

IS THERE A PHYSICIAN PEER EFFECT? EVIDENCE FROM NEW DRUG PRESCRIPTIONS

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We investigate whether and how physicians' prescriptions of a new drug are influenced by their colleagues in the same hospital during shared working time. We use longitudinal data of physicians who prescribed antipsychotic drugs for schizophrenia patients in Taiwan between 1997 and 2010. We find that peer effects are small, but stronger among physicians of similar age and among those sharing a longer, larger, or more stable group. Peer effects are also stronger when drugs are newly introduced. We also find that peer effects are more likely to be overestimated using fixed-effect models than using first-difference models. (JEL D01, D83, I10)

I. INTRODUCTION

Is there a peer effect among physicians? The answer may not be so obvious as people commonly believe. Take the existence of two conflicting practices as an example. The pharmaceutical industry spends almost twice as much on promotion as it does on R&D (Gagnon and Lexchin 2008). In spreading its pharmacological innovations, the industry has targeted opinion leaders who are research-active specialists, ostensibly in response to the power of peer influence (Nair, Manchanda, and Bhatia 2010). Conversely, in promoting evidence-based medicine, practitioners and researchers generally believe that the process of information diffusion is very slow.¹ Therefore, one of

the policy recommendations in the Institute of Medicine report is to “enhance dissemination efforts to communicate evidence and guidelines to the general public and professional communities” (Institute of Medicine 2001). Limited evidence on the role of physician peer networks in medical knowledge diffusion and technology adoption perhaps could explain these conflicting practices.²

In this study, we investigate the existence and the pattern of peer effects through social learning among physicians. We refer to social learning as a process in which decision makers collect information by observing others in

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1. For example, in Balas and Boren (2000), it states that “relying on the passive diffusion of information to keep health professionals’ knowledge up to date is doomed to failure in a global environment....”

2. Early studies by Coleman, Katz, and Menzel (1957 and 1966) have shown the impact of interpersonal networks on a physician’s adoption of new drugs. Escarce (1996) finds that early adoption of laparoscopic cholecystectomy by some surgeons in a hospital would lead other surgeons in the same hospital to adopt it nearly 1 year earlier than they otherwise would have done. Burke, Fournier, and Prasad (2003) find that a patient will be more likely to receive angiography or surgical interventions (such as bypass surgery or angioplasty) if the attending physician is in a group that performs more of those procedures. Epstein and Nicholson (2009) find that an increase in the overall c-section rate of a physician’s local peer group leads to an increase in his or her own rate.

ABBREVIATIONS

EGMM: Efficient Generalized Method of Moments
 FD: First-Difference
 FE: Fixed-Effect
 FGA: First-Generation Antipsychotics
 GB: Global Budgeting
 NHI: National Health Insurance
 PIMC: Psychiatric Inpatient Medical Claims
 SGA: Second-Generation Antipsychotics

their social network.³ Through this process, individuals' behaviors may change the behaviors of others in the network via revealed information, and an information externality can arise. If social learning occurs, then peer effects can be detected and revealed by changes in an individual's behavior in response to that of his or her peers.⁴ Our study contributes to the existing literature in three ways. First, we examine not only whether peer effects exist, but also where they exist. If peer effects are heterogeneous, identifying the circumstances to promote physician social interactions can better nurture the dissemination of medical innovations. Second, estimates of peer effects are important for policy consideration. It has long been recognized that health policies play a role in affecting the diffusion of medical innovation (Weisbrod 1991). To gauge the full impact of a policy to be implemented, researchers need to know an aggregate coefficient, including both individual direct responses and a social multiplier based on the peer effects (Glaeser, Sacerdote, and Scheinkman 2003). Third, we examine the persistence of peer effects. Our results will shed important light on the duration and mechanism that lead to productivity spillovers and the well-documented geographic variations in health care provisions (Phelps 2000; Chandra and Staiger 2007).

Specifically, we study whether and how a physician's prescription of second-generation antipsychotics (SGA) for schizophrenia patients could be influenced by the prescription decisions of his or her colleagues working in the same hospital.⁵ We focus on drug prescriptions not only because new drugs can play important roles

in the dissemination of medical innovations, but also because, in contrast to new technology or equipment, drug prescriptions usually do not require a significant amount of fixed inputs. Therefore, its peer effect is less likely to be confounded by the externalities of large input costs, one example of the correlated effects discussed in Manski (1993).⁶ We focus on schizophrenia patients treated by antipsychotic drugs because there have been serious debates about the effectiveness of SGA despite the rapid shift from the first-generation antipsychotics (FGA) to SGA.⁷ Such controversies will provide strong incentives for physicians to continuously acquire new knowledge about SGA.

Identifying peer effects is difficult because of self-selection of a peer group, unobserved heterogeneities of a peer group, and the simultaneity (or "reflection") problem (Manski 1993, 2000; Moffitt 2001; Brock and Durlauf 2007).⁸ We confront these problems by using a unique dataset derived from antipsychotic medications

patients often have difficulty in working or even conducting basic social functions. Most common symptoms include hallucinations, delusions, disordered thinking, and cognitive deficits.

6. That is, followers of a treatment adoption may incur fewer fixed costs than the first adopter if the treatment involves getting new technology or equipment in a hospital. Once the technology is present, all physicians in the hospital can have access to it, and the peer effects can be driven purely by the open access to the common resource in the hospital.

7. The adherence of FGA (such as haloperidol) is low due to very uncomfortable side effects, such as tardive dyskinesia or parkinsonism. Since the approval of SGA (such as clozaril) by the Food and Drug Administration in the United States in 1990, there has been a rapid shift from FGA to SGA, possibly because SGA produces more tolerable side effects. However, whether SGA performs better than FGA in terms of fewer side effects and lower total health care expenditure is still the subject of debate (Duggan 2005). According to a recent large-scale experiment (CATIE), SGA, which is more costly, is found to be no more effective than FGA (Lieberman et al. 2005; Rosenheck et al. 2006).

8. The reflection problem arises when "data on equilibrium outcomes cannot distinguish endogenous interactions from contextual interactions" (Manski 2000, 128). We henceforth refer to the reflection problem as the collinearity problem between the average behavioral outcome in a peer group and the within-group average characteristics of peers. More discussions on the reflection problem are provided by Brock and Durlauf (2007) and Lee (2007). "Manski (1993) has considered a group effect model where social interaction is modeled with expected outcomes and the expected outcomes are solutions from social equilibrium. Manski has pointed out some difficult identification issues on his social effect model as the expected outcome from social equilibrium might be linearly dependent on observed exogenous variables of a group in the model—the 'reflection' problem. The reflection problem refers to the difficulty to distinguish between behavioral and contextual factors" (Lee 2007, 334).

3. Social learning has been studied widely in a variety of contexts including, but not limited to, employer-sponsored health plan choices (Sorensen 2006), retirement plan choices (Duflo and Saez 2003), welfare program participation (Bertrand, Luttmer, and Mullainathan 2000), health care utilization in Milan (Devillanova 2008), consumption of movies (Moretti 2011), and other examples, such as crime and labor market outcomes, which are cited in those studies.

4. The literature generally suggests that such social interactions will affect individuals' behaviors through two mechanisms (Manski 2000). One is social learning, and the other is social norms. Our study focuses on documenting social learning to describe the mechanism of social interaction. Our research goal is analogous to the studies in agriculture (Foster and Rosenzweig 1995; Munshi 2004; Bandiera and Rasul 2006; Conley and Udry 2010), which examine the importance of social learning among farmers on the adoption of new crops or new technologies.

5. Antipsychotic drugs are often used for controlling symptoms of schizophrenia, which is one of the most serious, relapsing, and disabling mental illnesses. Schizophrenia

prescribed for schizophrenia patients in Taiwan between January 1997 and December 2010. The dataset has several features allowing us to address some major challenges impeding the empirical studies on peer effects. First, we have unique and consistent identifiers for patients, physicians, and hospitals over the 14-year period. These identifiers enable us to construct a longitudinal dataset consisting of complete prescriptions of antipsychotic medications for each observed hospital–physician–patient pair during the sample period. To identify the peer effect, we use variations over time within each hospital–physician–patient pair, as opposed to relying exclusively on cross-sectional variations that are often confounded by time-invariant unobserved heterogeneities within each hospital–physician–patient pair (such as a physician’s training background, a patient’s genetic factors, a hospital’s location, and the matching between physicians and patients based on time-invariant unobservables).

Second, a challenge in studying peer effects is how to define peers properly. In the health care industry, physicians working in the same hospital may form a natural peer group, which is especially the case in our empirical setting: most physicians in Taiwan work only in one hospital, and they may interact with their colleagues in the same hospital frequently. However, such interactions may attract physicians with similar characteristics to work in the same hospital and therefore bias the peer effect estimates. Although the self-selected peer group is a legitimate concern, it seems unlikely that a physician is hired, or that his or her peer group is self-selected, based on the propensity to prescribe SGA, given that the effectiveness of SGA is still in debate.

Third, because physicians can affect each other simultaneously, it is difficult to identify the causal effect of peers. Although we are unable to directly address the simultaneity problem given our empirical setting, we conduct falsification checks on the presence of the simultaneity problem. We apply the falsification checks to both the fixed-effect (FE) estimator and the first-difference (FD) estimator used in our study. Although both estimators control for the time-invariant unobserved heterogeneity at the hospital–physician–patient pair level, the consistency of the FD estimator requires a weaker assumption on the exogeneity of regressors than the strong exogeneity assumption required for

the consistency of the FE estimator (Cameron and Trivedi 2005, 730). Therefore, compared with the FE estimator, the FD estimator is relatively less likely to suffer from the simultaneity problem that invalidates the strong exogeneity assumption. By comparing the FD and the FE estimates, we may gauge the extent of the simultaneity bias of the peer effect estimates.

Our empirical findings are consistent with the presence and persistency of peer effects. Our results also indicate that the FE estimator can overestimate peer effects when the strong exogeneity assumption fails (likely due to the simultaneity problem). Overall, we find that peer effects among physicians are small. Our estimates suggest that an increase of 10 percentage points in the SGA prescription share of a physician’s peers during a month will induce an increase of approximately 0.07–0.10 percentage points in the physician’s own SGA prescription share. Our estimates are comparable to the findings of 0.30–0.40 percentage-point increase (in response to a 10 percentage-point increase in opinion leaders’ drug prescriptions) in Nair, Manchanda, and Bhatia (2010). Nevertheless, if the effect is persistent over a long period of time, the cumulative effects are nontrivial. For example, we find that about 69% of the increase in SGA prescriptions in Taiwan over the 14 years (between January 1997 and December 2010) can be explained by a multiplier effect in the presence of the peer effect. Although the peer effect can manifest its impact over time, the long duration for research advancement to reach clinical practice can be costly and harmful to a society (Lenfant 2003).⁹

Furthermore, we find the peer effects to be heterogeneous. First, positive effects appear to exist among physicians of similar age, while in some cases inter-generational peer effects are negative.¹⁰ Second, peer effects are stronger among the peer group that has existed longer,

9. Studies have suggested that it takes about 17 years on average for research advancement to reach clinical practice (Balas and Boren 2000).

10. It has been shown that the information externality can lead individuals to strategically delay an action and wait for more information revealed from their peers (Caplin and Leahy 1998; Bandiera and Rasul 2006). Miguel and Kremer (2003) offer another explanation for their finding that individuals who are randomly exposed to more information about deworming drugs through their social network are significantly less likely to take the drugs and more likely to believe that the drugs are not effective. That is because they have overly optimistic prior beliefs about private drug benefits.

or when the group's composition is more stable, or when the group is larger. Third, peer effects are larger when the drugs are newly approved; the magnitude of peer effects appears to decline over time. Lastly, peer effects diminish if the increase in the SGA prescription is induced by an exogenous shock that adds little information about SGA effectiveness. Our findings suggest that it is important to take into account the heterogeneity of peer effects when designing policies to promote or facilitate social learning among physicians.

The rest of the paper is organized as follows. Section II describes the empirical setting. Section III discusses the identification strategy and lays out the econometric specifications. Section IV presents the empirical findings. Concluding remarks are in Section V.

II. EMPIRICAL SETTING

For our empirical study we combine several data sources from Taiwan; all of them come from the National Health Insurance (NHI) database, which contains the medical claims and eligibility files of all NHI enrollees. Because enrollment in NHI is mandatory in Taiwan, we actually have the utilizations of all (more than 20 million) individuals since the beginning of NHI. Our primary data source is the psychiatric inpatient medical claims (PIMC) database, which records the inpatient and also the outpatient care that occurred between January 1997 and December 2010 of those who ever had inpatient admissions for psychiatric treatment between 1996 and 2007.¹¹ For each inpatient admission and outpatient visit, we have information on the date, diagnosis, payment, and a list of medical codes that indicate the type and units of services provided (e.g., drugs or procedures), from which we can identify whether SGA was prescribed. Moreover, each claim has three identifiers for a patient, a physician, and a hospital, respectively. Using hospital and physician identifiers, we link PIMC with several files containing characteristics of hospitals and physicians, such as hospital type, age, gender, and specialty of a physician (e.g., psychiatry or neurology). We

11. PIMC selects patients who ever had inpatient admissions between 1996 and 2007, and who had the ICD-9 diagnostic codes between 290 and 319 under the supervision of the department of psychiatry; among those patients, PIMC obtains their complete claims (including both inpatient and outpatient use) between January 1997 and December 2010.

also link prescriptions from the PIMC data with the file detailing the descriptions of all drugs approved by NHI, which includes the drug's name, formula, price, dosage, and approval date. As discussed later, the information about the drug allows us to further examine the peer effect in newly approved SGA by different approval years.

Although the PIMC database only includes schizophrenia patients who had at least one inpatient admission for psychiatric treatment between 1996 and 2007, it actually includes about 60% to 70% of schizophrenia patients in Taiwan during our study period (1997–2010), because schizophrenia is a chronic and relapsing illness and episodes of inpatient care are common. Based on other NHI datasets we identify the schizophrenia patients who never had inpatient admissions for psychiatric treatment between 1996 and 2007, and we compare them with our study population from the PIMC. We find that on average the patients of our study population are about 1 to 6 years younger and are slightly more likely to be male; they also have about one more outpatient visit per year, have higher average treatment and drug expenses per visit, and receive more SGA prescriptions per visit. We also find that the PIMC database includes the majority of the outpatient visits (70%–80%) of all schizophrenia patients.

Following early studies (Duggan 2005), we use diagnosis codes to identify individuals who were treated for schizophrenia. To ensure the accuracy of the diagnosis codes among outpatient claims, we restrict our sample to those whose visits were seen by specialists¹² (i.e., psychiatrists and/or neurologists) and who were given at least one week of medications.¹³ In the end, our sample used for estimation based on first differences includes 1,100 physicians (psychiatrists and/or neurologists), 72,273 patients, and 373 hospitals, among which we observe 291,821 hospital–physician–patient pairs in the period of January 1997–December 2010.¹⁴

12. Thus, in our sample all SGA prescriptions are to specialists. This restriction keeps about 90% of the visits in the original sample.

13. More than 87% of the outpatient visits with the diagnosis with schizophrenia involved at least one week of medications.

14. Among the 291,821 observed hospital–physician–patient pairs, there are 276,746 observed physician–patient pairs, which implies that nearly all physician–patient pairs did not change their hospitals ($292,821/276,746=1.06$).

We conduct our study at the level of a hospital–physician–patient pair. The dependent variable of our study is the SGA prescription share for each hospital–physician–patient pair. To calculate the SGA prescription share, we first identify whether each prescribed drug is SGA based on the drug identifier recorded in our PIMC data. Next, we count the number of SGA prescribed for each hospital–physician–patient pair on each date of the treatment (or the physician visit). Then, we divide the SGA count by the total number of drugs prescribed, which ranges from one to three in our PIMC data, for that hospital–physician–patient pair and on that treatment (or the physician visit) date. Using the treatment (or the physician visit) date information, we calculate the age of the physician and the age of the patient, as of the drug prescription date (which is the treatment or the physician visit date). For the physician’s age, we divide the elapsed days between the drug prescription date and the physician’s birth date by 365, and we use the same calculation for the patient’s age based on the patient’s birth date. Thus, patient age and physician age are measured in days.¹⁵

Our main estimation uses data values averaged monthly (from January 1997 to December 2010) by each hospital–physician–patient pair. The sample size for our main estimation is 2,772,966. In our main estimation sample and averaged monthly across hospital–physician–patient pairs, the distribution of the number of prescribed drugs is the following: one drug prescribed, accounting for 70.69% of the observations; two drugs prescribed, accounting for 25.20% of the observations; and three drugs prescribed, accounting for the rest (i.e., 4.11%) of the observations. For patients with major psychiatric disorders, it is a common clinical practice to use multiple antipsychotic drugs (i.e., polytherapy), such as combinations of two SGAs, or older and newer antipsychotics. However, the clinical benefits of antipsychotic polytherapy have not been well studied (Centorrino et al., 2004).

During our sample period and on a monthly basis, the SGA prescription share increased substantially from 3.5% in January 1997 to 62.4% in December 2010, which is shown in Figure 1. During this period, the FGA prescription share dropped while several SGAs were subsequently

15. We conduct a robustness check, using age measured in years, and confirm that our estimates are not affected by this change.

introduced to Taiwan.¹⁶ Despite the continual entries of new SGAs as they were approved for use during our study period, the prescription share for each of the SGAs increased over time, that is, the SGAs are not cannibalizing share from one another.¹⁷ Our study focuses on drug prescriptions by a hospital–physician–patient pair at the drug category level, that is, whether an SGA is prescribed or not. We do not further investigate any possible substitution pattern among SGAs prescribed by a physician for his or her patient. In fact, SGAs all have different pharmacological properties, and schizophrenia patients may also respond to the same SGA differently. As a result, there is still no consensus regarding which SGA should be prescribed first for schizophrenia patients (Johnsen et al. 2010), and therefore, it is not surprising to see the coexistence of several SGAs with different years of market entries.

Summary statistics based on our main estimation sample are reported in column (1) of Table 1. In our study, we define the peer physicians as those physicians who work with the focal physician, excluding the focal physician himself or herself, in the same hospital in each month. The average number of peers based on this definition and in our main estimation sample is 14.752 (shown in column [1] of Table 1).¹⁸ The peer physicians’ average SGA prescription share is 0.476. Note that the calculation of peers’ average SGA prescription share is based on the remaining physicians (i.e., excluding the focal physician) who work in the same hospital with the focal physician in each month. In comparison, the average SGA prescription share for

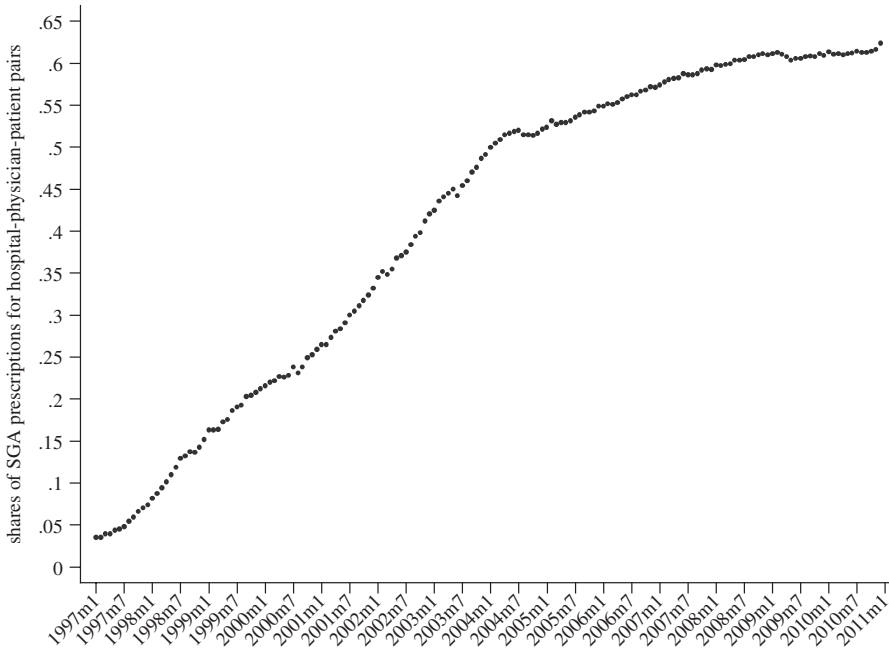
16. The most important new pharmaceutical products (on which SGA is defined) approved and included in the NHI formulary are the following (with the associated drug names in parentheses): Zyprexa (Olanzapine) in 1999; Seroquel (Quetiapine) and Lodopin (Zotepin) in 2000; and Geodon (Ziprasidone) and Solian (Amisulpride) in 2003.

17. For each SGA and each year, we calculated the percentage of SGA prescriptions out of all drug prescriptions using our full sample. Our calculations, for example, show that the prescription shares of Clozapine and Risperido, both of which were introduced to Taiwan prior to our study period, increased from about 4% in 1997 to about 17% in 2010, and from about 1% in 1997 to about 16% in 2010, respectively.

18. In our main estimation sample (used for column [4] of Table 1), there are 3,418 observed hospital–physician pairs and 373 hospitals, which implies that on average there are 9.16 physicians per hospital. Note that the average peer group size is 14.752, which is greater than 9.16. This can be explained by the fact that the number of physicians working in large hospitals is greater than the number of physicians working in small hospitals, and the number of large hospitals outweighs the number of small hospitals.

FIGURE 1

Monthly Average of the Shares of the SGA Prescriptions for Hospital–Physician–Patient Pairs Between January 1997 and December 2010



a hospital–physician–patient pair in our main estimation sample is 0.477 (not shown in the table).

A notable feature of the prescription drug market in Taiwan is that, similar to Japan, health care providers can prescribe and also dispense drugs. To purchase prescription drugs, hospitals (or occasionally private-practice physicians) usually bargain with pharmaceutical companies to set acquisition prices, which are not regulated. In comparison, drug reimbursements to health care providers are fixed and predetermined by the Bureau of National Health Insurance. In Taiwan, most physicians are hospitals’ salary-based employees. Therefore, employed physicians may have the same incentive as the hospital to choose prescription drugs according to the markup. There has been anecdotal evidence that drug dispensing has been a profitable venue for physicians and hospitals in Taiwan. In our data we do not have the information on acquisition prices, but only the reimbursement price per prescription.¹⁹ In our regression analysis

19. If drugs prescribed to a patient exceed a certain amount, then the patient will pay a modest copayment for the

we include the average reimbursement price per prescription by a hospital–physician–patient pair as a proxy variable for the financial incentive that a physician or a hospital may have.²⁰

III. IDENTIFICATION STRATEGY AND SPECIFICATION

To identify a peer effect, we need to deal with three problems extensively discussed in Brock and Durlauf (2007), Lee (2007), Manski (1993, 2000), and Moffitt (2001)—the problems of

drugs. In our sample 87% of the cases pay no copayment; 12% pay less than NT\$200; and 1% pay the maximum copayment of NT\$200.

20. Our regression model uses hospital–physician–patient pair fixed effects and also controls for the average reimbursement price per prescription measured in NT\$1,000 unit (New Taiwan dollar). We compared the estimation results from the regression models with and without including the average reimbursement price of prescribed drugs by a hospital–physician–patient pair. The results are very similar, which suggests that the financial incentive of an individual physician could be uncorrelated with his or her peers’ SGA prescription decisions, after controlling for the hospital–physician–patient fixed effects. Those results are available upon request.

TABLE 1
Peer Effect Estimates Based on Peers Formed on a Monthly Basis

	(1) Summary Statistics Based on Column (4)	(2) FE Estimates	(3) Falsification Check	(4) FD Estimates	(5) Falsification Check	(6) FD Estimates of Lagged Effects
Peers' average SGA prescription share (t)	0.476 [0.228]	0.201*** (0.004)	0.172*** (0.005)			
Number of physicians in a peer group	14.752 [16.753]	0.001*** (0.000)	0.001*** (0.000)			
Physician's age	41.411 [7.459]	0.035*** (0.007)	-0.010 (0.009)			
Patient's age	40.537 [11.851]	-0.002** (0.001)	-0.003*** (0.001)			
Average price (NT\$1,000) of prescribed drugs	0.088 [0.348]	0.110*** (0.002)	0.090*** (0.003)			
Δ Peers' average SGA prescription share (t)				0.007*** (0.001)		0.009*** (0.002)
Δ Peers' average SGA prescription share ($t + 1$)					-0.000 (0.001)	
Δ Peers' average SGA prescription share ($t - 1$)						0.010*** (0.002)
Δ Peers' average SGA prescription share ($t - 2$)						0.007*** (0.002)
Δ Peers' average SGA prescription share ($t - 3$)						0.006*** (0.002)
Δ Peers' average SGA prescription share ($t - 4$)						0.003* (0.001)
Δ Number of physicians in a peer group				0.000** (0.000)	-0.000 (0.000)	0.000 (0.000)
Δ Physician's age				0.029*** (0.005)	-0.041*** (0.006)	0.027*** (0.007)
Δ Patient's age				-0.000 (0.000)	0.001* (0.000)	-0.000 (0.000)
Δ Average price (NT\$1,000) of prescribed drugs				0.084*** (0.002)	-0.006*** (0.002)	0.083*** (0.004)
Number of hospital-physician-patient pairs	291,821					
Number of observations	2,772,966	4,190,722	2,703,993	2,772,966	1,817,871	1,338,784

Notes: Estimation results in columns (2)–(6) are based on Equations (1)–(5), respectively. ΔX represents the change in the value of variable X from period $t - 1$ to period t . Other control variables include the dummy variables for monthly fixed effects. Sample means of the regressors used in column (4) are reported in column (1). Standard deviations of the regressors used in column (4) are reported in brackets in column (1). Standard errors (reported in parentheses) are robust to the hospital-physician-patient level clustering in the conditional variance-covariance matrix of the regression disturbance term.

***Significant at the 1% level; **significant at the 5% level; *significant at the 10% level.

the simultaneity, the correlated unobservables, and the endogenous group formation. In our context, the simultaneity problem stems from the fact that physicians affect each other simultaneously; as a result, it is likely to overestimate the effect of peers on a focal physician. The associated reflection problem occurs because it is difficult to break the collinearity between peers' average SGA prescriptions (generating the endogenous peer effect) and peers' average characteristics (generating the exogenous

effect). Correlated unobservables arise if there are commonly shared factors, such as hospital resources, pharmaceutical marketing, patients' severity, or learning mechanisms, that are unobservable to a researcher but are correlated with the SGA prescriptions of both the focal physician and his or her peers. Finally, the endogenous group formation in our empirical setting means that physicians choose a particular hospital because of similar preferences, motivations,

and other unobserved characteristics that influence the SGA prescriptions.

To deal with these problems, we take advantage of our longitudinal data with information on antipsychotic drug prescriptions by each hospital–physician–patient pair over time. With the longitudinal data we could address the problems of correlated unobservables and endogenous group formation as long as the unobserved heterogeneities are time-invariant at a hospital level (e.g., pharmaceutical marketing or other common learning sources), at a physician level (e.g., preference, motivation, or training background), at a patient level (e.g., preference or innate factors), and at a hospital–physician–patient pair level (e.g., the matching between physicians and patients in a hospital based on time-invariant unobservables). We could also circumvent the reflection problem to the extent that peers’ average characteristics (such as gender, educational attainment, and training background) are time-invariant, whereas their SGA average prescription shares change over time.²¹ In all our econometric specifications, we also control for time effects, which can arise from advertising that varies over time.

In our empirical setting the focal physician’s SGA prescription is measured at the hospital–physician–patient pair level, while the peer physicians’ SGA prescriptions are measured at the level of peer physicians who work in the same hospital with the focal physician, not at the level of hospital–physician–patient pairs. In the latter case, the SGA prescriptions of the focal physician and his or her peers can affect each other if they share the same patient. In the former case (which is our case), that simultaneous influence might be mitigated but will not be avoided completely. Thus, we are unable to completely solve the simultaneity problem with our empirical setting. Instead, we use falsification checks to examine the presence of the simultaneity problem.

We start our estimation of the peer effect with the following regression model:

21. We herein rely on the time-invariant feature of a peer group’s characteristics, such as gender, educational attainment, and training background, to break the collinearity between “exogenous effects” and “endogenous effects” (Manski 1993). This reflection problem discussed by Manski (1993) is pervasive in empirical studies based on Manski’s linear-in-expectation models, unless models proposed by Brock and Durlauf (2007) for studying discrete choices with social interactions are employed.

(1)

$$y_{ijk,t} = \alpha_{ijk} + \gamma \bar{y}_{G_{ik},t} + \beta_1 x_{1G_{ik},t} + \beta_2 x_{2ik,t} + \beta_3 x_{3jk,t} + \beta_4 \bar{p}_{ijk,t} + \delta_t + u_{ijk,t},$$

where we denote a physician by i , a patient by j , a hospital by k , physician i ’s peer group by (G_{ik}, t) , and t indexes the year-month from January 1997 to December 2010. The dependent variable, $y_{ijk,t}$, is the SGA prescription share, measured by the proportion of SGA prescriptions out of total drug prescriptions for the focal hospital–physician–patient pair indexed by ijk at time t ; peer physicians defined as those who work with the focal physician i (excluding the focal physician himself or herself) in the same hospital k in each month are indexed by (G_{ik}, t) ; and $\bar{y}_{G_{ik},t}$ is the SGA prescription share averaged across the peer physicians of the focal physician i , who work with the focal physician i in hospital k at time t . Other control variables include the number of physicians in the focal physician’s peer group ($x_{1G_{ik},t}$), the focal physician’s age ($x_{2ik,t}$), the patient’s age ($x_{3jk,t}$), the price of the drug prescribed by the focal hospital–physician–patient pair averaged across the reimbursement prices of the drugs prescribed by that hospital–physician–patient pair ($\bar{p}_{ijk,t}$). In this regression model, we use the hospital–physician–patient pair fixed effect (α_{ijk}) and the monthly time effect (δ_t). Note that both the physician’s age and the patient’s age are measured by the elapsed days (divided by 365) between their birth dates and the date of the treatment (i.e., drug prescriptions) or the physician visit. Thus, the month-to-month variation in the physician’s age and the patient’s age can be different because the day of the treatment (i.e., drug prescription) or the physician visit can be different from month to month. We also take into account the within hospital–physician–patient pair clustering in the conditional variance–covariance matrix of the disturbance term ($u_{ijk,t}$) for panel-robust statistical inference (Cameron and Trivedi 2005, 727).

In Equation (1), the peer effect is indicated by the parameter γ . To examine the presence of the simultaneity problem, which hinders the identification of γ , we conduct the following falsification check:

(2)

$$y_{ijk,t-1} = \alpha_{ijk} + \gamma_0 \bar{y}_{G_{ik},t} + \beta_1 x_{1G_{ik},t} + \beta_2 x_{2ik,t} + \beta_3 x_{3jk,t} + \beta_4 \bar{p}_{ijk,t} + \delta_t + u_{0ijk,t}.$$

Under the null hypothesis (which is known to be true), γ_0 equals zero because the current peer physicians' average SGA prescription should not predict the focal physician's past SGA prescription (based on chronology). Rejecting the null hypothesis means a nonzero γ_0 , which indicates a correlation between $y_{ijk,t-1}$ and $\bar{y}_{G_{ik},t}$. And, this correlation can be driven by the impact of $y_{ijk,t-1}$ on $\bar{y}_{G_{ik},t}$, suggesting the presence of a simultaneity bias.

We use the fixed-effect (FE) estimator to estimate Equations (1) and (2). The consistency of the FE estimator depends on the strong (or strict) exogeneity assumption, which requires that the disturbance term in Equation (1) should not be correlated with any leads or lags of the regressors, because these leads and lags are used for the within-panel variations in the FE estimator (Cameron and Trivedi 2005, 727). A nonzero γ_0 in Equation (2) means that $u_{0ijk,t-1}$ is correlated with $\bar{y}_{G_{ik},t}$, which provides direct evidence against the strong exogeneity assumption.

Alternatively, we use the following first-difference (FD) model to estimate γ :

$$(3) \quad \Delta y_{ijk,t} = \gamma \Delta \bar{y}_{G_{ik},t} + \beta_1 \Delta x_{1G_{ik},t} \\ + \beta_2 \Delta x_{2ik,t} + \beta_3 \Delta x_{3jk,t} \\ + \beta_4 \Delta \bar{p}_{ijk,t} + \delta_t + \Delta u_{ijk,t}.$$

Here, we take into account the within hospital-physician-patient pair clustering in the conditional variance-covariance matrix of the disturbance term ($\Delta u_{ijk,t}$) for panel-robust statistical inference (Cameron and Trivedi 2005, 730). The FD estimator uses variations only in adjacent periods and thus imposes a weaker assumption on the exogeneity of regressors than the strong exogeneity assumption that is required for the consistency of the FE estimator (Cameron and Trivedi 2005, 730). Next, we conduct the following falsification check using the FD estimator:

$$(4) \quad \Delta y_{ijk,t-1} = \gamma_0 \Delta \bar{y}_{G_{ik},t+1} + \beta_1 \Delta x_{1G_{ik},t} \\ + \beta_2 \Delta x_{2ik,t} + \beta_3 \Delta x_{3jk,t} \\ + \beta_4 \Delta \bar{p}_{ijk,t} \\ + \delta_t + \Delta u_{0ijk,t}.$$

Under the null hypothesis (which is known to be true based on chronology), γ_0 equals zero because the change in peer physicians' average SGA prescription between t and $(t+1)$ should not affect the change in the focal physician's SGA prescription between $(t-2)$ and $(t-1)$.

A nonzero γ_0 in Equation (4) would suggest the association between the focal physician's past SGA prescription and peer physicians' future SGA prescription, which indicates the presence of a simultaneity bias.

IV. ESTIMATES OF LEARNING-BASED PEER EFFECTS

In this section, we examine the presence and the heterogeneity of peer effects.²² For example, between senior and junior physicians, physicians may be more willing to learn from the same generation. Thus, intra-generational (as opposed to inter-generational) social learning is likely to generate positive (instead of negative) information externality and lead to positive (as opposed to negative) peer effects. Furthermore, we also examine whether the peer effects vary with the changes in the environment of social learning. We first investigate the peer effects by the stability and the size of the peer group. If peer effects are indeed driven by social learning, then the effects would become more salient when the group is more stable or when the group size is larger. Next, we examine peer effects by drugs' approval years. We expect social learning to be more relevant when drugs are more recently approved, and thus the peer effects could be stronger. Peer effects may decline over time when more information is revealed and physicians have more knowledge about the new drug's effectiveness.

A. Presence and Heterogeneity of Peer Effects

Table 1 presents our first set of peer effect estimates. Considering the colleagues working with the focal physician in the same hospital in the same month, we find significant peer effects. Notably, in columns (2) and (4), the FE peer effect estimate is larger than the FD estimate. The source of this discrepancy could be indicated by the falsification checks based on Equations (2) and (4). The results of the falsification checks are reported in columns (3) and (5).

22. If social learning drives peer effects, then we could detect heterogeneous peer effects resulting from different information externalities. For brevity, we herein skip a theoretical model (available upon request) that explains why we would expect peer effects to exist among physicians' prescriptions of a new drug when there is insufficient knowledge about the drug's effectiveness and information externalities are likely to occur.

The FE estimator uses all within-panel variations over time, requiring that all of the leads and the lags in the time-varying regressors should be uncorrelated with the contemporaneous disturbance term. This identifying assumption rules out any effect of the unobserved heterogeneities in the current period on future observed heterogeneities. In column (3) of Table 1, we find a significant nonzero estimate of γ_0 (0.172), of which the true value is known to be zero. This provides evidence against the strong exogeneity assumption, which invalidates the consistency of the FE estimator. This also suggests that the peer effect estimate (0.201, shown in column [2]) by the FE estimator is biased when in fact the focal physician's SGA prescription affects the peer physicians' SGA prescriptions. Thus, the FE estimate (0.201) will be an overestimate, because it fails to take into account the effect from the focal physicians on his or her own peers.

In contrast, the FD estimator uses variations in time-varying regressors from the adjacent periods only, which requires that the response from the unobserved heterogeneities in the current period should not affect the observed heterogeneities in the immediate next period—a weaker assumption than the strong exogeneity assumption needed for the consistency of the FE estimator (Cameron and Trivedi 2005, 730). In column (4), we see that the FD estimate is much smaller than the FE estimate; this reduction in magnitude could be explained by the elimination of the possible effects of focal physicians' SGA prescriptions in the current period on their peer physicians' SGA prescriptions in the future periods. Based on Equation (4), we find the FD estimate of γ_0 to be statistically insignificant, which supports the null hypothesis that the peer effect described in Equation (4) is known to be zero. Since the FD estimator is less likely to suffer from the simultaneity problem than the FE estimator, we focus on FD estimations in the following analyses.

Our results suggest that on a monthly basis there is an increase of approximately 0.07 percentage points in the SGA prescription share of the focal hospital–physician–patient pair in response to an increase of 10 percentage points in the peer physicians' SGA prescription share (shown in column [4]). This is our baseline peer effect estimate. It has been suggested that

a patient's response to an antipsychotic treatment during the first 1–2 weeks is highly predictive of the long-term effectiveness (Stauffer et al., 2011). If there is no response to the treatment or no symptom improvement during the first 1–2 weeks, then it should be considered to switch to other antipsychotic drugs. Thus, it is plausible for us to examine the peer effect during a month as physicians who consider the switching may need to learn about certain SGA effectiveness from peers' SGA prescriptions.

If the peer effects stem from social learning, then as long as the uncertainty of SGA effectiveness exists, we would expect that such effects will persist over time. To test for this persistence, we include four additional terms for peers' lagged SGA prescription shares to Equation (3). The augmented regression model is specified as follows:

$$\begin{aligned}
 (5) \quad \Delta y_{ijk,t} = & \gamma_0 \Delta \bar{y}_{G_{ik,t}} + \gamma_1 \Delta \bar{y}_{G_{ik,t-1}} \\
 & + \gamma_2 \Delta \bar{y}_{G_{ik,t-2}} + \gamma_3 \Delta \bar{y}_{G_{ik,t-3}} \\
 & + \gamma_4 \Delta \bar{y}_{G_{ik,t-4}} + \beta_1 \Delta x_{1G_{ik,t}} \\
 & + \beta_2 \Delta x_{2ik,t} + \beta_3 \Delta x_{3jk,t} + \beta_4 \Delta \bar{p}_{ijk,t} \\
 & + \delta_t + \Delta u_{ijk,t},
 \end{aligned}$$

where the parameters $\gamma_1, \gamma_2, \gamma_3,$ and γ_4 indicate the influence from the SGA prescription shares of peers in the past 1–4 months on the focal hospital–physician–patient pair. The parameter γ_0 captures the contemporaneous peer effect, which is indicated by γ in Equation (3). The estimates in column (6) of Table 1, based on Equation (5), confirm this persistency feature in the learning-based peer effects. The contemporaneous peer effect estimate (0.009) is similar to the baseline estimate (0.007). In addition to this contemporaneous learning-based peer effect, we find that peers' past SGA prescriptions over the last 1–4 months have continued to exert significant influence on the focal hospital–physician–patient pair, with the magnitude of the effect ranging from 0.003 to 0.010. If the amount of knowledge transferred from peers to the focal physician increases with the length of the learning period, then we would expect the peers' lagged SGA prescription decisions to have a greater impact than their contemporaneous ones. Our estimates in column (6) of Table 1 have confirmed this. The influence from peers' prescription decisions in the past month increases by roughly

11% when compared with the peer influence in the current month. We also notice that the peer influence begins to diminish from the past month and becomes marginally significant after 3 months.

The finding of a significant peer effect implies a social multiplier (Glaeser, Sacerdote, and Scheinkman 2003) approximately equal to 1.007.²³ This social multiplier derived from the monthly peer effect appears small. However, its cumulative effect over time is not trivial. Based on our empirical finding that the peer effect is persistent, we could uncover a total multiplier effect over the 168-month period (from January 1997 to December 2010) approximately equal to 3.228.²⁴ Our data show that the monthly SGA prescription share increased from 0.035 in January 1997 to 0.624 in December 2010, a nearly 17-fold increase over the 168-month period. It is important to recognize that approximately 69% of this observed nearly 17-fold increase in the SGA prescription share could potentially be explained by the underlying social multiplier in the presence of the peer effect.²⁵

Next, we examine the heterogeneity of peer effects. We examine the influences of peer effects among junior, medium-aged, and senior physicians. We modify Equation (3) by using three interaction terms based on three binary indicators: one for peers aged under 35 (junior

physicians), one for peers aged between 35 and 55 (medium-aged physicians), and another for peers aged above 55 (senior physicians). The regression model is specified as follows:

$$(6) \quad \begin{aligned} \Delta y_{ijk,t} = & \gamma_0 \Delta \bar{y}_{G_{ik,t}} \cdot 1\{\text{peer's age} < 35\} \\ & + \gamma_1 \Delta \bar{y}_{G_{ik,t}} \cdot 1\{35 \leq \text{peer's age} \leq 55\} \\ & + \gamma_2 \Delta \bar{y}_{G_{ik,t}} \cdot 1\{\text{peer's age} > 55\} \\ & + \beta_1 \Delta x_{1G_{ik,t}} + \beta_2 \Delta x_{2ik,t} + \beta_3 \Delta x_{3jk,t} \\ & + \beta_4 \Delta \bar{p}_{ijk,t} + \delta_t + \Delta u_{ijk,t}, \end{aligned}$$

where γ_0 , γ_1 , and γ_2 capture the peer influence among junior, medium-aged, and senior physicians, respectively. We estimate Equation (6) for three subgroups of focal physicians: junior physicians under 35, senior physicians above 55, and physicians aged between 35 and 55. Separate results are reported in Table 2.

Given that insufficient knowledge about the effectiveness of SGA is likely and thus learning among physicians is possible, we find that such learning appears to be most salient for physicians aged 35–55 (shown in column [2] of Table 2): an increase of 10 percentage points in the SGA prescription share of physicians who work in the same hospital and in the same month with the focal physician aged 35–55 is associated with an increase of approximately 0.07–0.08 percentage points in that focal hospital–physician–patient pair’s own SGA prescription share. The results in columns (1) and (2) suggest an intra-generational peer effect, which is probably due to similar backgrounds and experiences among physicians of similar age: physicians could regard the prescription decisions of their peers of similar age as a relevant source of information because they might share similar backgrounds or experiences. The results in columns (1) and (2) also suggest a pattern for inter-generational peer effects. In column (1) we find that junior physicians’ SGA prescription decisions respond to their medium-aged peer physicians. Similarly, in column (2) we find that medium-aged physicians’ SGA prescription decisions respond to their senior peer physicians. In contrast, in column (3) we find that all peer effects for senior physicians are insignificant. These findings suggest that in the presence of insufficient knowledge about SGA effectiveness, the differences in backgrounds or experiences between senior physicians and junior (or medium-aged) physicians may counterveil the perceived information or knowledge

23. That is, $1/(1 - 0.007) \approx 1.007$. This is calculated based on the estimated peer effect equal to 0.007 on a monthly basis for repeated cross sections. If an individual’s outcome rises by α (where $0 < \alpha < 1$) as his or her peers’ average outcome rises by 1, then the social multiplier equals roughly $1/(1 - \alpha)$ for large enough groups (Glaeser, Sacerdote, and Scheinkman 2003). In our empirical setting, for not quite large groups, if we can reasonably assume that the interaction between the focal physician and his or her peer physicians occurs many times within a month and each time during that month with a peer effect equal to α , then the calculation above is still valid.

24. That is, $1.007^{168} \approx 3.228$. This calculation is based on the result that peer effect is persistent between period t and period $t - 1$ so that the monthly-based social multiplier carries over month to month.

25. Let Δy be the outcome change between the first and the last period (i.e., $\Delta y = 0.624 - 0.035 = 0.589$). Let $\Delta \mathbf{x}'\beta$ be the change (in the outcome) explained by exogenous factors ($\Delta \mathbf{x}$) between the first and the last period, such as drug advertising and payment structures. Removing the multiplier effect which inflates the outcome change in each month, we uncover the change in the outcome due to the changes in exogenous factors in the absence of the multiplier effect, which is $\Delta \mathbf{x}'\beta = 0.589/3.228 \approx 0.182$. In this sense, those exogenous factors in the absence of the multiplier effect can account for approximately 31% ($0.182/0.589 \approx 0.309$) of the total increase in the SGA prescription share over the 168-month period.

TABLE 2

Intra-Generational and Inter-Generational Peer Effects Based on Peers Formed on a Monthly Basis

	(1) Junior Physicians (age <35)	(2) Physicians (age 35–55)	(3) Senior Physicians (age >55)
Δ Junior (age <35) peers’ average SGA prescription share	0.010*** (0.003)	0.007*** (0.001)	0.005 (0.008)
Δ Medium-aged (35–55) peers’ average SGA prescription share	0.009*** (0.003)	0.007*** (0.001)	0.004 (0.007)
Δ Senior (age >55) peers’ average SGA prescription share	−0.001 (0.009)	0.008*** (0.003)	−0.001 (0.008)
Δ Number of physicians in a peer group	0.000 (0.000)	0.000 (0.000)	0.001 (0.000)
Δ Physician’s age	0.026** (0.011)	0.030*** (0.006)	0.046** (0.023)
Δ Patient’s age	0.000 (0.001)	−0.000 (0.000)	0.000 (0.000)
Δ Average price (NT\$1,000) of prescribed drugs	0.077*** (0.005)	0.085*** (0.003)	0.102*** (0.010)
Number of observations	562,872	2,064,536	145,558

Notes: Estimation results in columns (1)–(3) are based on Equation (6) and are obtained from the subsamples of physicians aged under 35, between 35 and 55, and above 55, respectively. ΔX represents the change in the value of variable X from period $t - 1$ to period t . Other control variables include the dummy variables for monthly fixed effects. Standard errors (reported in parentheses) are robust to the hospital–physician–patient level clustering in the conditional variance–covariance matrix of the regression disturbance term.

***Significant at the 1% level; **significant at the 5% level; *significant at the 10% level.

transferrable from peer physicians’ prescription decisions. Thus, “wait-and-see” could arise, especially for senior physicians. In contrast, for junior and medium-aged physicians, similar backgrounds or experiences shared by them may facilitate knowledge transfer from peers and thus increase the likelihood of “following the crowd.”

Note that in our main estimation sample (used for column [4] of Table 1) there are 291,821 hospital–physician–patient pairs and 132,064 hospital–patient pairs. Thus, within a hospital, which is the basis of our peer group definition, a patient could be treated by 2.21 physicians on average, and there are patients who switched physicians within a hospital.²⁶ It is possible that a patient switches to another physician because the patient wants the new physician to prescribe SGA for him or her. If this is true and since the peer group of a focal physician consists of his or her colleagues who work in the same hospital in

the same month and who may have prescribing habits similar to the focal physician, it would appear that the physicians learn from one another when in fact it is the patients who switch physicians who are driving the observed correlation of prescribing behaviors among physicians.

Comparing patients who switched physicians within a hospital with those who stayed with the same physician, we find that the average peer physicians’ SGA prescription share among those switchers is slightly higher, by 0.0005, than the average peer physicians’ SGA prescription share among those non-switchers. This is consistent with the possibility that patients switch to different physicians for more SGA prescriptions. But, given the tiny difference in SGA prescription shares between the two groups, we would expect the influence of the switchers on physicians’ SGA prescriptions to be small. In fact, when we regress the SGA prescription share of a focal hospital–physician–patient pair on the dummy variable indicating whether the patient switches to other physicians in the same hospital (equal to 1) or not (equal to 0), we find the coefficient estimate to be −0.0002, which is not statistically significant.

26. In our estimation sample, 42.39% of patients stayed with the same physician in the same hospital; 17.78% of patients had two physicians in the same hospital; 10.29% of patients had three physicians in the same hospital; and 29.54% of patients had more than three physicians in the same hospital.

Next, we repeat the same analyses conducted in Tables 1 and 2, but we restrict the sample to patients who never switch physicians in the same hospital. The results of this subsample analysis are reported in Table 3. Here, we find the peer effect estimate to be 0.008 (column [1]), which is very similar to the estimate based on the full sample (0.007, shown in column 4 of Table 1); the effects are also persistent (column [2]) and heterogeneous among junior, medium-aged, and senior physicians (columns [3]–[5]). Here, we also confirm the pattern that junior and medium-aged physicians' SGA prescription behaviors are likely to be affected by peers of similar age, but senior physicians' SGA prescription behaviors are likely to be unaffected by peers.

B. Monotonicity of Learning-Based Peer Effects

Next, we examine the monotonicity of the learning-based peer effect—the effect should be larger (or smaller) when an empirical setting promotes more (or less) social learning.

Group Stability and Size. If the peer effects among physicians' prescription decisions are driven by learning about SGA effectiveness, then we would also expect such peer influence to be strengthened in the group that stays stable.²⁷ In that group, more interactions and exchanges of knowledge are likely, and therefore more social learning would occur. Here, we consider another peer group for the FD estimator, in which there is no change in group size (but not necessarily no change in group composition) in adjacent periods. Comparing columns (1)

27. If peer influence in physicians' SGA prescription decisions is induced by learning through observing peers' decisions, then we would expect such influence to be strengthened (or weakened) once the learning period is longer (or shorter), because learning takes time. In the Appendix Tables A1 and A2, we consider two alternative definitions of the focal physician's peer group, one on a weekly basis (with a shorter learning period) and the other on a quarterly basis (with a longer learning period). Thus, the peer group of a focal physician comprises those working with him or her in the same hospital in the same week, or in the same hospital in the same quarter. If the learning period becomes longer, then we would expect the peer effect estimates in Table 1 (columns [4] and [6]) to increase, which will be consistent with the presence of social learning. Our estimates in Appendix Tables A1 and A2 are consistent with this prediction: the peer effects become larger when the peer group is formed over a longer period. For the peer group formed on a quarterly basis, we also confirm that the peer effect is larger when the group is more stable or larger (results reported in Appendix Table A2).

and (2) in Table 4, we find that the peer effect estimate doubled (from 0.004 to 0.008) in the peer group that remains constant in size between two adjacent months. Next, we conduct the estimation by group size and report the results in columns (3) and (4). We choose 10 as the cutoff number of physicians in a focal physician's peer group, which is close to the median of the size of the peer group when it is defined on a monthly, weekly, or quarterly basis. Here, we find that the peer effect estimate is greater (0.010 vs. 0.007) in a larger peer group. This suggests that peer influence increases with the size of the peer group in which the opportunity of social learning may increase.²⁸

Peer Effects by SGA Approval Years. In the presence of social learning, peer effects could be strongest when drugs are just approved for use. Over time the peer effect for one specific drug may decline as physicians can learn about

28. In Appendix Table A3, we consider SGA prescriptions of two particular types of physicians in a peer group, who may generate more plausibly exogenous source of variation in a focal physician's information source. The first type of physicians includes those entering a hospital in a given month. We identify those entering physicians if their first month of work is that given month and they work in the same hospital with the focal physician (but we do not count January 1997 as the first month of work since it is the start month of our sample period). The second type of peers includes those exiting a hospital in a given month. Similarly, we identify those exiting physicians if their last month of work is that given month and they work in the same hospital with the focal physician (but we do not count December 2010 as the last month of work since it is the end month of our sample period). Since we cannot identify the actual turnover of physicians in a hospital, we view those results as robustness checks. For comparison purpose, we report column (4) of Table 1 again in column (1). Columns (2) and (3) are based on the regressions controlling for the change in peer physicians' average SGA prescription share due to the entering physicians and due to the exiting physicians, respectively. Here we find an asymmetric effect from those entering and exiting peers: having an entering peer who prescribes more SGA increases the SGA prescription of the focal physician, while having an exiting peer who prescribes more SGA reduces the SGA prescription of the focal physician. This asymmetric pattern could be driven by the addition or reduction of information source about SGA for the focal physician. In the joint estimation with results reported in column (4), we find that the peer effect estimates (0.003 and -0.003) are almost the same as the ones based on the two separate estimations (columns [2] and [3]). This suggests that, in our empirical setting, a common "shock" to all physicians in a hospital might not be a major concern because if that common shock exists, then the entering physicians' SGA prescription shares would be correlated with the exiting physicians' SGA prescription shares. In this case, results of the joint estimation (column [4]) would differ from the results from the separate estimations (columns [2] and [3]).

TABLE 3

Peer Effect Estimates Based on Peers Formed on a Monthly Basis and Patients Who Never Switch Physicians Within Hospitals

	(1) Main Results	(2) Lagged Effects	(3) Junior Physicians (age <35)	(4) Physicians (age 35–55)	(5) Senior Physicians (age >55)
Δ Peers' average SGA prescription share (t)	0.008*** (0.003)	0.012*** (0.004)			
Δ Peers' average SGA prescription share ($t - 1$)		0.008* (0.005)			
Δ Peers' average SGA prescription share ($t - 2$)		0.004 (0.005)			
Δ Peers' average SGA prescription share ($t - 3$)		0.006 (0.004)			
Δ Peers' average SGA prescription share ($t - 4$)		-0.001 (0.004)			
Δ Junior (age <35) peers' average SGA prescription share			0.053*** (0.013)	0.002 (0.003)	0.006 (0.038)
Δ Medium-aged (35–55) peers' average SGA prescription share			0.024** (0.011)	0.007** (0.003)	-0.012 (0.019)
Δ Senior (age >55) peers' average SGA prescription share			0.038 (0.030)	-0.001 (0.008)	0.017 (0.021)
Δ Number of physicians in a peer group	0.000 (0.000)	0.001** (0.000)	-0.000 (0.001)	0.000 (0.000)	0.004* (0.002)
Δ Physician's age	0.010 (0.019)	-0.020 (0.026)	-0.026 (0.055)	0.008 (0.021)	0.128* (0.078)
Δ Patient's age	0.000 (0.000)	0.000 (0.000)	-0.000 (0.000)	0.000 (0.000)	0.000 (0.000)
Δ Average price (NT\$1,000) of prescribed drugs	0.103*** (0.008)	0.106*** (0.013)	0.091*** (0.018)	0.100*** (0.009)	0.134*** (0.027)
Number of observations	225,941	121,854	26,875	183,343	15,723

Notes: Estimation results in columns (1) and (2) are based on Equations (3) and (5), respectively. Estimation results in columns (3)–(5) are based on Equation (6) and are obtained from the subsamples of physicians aged under 35, between 35 and 55, and above 55, respectively. ΔX represents the change in the value of variable X from period $t - 1$ to period t . Other control variables include the dummy variables for monthly fixed effects. Standard errors (reported in parentheses) are robust to the hospital–physician–patient level clustering in the conditional variance–covariance matrix of the regression disturbance term.

***Significant at the 1% level; **significant at the 5% level; *significant at the 10% level.

the drug through their own studies or their experiences of treating patients. In our dataset we have information on drug approval years, which allows us to further examine the peer effects by SGA approval years. Here, we consider the following two sets of SGA approved for use by the Bureau of National Health Insurance in Taiwan: Zyprexa in 1999, and Geodon and Solian in 2003.²⁹ In Table 5 we report

the peer effect estimates for SGA approved for use in 1999 (in Panel A), and for SGA approved for use in 2003 (in Panel B). In Panel A (or B), the SGA prescription share, for the focal hospital–physician–patient pair and for the peer physicians, is measured by the proportion of prescribed SGA approved for use in 1999 (or 2003) out of total drug prescriptions. Peer effects are estimated in each year after the approval year, through 2010, based on Equation (3).

Overall, we find that peer effects are strongest within the first year of approval. In Panel A, within the first year of approval (1999–2000), the peer effect estimate is 0.179. The magnitude of the peer effects decreases by about

29. To be more precise, SGA is defined on the basis of pharmaceutical products that were approved and included in the NHI formulary. In Taiwan, the following pharmaceutical products were approved by the Department of Health: Olanzapine, Ziprasidone, and Amisulpride. These pharmaceutical products are sold under the following drug names Zyprexa, Geodon, and Solian, respectively.

TABLE 4

Peer Effects FD Estimates by the Stability and Size of Peer Groups Formed on a Monthly Basis

	(1) Group Size Not Fixed	(2) Group Size Fixed	(3) Group Size ≤10	(4) Group Size >10
Δ Peers' average SGA prescription share	0.004** (0.002)	0.008*** (0.001)	0.007*** (0.001)	0.010*** (0.003)
Δ Physician's age	0.028*** (0.008)	0.030*** (0.006)	0.029*** (0.005)	0.034*** (0.007)
Δ Patient's age	0.000 (0.000)	−0.000 (0.000)	−0.000 (0.000)	0.001 (0.001)
Δ Average price (NT\$1,000) of prescribed drugs	0.086*** (0.003)	0.083*** (0.003)	0.084*** (0.002)	0.084*** (0.003)
Falsification check	Passed	Passed	Passed	Passed
Number of observations	1,016,439	1,756,527	2,772,002	1,195,758

Notes: Estimation results in columns (1)–(4) are based on Equation (3). The falsification check is based on Equation (4). Group size refers to the number of physicians in a focal physician's peer group. ΔX represents the change in the value of variable X from period $t - 1$ to period t . Other control variables include the dummy variables for monthly fixed effects. Standard errors (reported in parentheses) are robust to the hospital–physician–patient level clustering in the conditional variance–covariance matrix of the regression disturbance term.

***Significant at the 1% level; **significant at the 5% level; *significant at the 10% level.

53% (from 0.179 to 0.084) within the second year (2000–2001) after the approval year, and decreases by about 56% (from 0.084 to 0.037) within the third year (2001–2002), and decreases by about 51% (from 0.037 to 0.018) in the fourth year (2002–2003). In Panel B, we also find that the peer effect appears to be the strongest within the first year after the SGA approval year, and there is a decline in the peer effect between the first year and the second year after the SGA approval year. These results imply that the peer effect could be stronger soon after a drug is approved for use, when there is more uncertainty about the drug's effectiveness so that social learning is more relevant or important. These results also imply that for a specific SGA, an individual physician, through his or her own experience of treating patients with that SGA, may rely decreasingly on peers.

One factor that may contribute to the decreasing pattern is that physicians may substitute a new SGA for an old SGA when the new one is introduced. For example, physicians may substitute Geodon or Solian (approved in 2003) for Zyprexa (approved in 1999) after 2003. If most physicians have this substitution, then the prescriptions of Zyprexa will be likely to go down for both the focal and peer physicians, which will lead to an overestimate of peer effect. For SGA approved in 1999 (Panel A), the estimate for 2003–2004 (column [5])

is slightly larger than the one for 2002–2003 (column [4]), but the difference is almost negligible. It suggests that the SGA substitutions are not likely to bias our estimates for the peer effects.

C. Alternative Explanation

In this section, we investigate whether the learning-based peer effect can be falsified by an exogenous shock in which there is possibly no knowledge about SGA effectiveness learned from peers' prescription decisions. If significant peer effects are found in this situation, we would suspect that the earlier peer effect estimates could have an alternative interpretation—for example, being the result of the social norm under a common shock unobserved to researchers.

Here we use the global budgeting (GB) policy, which imposed a cap on total expenditures in hospital care, to conduct the falsification test. Because drug expenditures are subtracted from the cap first, the reimbursement for drug expenditures is not subject to any uncertainty. Consequently, hospitals and thus their employed physicians have stronger incentives to increase drug expenditures by prescribing more drugs, such as SGA (Chou et al. 2010). In this situation, the increase in SGA prescriptions from peers could contain little new information about SGA effectiveness.

TABLE 5
Peer Effects by SGA Approval Years

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
	1999– 2000	2000– 2001	2001– 2002	2002– 2003	2003– 2004	2004– 2005	2005– 2006	2006– 2007	2007– 2008	2008– 2009	2009– 2010	Approval year– 2010
<i>Panel A</i>												
Peer effect in SGA approved for use in 1999	0.179*** (0.018)	0.084*** (0.008)	0.037*** (0.005)	0.018*** (0.004)	0.021*** (0.003)	0.019*** (0.003)	0.005 (0.003)	0.007** (0.003)	0.018*** (0.003)	0.029*** (0.003)	0.037*** (0.003)	0.026*** (0.002)
Number of observations	286,735	330,837	381,759	407,034	429,780	458,513	486,448	496,968	492,079	495,889	488,855	2,565,656
<i>Panel B</i>												
Peer effect in SGA approved for use in 2003					0.167*** (0.015)	0.139*** (0.010)	0.089*** (0.006)	0.084*** (0.005)	0.051*** (0.004)	0.013*** (0.003)	0.008*** (0.003)	0.052*** (0.003)
Number of observations					429,780	458,513	486,448	496,968	492,079	495,889	488,855	1,897,162

Notes: Estimation results in columns (1)–(12) are based on Equation (3) for peers formed on a monthly basis and by each subsample. The subsamples used in columns (1)–(11) are selected by 1 up to 11 years after the year when the SGA, which was prescribed by both the focal physician and the peer physicians, was approved for use. Other control variables include the change in the number of physicians in a focal physician’s peer group, the change in the focal physician’s age, the change in the patient’s age, the change in the average price of drugs prescribed by the focal hospital–physician–patient pair, and the dummy variables for monthly fixed effects. Standard errors (reported in parentheses) are robust to the hospital–physician–patient level clustering in the conditional variance–covariance matrix of the regression disturbance term.

***Significant at the 1% level; **Significant at the 5% level; *Significant at the 10% level.

TABLE 6
Information Externality from Weekly Peers under a Policy Intervention

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	±1 Day	±2 Days	±3 Days	±4 Days	±5 Days	±6 Days	±7 Days	±8 Days	±9 Days	±10 Days
Change in peers' average SGA prescription share	0.298 (0.193)	-0.009 (0.274)	-1.044 (0.950)	-1.208 (0.736)	-0.795 (0.670)	-0.811 (0.597)	-0.760 (0.553)	-0.355 (0.552)	-0.395 (0.456)	-0.161 (0.455)
Change in average price of prescribed drugs	3.265*** (0.851)	3.137*** (0.691)	2.341*** (0.682)	1.111 (0.705)	1.372** (0.593)	1.248*** (0.478)	1.143** (0.455)	0.520 (0.345)	0.465 (0.291)	0.610* (0.334)
Number of observations	191	268	383	512	604	693	905	991	1,070	1,209
<i>p</i> value of Hansen's <i>J</i> -statistic	.727	.590	.592	.327	.531	.413	.568	.408	.666	.585
	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)	(19)	(20)
	±11 Days	±12 Days	±13 Days	±14 Days	±15 Days	±16 Days	±17 Days	±18 Days	±19 Days	±20 Days
Change in peers' average SGA prescription share	-0.228 (0.381)	0.141 (0.501)	0.109 (0.503)	0.247 (0.429)	0.384 (0.609)	0.972 (1.124)	0.095 (0.939)	-0.956 (1.575)	-2.747 (3.966)	-1.692 (2.109)
Change in average price of prescribed drugs	0.608* (0.328)	0.536* (0.292)	0.538** (0.270)	0.664** (0.281)	0.621** (0.269)	0.490* (0.260)	0.509** (0.249)	0.669** (0.269)	0.779* (0.399)	0.755** (0.303)
Number of observations	1,344	1,435	1,516	1,721	1,795	1,880	2,008	2,141	2,236	2,316
<i>p</i> value of Hansen's <i>J</i> -statistic	.579	.538	.743	.893	.920	.791	.426	.700	.976	.948
	(21)	(22)	(23)	(24)	(25)	(26)	(27)	(28)	(29)	(30)
	±21 Days	±22 Days	±23 Days	±24 Days	±25 Days	±26 Days	±27 Days	±28 Days	±29 Days	±30 Days
Change in peers' average SGA prescription share	-1.595 (1.589)	-1.575 (1.479)	-1.682 (1.684)	-1.586 (1.415)	-0.947 (0.871)	-1.010 (0.897)	-1.022 (0.907)	-1.073 (0.818)	-1.486 (1.180)	-1.721 (1.292)
Change in average price of prescribed drugs	0.688** (0.287)	0.689** (0.281)	0.710** (0.292)	0.660** (0.265)	0.730*** (0.215)	0.747*** (0.208)	0.783*** (0.204)	0.816*** (0.213)	0.924*** (0.251)	0.961*** (0.255)
Number of observations	2,527	2,603	2,695	2,831	2,944	3,047	3,123	3,325	3,400	3,485
<i>p</i> value of Hansen's <i>J</i> -statistic	.926	.945	.977	.996	.984	.961	.991	.964	.979	.979

Notes: Estimation results in columns (1)–(30) are based on Equation (3) for peers formed on a weekly basis. The column *X* uses a subsample of physicians working *X* days before or after July 1, 2002, where *X* = 1, 2, ..., 30. Three instrumental variables are used for the change in peers' average SGA prescription share in each subsample regression based on Equation (3). These three instrumental variables are: (a) the binary indicator of whether the physician worked after July 1, 2002; (b) the number of days that the physician worked before or after July 1, 2002; and (c) the interaction between (a) and (b). The two-step efficient GMM is used, allowing for the hospital-level clustering in the conditional variance-covariance matrix of the regression disturbance term. Other control variables include the change in the number of physicians in a focal physician's peer group. Standard errors are reported in parentheses. Also presented are the *p* values of the Hansen's *J*-statistics for testing the overidentifying restrictions based on the GMM criterion function.

***Significant at the 1% level; **Significant at the 5% level; *Significant at the 10% level.

The GB policy took effect on July 1, 2002, which we treat as a break point in our estimation sample. To investigate this policy-induced peer effect, we use a before–after (relative to the break point) design for a series of subsamples of physicians working *within* 1 to 30 days before and after this break point. The peer physicians are defined on a weekly basis, because our focus is the short-term impact (under this policy change) of peers who have worked lately with the focal physician. Next we use GB (a binary dummy variable equal to 1 indicating the periods after July 1, 2002) together with the other two related variables—the number of days (within 1 to 30 days) that the focal physician works before or after the break point and its interaction with GB—to construct orthogonality conditions for the disturbance term in Equation (3). Then we estimate Equation (3) using a two-step efficient generalized method of moments (EGMM) based on the three orthogonal conditions derived from the GB, the number of days, and the interaction of the two. We also obtain the p values associated with the Hansen’s J -statistic for testing the validity of those three orthogonality conditions, based on the GMM criterion function evaluated at the EGMM estimates.

Our peer effect estimates reported in columns (1)–(30) of Table 6 passed this falsification test. In a range of within 1 to 30 days before and after the break point, we do not find significant peer effects induced by GB. Our falsification test conducted on a series of subsamples provides additional support for the learning-based peer effects on physicians’ SGA prescription decisions on the basis of confirming no peer effect when there is possibly no knowledge about SGA effectiveness learned from peers’ prescription decisions.

V. CONCLUSION

We provided empirical evidence consistent with the presence of peer effects among physicians and further examined whether social learning could be an important driving force behind the peer effects. Specifically, we examined how a physician’s SGA prescription decision could be influenced by his or her peers. We found that positive peer effects are more likely to exist when peers are of similar age, presumably having similar (and comparable) experience and background. Peer effects are stronger when the peer group is more stable, when the peer

group is larger, or when the period of social learning (through observation) is longer. Peer effects also are stronger when drugs are newly approved.

Our findings have several implications. First, although the peer effect among physicians is small in general, it is persistent, heterogeneous, and could manifest its impact over time. One possible implication of our findings, and something worth further research to understand, is the extent to which more interaction, facilitated through changes in physical infrastructure, could increase peer effects through a multiplier effect. As Berwick (2003, 1974) points out, “the crucial interface between the early adopter and the early majority cannot be effectively supported by memoranda or publications. Spread requires social interaction.” Peer effects will be stronger when physicians have direct interactions with their colleagues through observations or conversations.

Second, peer effects are the strongest when the innovation is newly introduced. Based on our estimates, the multiplier effect in the first year of introducing new drugs could be eight times as large as the baseline multiplier effect.³⁰ It implies that promoting medical innovations or new scientific findings that are beneficial to a society can be most effective when the new drug, technology, device, or practice is first introduced.

Third, our results contribute to the literature on the productivity spillovers and geographic variations in health care provisions (Chandra and Staiger 2007). Phelps (2000) and his colleagues propose a Bayesian learning process to explain the persistency of local treatment styles once they emerge. The initial beliefs usually were formed during physicians’ medical schooling and residency training. Through the Bayesian learning process by observing others, treatment styles are expected to eventually converge and persist within the same geographic area. Thus, different areas represent different clusters of treatment styles. It is learning among physicians that could lead to treatment style clustering.

30. Taking the coefficient estimate of peer effect in SGA approved for use in 2003 (column [5] in Table 5) as an example, the multiplier effect over a 1-year period is equal to $(1/(1-0.167))^{12} \approx 8.96$. The baseline multiplier effect over a 1-year period is $(1/(1-0.007))^{12} \approx 1.09$.

There are several caveats to our study. First, our peer effect estimated at a hospital–physician–patient pair level may capture patients’ herd behavior too, because some patients may share the same physician. In this case, a patient may request that his or her physician prescribe a particular SGA when more and more patients of the same physician request this particular SGA prescription. However, we suspect that the effects attributable to physicians’ learning about SGA effectiveness still dominate the effects due to patients’ herd behavior. If the opposite is true, then we would not be able to detect negative peer effects from the inter-generational social learning. Second, the peer effect in physicians’ SGA prescription decisions is detected based on a generic definition of peer

groups. This can be an underestimate because the actual social learning network is unknown to us. And, there will be measurement errors in our defined peer groups, causing the attenuation bias in the peer effect estimate. Third, we do not have the data on acquisition prices paid by health care providers for drug purchases. We have the data only on prices reimbursed by the payer, which is the Bureau of National Health Insurance in Taiwan. It is possible that physicians choose certain drugs based on the markup, or the payer steers physicians away from certain drugs for cost reasons, which can induce similar drug prescription behaviors among physicians and thus bias our peer effect estimates upward.

TABLE A1
Peer Effect Estimates Based on Peers Formed on a Weekly Basis

	(1) FD Estimates	(2) Falsification Check	(3) FD Estimates of Lagged Effects	(4) Group Size Not Fixed	(5) Group Size Fixed	(6) Group Size ≤10	(7) Group Size >10
Δ Peers’ average SGA prescription share (t)	0.001 (0.003)		0.004 (0.004)	−0.002 (0.005)	0.002 (0.003)	0.001 (0.003)	0.001 (0.012)
Δ Peers’ average SGA prescription share ($t + 1$)		0.004 (0.003)					
Δ Peers’ average SGA prescription share ($t - 1$)			0.006 (0.005)				
Δ Peers’ average SGA prescription share ($t - 2$)			0.010* (0.005)				
Δ Peers’ average SGA prescription share ($t - 3$)			0.001 (0.005)				
Δ Peers’ average SGA prescription share ($t - 4$)			0.001 (0.004)				
Number of observations	296,595	97,729	59,739	123,464	173,131	213,650	82,945

Notes: Estimation results in columns (1)–(3) are based on Equations (3)–(5), respectively. Estimation results in columns (4)–(7) are based on Equation (3). Group size refers to the number of physicians in a focal physician’s peer group. ΔX represents the change in the value of variable X from period $t - 1$ to period t . Other control variables include the change in the number of physicians in a focal physician’s peer group (except in columns [4]–[7]), the change in the focal physician’s age, the change in the patient’s age, the change in the average price of drugs prescribed by the focal hospital–physician–patient pair, and the dummy variables for weekly fixed effects. Standard errors (reported in parentheses) are robust to the hospital–physician–patient level clustering in the conditional variance–covariance matrix of the regression disturbance term.

***Significant at the 1% level; **significant at the 5% level; *significant at the 10% level.

TABLE A2
Peer Effect Estimates Based on Peers Formed on a Quarterly Basis

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	FD Estimates Falsification Check		FD Estimates of Lagged Effects	Group Size Not Fixed	Group Size Fixed	Group Size ≤10	Group Size >10
Δ Peers' average SGA prescription share (<i>t</i>)	0.041*** (0.002)		0.049*** (0.003)	0.037*** (0.003)	0.042*** (0.003)	0.035*** (0.003)	0.076*** (0.006)
Δ Peers' average SGA prescription share (<i>t</i> +1)		-0.003 (0.003)					
Δ Peers' average SGA prescription share (<i>t</i> - 1)			0.040*** (0.004)				
Δ Peers' average SGA prescription share (<i>t</i> - 2)			0.032*** (0.004)				
Δ Peers' average SGA prescription share (<i>t</i> - 3)			0.017*** (0.003)				
Δ Peers' average SGA prescription share (<i>t</i> - 4)			0.008** (0.003)				
Number of observations	1,411,475	835,608	574,566	679,216	732,259	789,987	621,488

Notes: Estimation results in columns (1)–(3) are based on Equations (3)–(5), respectively. Estimation results in columns (4)–(7) are based on Equation (3). Group size refers to the number of physicians in a focal physician's peer group. Δ*X* represents the change in the value of variable *X* from period *t* - 1 to period *t*. Other control variables include the change in the number of physicians in a focal physician's peer group (except in columns [4]–[7]), the change in the focal physician's age, the change in the patient's age, the change in the average price of drugs prescribed by the focal hospital–physician–patient pair, and the dummy variables for quarterly fixed effects. Standard errors (reported in parentheses) are robust to the hospital–physician–patient level clustering in the conditional variance–covariance matrix of the regression disturbance term.

***Significant at the 1% level; **significant at the 5% level; *significant at the 10% level.

TABLE A3
Peer Effect Estimates Based on Peers Formed on a Monthly Basis

	(1)	(2)	(3)	(4)
Δ Peers' average SGA prescription share	0.007*** (0.001)			
Δ Peers' average SGA prescription share due to those who conducted the first month of work in their hospitals		0.003* (0.001)		0.003* (0.001)
Δ Peers' average SGA prescription share due to those who conducted the last month of work in their hospitals			-0.003* (0.001)	-0.003* (0.001)
Δ Number of physicians in a peer group	0.000** (0.000)	0.000* (0.000)	0.000** (0.000)	0.000* (0.000)
Δ Physician's age	0.029*** (0.005)	0.029*** (0.005)	0.029*** (0.005)	0.029*** (0.005)
Δ Patient's age	-0.000 (0.000)	-0.000 (0.000)	-0.000 (0.000)	-0.000 (0.000)
Δ Average price (NT\$1,000) of prescribed drugs	0.084*** (0.002)	0.084*** (0.002)	0.084*** (0.002)	0.084*** (0.002)
Number of observations	2,772,966	2,772,966	2,772,966	2,772,966

Notes: Estimation results in columns (1)–(4) are based on Equation (3). Δ*X* represents the change in the value of variable *X* from period *t* - 1 to period *t*. Other control variables include the dummy variables for monthly fixed effects. Standard errors (reported in parentheses) are robust to the hospital–physician–patient level clustering in the conditional variance–covariance matrix of the regression disturbance term.

***Significant at the 1% level; **significant at the 5% level; *significant at the 10% level.

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