac{1}{c}\right)^{\frac{1}{2}}\left(\frac{1}{a}\right)^{\frac{1}{4}}(cq)^{-\frac{1}{4}}\left(1+w-\frac{3}{2}pcq\right)$$

$$= p\left(1+w-\frac{3}{2}pcq\right)+(1-p)(1+w-\frac{3}{2}pcq-s$$

$$+ d\left(\frac{1}{c}\right)^{\frac{1}{2}}\left(\frac{1}{a}\right)^{\frac{1}{4}}(cq)^{\frac{3}{4}}. \qquad (A3)$$

The expression (A3) can be solved for q to obtain q^{FB}. Then, q^{FB} can be substituted into (A1) to obtain ε^{FB}. The expressions for q^{FB} and ε^{FB} can be substituted into the patient's expected utility function to obtain EU^{FB}, the patient's first-best expected utility. Expression (A3) cannot be solved to obtain a closed form solution for q. Therefore, I solve for q^{FB}, ε^{FB}, and EU^{FB} using simulations. I return to this later.

Second-Best Problem

Using the specified functions, (16) becomes

$$qc = a\varepsilon^2. \qquad (A1)$$

Substituting (A1) into the constraint (15), we obtain

$$\frac{3}{2}pcq = R + \frac{1}{2}pda^{-\frac{1}{4}}c^{\frac{1}{4}}q^{\frac{3}{4}}. \qquad (A4)$$

Using the remaining first-order condition, substituting (A1) and (A4), and simplifying, we obtain the following expression

$$\frac{\left(da^{-\frac{1}{4}}c^{\frac{1}{4}}q^{-\frac{1}{4}}\right)\left(1+w-\frac{3}{2}pcq+\frac{1}{2}pda^{-\frac{1}{4}}c^{\frac{1}{4}}q^{\frac{3}{4}}\right)}{da^{-\frac{1}{4}}c^{\frac{1}{4}}q^{-\frac{1}{4}}-4c}$$

$$= p\left(1+w-\frac{3}{2}pcq+\frac{1}{2}pda^{-\frac{1}{4}}c^{\frac{1}{4}}q^{\frac{3}{4}}\right)$$

$$+(1-p)\left(1+w-\frac{3}{2}pcq+\frac{1}{2}pda^{-\frac{1}{4}}c^{\frac{1}{4}}q^{\frac{3}{4}}-s+\frac{1}{2}da^{-\frac{1}{4}}c^{\frac{1}{4}}q^{\frac{3}{4}}\right) \qquad (A5)$$

The expression (A5) can be solved for q to obtain q^{SB}. Then, q^{SB} can be substituted into (A1) to obtain ε^{SB}. The expressions for q^{SB} and ε^{SB} can be substituted into the patient's expected utility function to obtain EU^{SB}, the patient's second-best expected utility. Expression (A5) cannot be solved to obtain a closed form solution for q. Therefore, I solve for q^{SB}, ε^{SB}, and EU^{SB} using simulations. I return to this later.

Third-Best Problem

To solve (18), I first determine $q(\varepsilon)$. From (5), I find

$$\beta = F_q(q,\varepsilon)$$

$$\beta = \frac{1}{2}dq^{-\frac{1}{2}}\varepsilon^{\frac{1}{2}}$$

$$q = \frac{d^2}{4\beta^2}\varepsilon. \qquad (A6)$$

Equation (A6) defines $q(\varepsilon)$. Using $G(\varepsilon)$ and $q(\varepsilon)$, I can rewrite (17) as

$$\beta = \frac{G'(\varepsilon)}{q'(\varepsilon)}$$

$$\beta = \frac{a\varepsilon}{\left(\frac{d^2}{4\beta^2}\right)}$$

$$\varepsilon = \frac{d^2}{4\beta a}. \qquad (A7)$$

Equation (A7) defines $\varepsilon(\beta)$. Substituting (A7) into (A6) yields

$$q = \frac{d^4}{16\beta^3 a}. \qquad (A8)$$

Equation (A8) defines $q(\beta)$. Using $q(\beta)$ and $\varepsilon(\beta)$ and the definitions of $F(q, \varepsilon)$, $G(\varepsilon)$, and $U(x)$, I can write (18) as

$$\frac{(1-p)\left(-\frac{3pcd^4}{16\beta^4 a} + \frac{pd^4}{16\beta^3 a}\right)}{1 + w - \frac{pcd^4}{16\beta^3 a} + \frac{pd^4}{32\beta^2 a}} = \frac{p\left(\frac{3pcd^4}{16\beta^4 a} - \frac{pd^4}{16\beta^3 a} - \frac{d^4}{8\beta^3 a}\right)}{1 + w - \frac{pcd^4}{16\beta^3 a} + \frac{pd^4}{32\beta^2 a} + \frac{d^4}{16\beta^2 a} - s}, \qquad (A9)$$

after simplifying. The expression (A9) can be solved for β to obtain β^{TB}. Then, β^{TB} can be substituted into (A8) and (A7) to obtain q^{TB} and ε^{TB}. Additionally, β^{TB} can be substituted into the patient's expected utility function to obtain EU^{TB}, the patient's third-best expected utility. Expression (A9) cannot be solved to obtain a closed form solution for β. Therefore, I solve for β^{TB}, q^{TB}, ε^{TB}, and EU^{TB} using simulations. I return to this later.

Simulation for the Three Equilibria

Using mathematical software (Mathematica), I solved for the equilibria for a range of parameter values. Here, I provide the values of the solutions for a specific set of parameters. (I can provide solutions for other sets of parameters upon request.) Results for the three equilibria are for the following parameter values: $d = 0.7$, $p = 0.25$, $w = 1{,}000$, $s = 999$, $a = 2$, and $c = 1$. The solution to the first-best problem is given by $\{q^{FB} = 1.87, \varepsilon^{FB} = 0.97, EU^{FB} = 5.383\}$. The solution to the second-best problem is given by $\{q^{SB} = 0.29, \varepsilon^{SB} = 0.38, EU^{SB} = 5.359\}$. The solution to the third-best problem is given by $\{q^{TB} = 0.20, \varepsilon^{TB} = 0.18, EU^{TB} = 5.358\}$. Furthermore, $\beta^{SB} = 0.40$ and $\beta^{TB} = 0.34$.

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