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LEARNING DURING A CRISIS: THE SARS EPIDEMIC IN TAIWAN

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ABSTRACT

When SARS struck Taiwan in the spring of 2003, many people feared that the disease would spread through the healthcare system. As a result, outpatient medical visits fell by over 30 percent in the course of a few weeks. This paper examines how both public information (SARS incidence reports) and private information (the behavior and opinions of peers) contributed to this public reaction. We identify social learning through a difference-in-difference strategy that compares long time community residents to recent arrivals, who are less socially connected. We find that people learned from both public and private sources during SARS. In a dynamic simulation based on the regressions, social learning substantially magnifes the response to SARS.

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1 Introduction

The public periodically confronts a novel and unfamiliar threat, such as a terrorist attack or new disease outbreak. These situations typically spur people to take extreme protective actions such as avoiding public places, putting down livestock, or curtailing air travel. In such a crisis, a person must assess a new risk and decide how aggressively to protect himself. However, it is unclear how people make these decisions given the scarcity of information about the severity or prevalence of the threat.

The 2003 SARS epidemic in Taiwan allows us to study the response to an unfamiliar risk. SARS (severe acute respiratory syndrome) is a respiratory illness that resembles severe pneumonia and is transmitted through close interpersonal contact. SARS reached Taiwan from mainland China in March of 2003. 312 people were confirmed to be infected and 82 people died before the epidemic disappeared in July of that year. Despite the low prevalence of SARS in the general population, the public strongly eschewed restaurants, shopping centers, and other public places (Chou et al. 2004, Siu and Wong 2004). The high infection rate in hospitals also caused people to avoid the health care system: outpatient visits fell by 31 percent in April and May of 2003 (Hsieh et al. 2004). This drop occurred both in locations with and without SARS, and persisted for months after the epidemic had passed.

Health care avoidance during SARS is an example of a "prevalence response," which is a familiar topic in the literature on economic epidemiology (Ahituv et al. 1996, Gersovitz and Hammer 2003, Lakdawalla et al. 2006). Facing an increase in disease risk, people protect themselves and thereby limit the spread of infection. With few exceptions (de Paula et al. 2010, Gong 2010), this literature has assumed that decision makers possess complete information. Such an assumption is unrealistic for a disease outbreak. In even the most saturated media environment, public announcements only weakly indicate a person's idiosyncratic infection risk. Without a precise public signal, people may rely on private signals such as the opinions or actions of their peers. This mechanism may cause an "information cascade" that magnifies the response to an unfamiliar threat (Bikchandani et

al. 1992, Banerjee 1992, Welch 1992). If social learning is an important determinant of behavior in this setting, it may mediate the effectiveness of public policies to address a crisis.

This paper measures the contributions of public and private risk information to the SARS response. Reports of local and national SARS incidence provide public risk signals. We proxy for private risk signals using the change in health care utilization among peers from a pre-SARS baseline. We derive this proxy from a simple model of health care demand. A regression of individual medical visits on these variables distinguishes between the contributions of public and private information sources. Our analysis utilizes a nationally representative panel of medical claims of 1 million people (4.3 percent of Taiwan's population). This source allows us to quantify the number of outpatient visits by patient, provider and two-week period from 2001 to 2003 for a sub-sample of 29,619 people. We proxy for peer groups, which the claims data do not directly measure, using cohorts of patients who visit a common physician and facility.

Identifying social learning through a regression of individual outcomes on group outcomes is challenging because common unobservables jointly determine both variables (Manski 2000). Patients in the health care market may sort into peer groups because of common risk or health preferences that affect their response to SARS. Heterogeneous supply shocks, such as office closures by some doctors, may also induce a spurious correlation. Patients in the same peer group may also receive correlated signals of SARS risk if they obtain news from the same media sources. We address these concerns through a difference-in-difference design that compares the response of longtime community residents ("non-movers") to the response of recent arrivals ("movers") who are less socially connected. We find that social learning has an effect that is slightly smaller than the effect of national incidence reports.

The identifying assumption of this approach is that unobservable shocks to visits do not differentially affect non-movers during the SARS period. We evaluate this assumption through a complementary identification strategy. This approach addresses common unobservables by controlling for the current level of peer visits and uses variation in peer visits

from the previous year to identify effects. Conceptually, this approach compares individual responses in peer groups with the same levels of current visits and different levels of visits one year earlier. Fixing current visits, a peer group whose visits were previously high conveys a stronger signal of SARS risk than a group whose visits were previously low. This approach allows us to address the residual concern about the difference-in-difference regression that unobservable shocks specific to non-movers cause a spurious correlation. In a final falsification test, we apply our methodology to the annual drop in visits that occurs during Chinese New Year and find that social learning does not explain this phenomenon.

Our study contributes to the literature on economic epidemiology as well as the literature on social learning. In economic epidemiology, we provide the first examination of the individual behavioral response to a new outbreak and demonstrate that under incomplete information, the response elasticity varies by information source. Social learning may cause the public perception of disease prevalence to diverge from reality, with important implications for infection control. This paper also contributes to the literature on social learning by analyzing a novel context in which social learning is likely to be important. Several studies have considered learning in the context of technology adoption or consumption (Foster and Rosenzweig 1995, Munshi 2004, Conley and Udry 2010, Moretti 2010). The few studies that examine medical utilization do not consider the context of a health emergency. The extreme behavior that many people exhibit during an emergency suggests that people may process information differently in this setting.

This paper proceeds in Section 2 to develop a simple model that relates learning to our empirical approach. This framework motivates the use of the change in visits as a proxy for perceived risk and clarifies the relationship between regression coefficients and structural parameters. Section 3 describes the health care setting in Taiwan, the SARS epidemic, and the data set. Regression results appear in Section 4. Section 5 describes a dynamic simulation of the aggregate response to SARS. This exercise utilizes the regression estimates to illustrate the dynamic impact of social learning. Section 6 concludes.

2 Theory

In this section, we motivate our empirical approach with a theoretical framework that relates learning and health care utilization. We present a simple model of individual belief formation about SARS risk. We then incorporate the decision to seek health care and illustrate the conditions under which a person's observable change in medical visits over time proxies for his perception of SARS risk.

2.1 Learning About SARS Risk

People are indexed by i and belong to peer groups that are indexed by j and have size N_j. Each person decides whether to visit the doctor during period t. By visiting, the patient faces perceived risk $s_{ijt} \in [0,1]$ of contracting SARS and dying. Individuals learn about s_{jt} by observing realizations of this parameter from various data sources we describe below. People assume that SARS is distributed binomially in the population. This implies that the posterior probability of an individual catching SARS is a linear function of the means from the different sources of information (Jewell 1974).

Three data sources provide individuals information on SARS risk. First, the government draws a sample from the distribution of SARS. Specifically it tracks new SARS cases and reports the mean SARS incidence, $s^c_{jt} \in [0,1]$. This is a common public signal of SARS risk. Second, people obtain an independent, private estimate of SARS risk, $s^p_{ijt} \in [0,1]$. The independent private estimate reflects personal risk factors, including frequency of contact with others and use of a mask outdoors.[1] Third, each individual samples his peers' private estimates of SARS risk, $\{s^p_{\neg ijt}\}$.[2]

Using these three data sources, individuals update their beliefs using Bayes theorem. A binomial distribution for SARS implies that person i's posterior on his own probability of

[1] Although the private draw is independent of the common signal, the individual's personal posterior will depend on the common signal as well.

[2] People may sample peers' beliefs by communicating directly or by observing behaviors like the change in health care utilization, which indicate beliefs. Ellison and Fudenberg (1995) and Offerman and Schotter (2009) develop models of social learning from peer behavior.

catching SARS is a linear weighted average of the means from each independent data source.

$$s_{ijt} = \phi_{1i} s^c_{jt} + \phi_{2i} s^p_{ijt} + \Sigma_{l \in I_j, l \neq i} \phi_{3il} \bar{s}^p_{ljt} + \phi_{4i} s_{ijt-1}$$

In this expression, I_j is the set of individuals in peer group j. The weights, ϕ, are increasing in the reliability and precision of the signal (**?**).

Our empirical objective is to test whether individual posterior beliefs on risk actually give positive weight to the common signal or peers' independent private signals. For tractability, we make two assumptions about these weights. First, we assume that individuals give identical weight to the private information from different peers, i.e., $\phi_{3il} = \phi_{3i}$. Second, we assume that all individuals place the same weight on each information source, i.e., $\phi_{mi} = \phi_m$ for $m \in \{1, 2, 3\}$.[3] Given these assumptions, we can represent each agent's learning process with the following linear function.

$$s_{ijt} = \phi_1 s^c_{jt} + \phi_2 s^{p*}_{ijt} + \phi_3 \bar{s}^{p*}_{\neg ijt} + \phi_4 s_{ijt-1} \tag{1}$$

where $\bar{s}^{p*}_{\neg ijt} = \sum_{l \in I_j, l \neq i} s^{p*}_{ljt} / (N_j - 1)$ is the average private signal of peers. People learn from the common signal if $\phi_1 > 0$, from their own independent private signal if $\phi_2 > 0$, and from the independent private signals of peers if $\phi_3 > 0$.

If each of these signals were observable, a regression based on equation (1) would identify these weights. Our primary empirical challenge is that the private signals of person i and his peers, s^p_{ijt} and $\bar{s}^p_{\neg ijt}$, are unobservable. We propose to address the inability to observe the private signal of peers, $\bar{s}^p_{\neg ijt}$, by replacing it with the average posterior risk perception of peers, $\bar{s}_{\neg ijt} = \sum_{l \in I, l \neq i} s_{ljt} / (N_j - 1)$, for which we will propose a proxy in the next section.

[3] This assumption is stronger than necessary. It would be sufficient if the weight placed placed by person i on source m had the structure $\phi_{mi} = \phi_m + u_{mi}$ where u_{mi} is mean independent of the SARS risk estimated from source m. Under this assumption, the weights function as coefficients in a random coefficients regression model. The mean independence assumption is reasonable for our data. The main source of variation in individual weights is the size of person i's peer group. The correlation between our proxy for the risk perceptions of peers (developed in the next section) and group size is very low ($\rho = -0.03$).

The average posterior on SARS risk among peers is

$$\bar{s}_{\neg ijt} = \phi_1 s^c_{jt} + \phi_2 \bar{s}^{p*}_{\neg ijt} + \phi_3 \left(\frac{\sum_{l \in I, l \neq i} \bar{s}^p_{\neg ljt}}{N_j - 1} \right) + \phi_4 \frac{\sum_{l \in I, l \neq i} s_{ljt-1}}{N_j - 1} \tag{2}$$

The first two terms of this expression have the same interpretation as in equation (1). The third term captures social learning by peers from other peers, including learning from person i. This feedback complicates substitution of overall peer beliefs for peers' private signals in equation (1).

In order to simplify expressions (1) and (2), we assume that N_j is large.[4] In small groups, individual risk perceptions can have a meaningful impact on average group risk perceptions. This impact vanishes as group size increases: $\lim_{N_j \to \infty} \bar{s}^p_{\neg ijt} = \bar{s}^p_{jt}$. Under this assumption, the individual and group risk perceptions become:

$$s_{ijt} \approx \phi_1 s^c_{jt} + \phi_2 s^p_{ijt} + \phi_3 \bar{s}^p_{jt} + w_{ijt} \tag{3}$$

$$\bar{s}_{\neg ijt} \approx \phi_1 s^c_{jt} + (\phi_2 + \phi_3) \bar{s}^p_{jt} + \bar{w}_{\neg ijt} \tag{4}$$

where $w_{ijt} = \phi_4 s_{ijt-1}$. We obtain an expression for individual beliefs in terms of the average signal of peers by isolating $\bar{s}^p_{jt}$ in equation (4) and substituting into equation (3).

$$s_{ijt} \approx \left[\frac{\phi_1 \phi_2}{\phi_2 + \phi_3} \right] s^c_{jt} + \phi_2 s^p_{ijt} + \left[\frac{\phi_3}{\phi_2 + \phi_3} \right] \bar{s}_{\neg ijt} + u_{ijt} \tag{5}$$

The equation above still cannot be estimated because we do not observe person i's independent private information. In its place, we propose a hypothetical regression of the individual's risk perception on the common signal and the group risk perception.

$$s_{ijt} = \alpha_0 + \alpha_1 s^c_{jt} + \alpha_2 \bar{s}_{\neg ijt} + u_{ijt} \tag{6}$$

[4] A large N_j assumption is reasonable because the median peer group size is 55 in our data. Limiting the sample to only large networks, for which this assumption is most valid, does not affect our results.

Person i's private signal, s^p_{ijt}, appears in equation (5) but is an omitted variable in equation (6). This omission does not bias the estimate of α_1 however, because the individual's private signal is independent of the common signal. Nor does the omission bias α_2 because independence also ensures that s^p_{ijt} is uncorrelated with $\bar{s}_{\neg ijt}$. For large values of N_j, $corr(s^p_{ijt}, \bar{s}_{\neg ijt}) \rightarrow corr(s^p_{ijt}, \bar{s}^p_{\neg ijt})$, which is zero.

2.2 The Change in Visits: a Proxy for Perceived Risk

To estimate regression (6), we must either observe or proxy for individual and group risk perceptions s_{ijt} and $\bar{s}_{\neg ijt}$. Here we motivate the use of the change in medical visits over time as a proxy for perceived risk and explain what a regression employing this proxy reveals about the structural parameters.

When deciding whether to see the doctor, a person compares his level of illness to his perceived cost of a visit. In general, this cost includes the copayment (which is less than U.S. $5 per visit) and the cost of transportation to the medical facility. During SARS, the cost also includes the risk of contracting SARS during the visit. Moreover, holding illness and other costs constant, a change in visits indicates a change in the perceived SARS risk.

More formally, people receive utility from health and other consumption, h_{ijt} and m_{ijt}, respectively. In each period, people experience a health shock, $d_{ijt} \geq 0$, and must decide whether to seek medical care, $v_{ijt} \in \{0, 1\}$. A visit to the doctor restores the patient to his baseline health, but requires him to pay a copayment c_t. During the SARS epidemic, people also face the risk that a visit may cause them to catch SARS and die. After normalizing the utility from death to be zero, the expected utility from visiting and not visiting the doctor

are:[5]

$$EU[v_{ijt} = 1] = (1 - s_{ijt})u(h_{ijt}, m_{ijt} - c_t) \tag{7}$$

$$EU[v_{ijt} = 0] = u(h_{ijt} - d_{ijt}, m_{ijt}) \tag{8}$$

A person seeks care if the value of alleviating his illness exceeds the cost of treatment: $EU[v_{ijt} = 1] > EU[v_{ijt} = 0]$. Taking logs yields to the following equivalent expression.

$$\ln(1 - s_{ijt}) + \ln u(h_{ijt}, m_{ijt} - c_t) - \ln u(h_{ijt} - d_{ijt}, m_{ijt}) > 0$$

In this formulation, the probability of a visit depends upon the person's health status and his perceived SARS risk.

An observer wishing to interpret the change in visits as an indicator of perceived risk must account for secular trends in health. To satisfy this requirement, we assume that the difference in log utility from seeking care rather than not seeking care is a trend-stationary function of the person's age: $\ln u(h_{ijt}, m_{ijt} - c_t) - \ln u(h_{ijt} - d_{ijt}, m_{ijt}) = \mu_{ij} + g a_{ijt} - e_{ijt}$, where a_{ijt} is the person's age.[6] The error term, e_{ijt}, is identically and independently distributed throughout the population, with mean zero, cumulative distribution $F(e)$, and density $f(e)$. Under this formulation, people experience idiosyncratic health shocks with a mean that linearly increases with age. The first expression below shows the probability of a visit under these assumptions. In the second expression, we apply a first-order Taylor-series expansion at a suitable common point, $\bar{e}$, in the distribution of e_{ijt}, and incorporate the approximation

[5] Without loss of generality, we ignore the dynamic effects of current health decisions. Our approach can incorporate these effects by reinterpreting the contemporaneous utility function as a value function that embeds future optimizing behavior.

[6] Under the assumption of a quadratic trend, equations (11) and (12) become non-linear functions of age.

that $\ln(1 - s_{ijt}) \approx -s_{ijt}$ for small values of s_{ijt}.

$$pr[v_{ijt} = 1] = F[\ln(1 - s_{ijt}) + \mu_{ij} + ga_{ijt}] \tag{9}$$

$$\approx F(\bar{e}) - f(\bar{e})[s_{ijt} - \mu_{ij} - ga_{ijt} + \bar{e}] \tag{10}$$

The probability of a visit is thus an affine transformation of SARS risk, the idiosyncratic health endowment, and age.

The first difference of this probability is our proxy for an individual's perceived SARS risk. We subtract the number of visits during a comparable pre-SARS period (when $s_{ijt} = 0$) from the number of visits in the index period. The change in visits, $\Delta v_{ijt} = v_{ijt} - v_{ijt-k}$, nets out the time-constant health endowment and $F(\bar{e})$. Likewise, the change in average peer visits, $\Delta \bar{v}_{\neg ijt} = \bar{v}_{\neg ijt} - \bar{v}_{\neg ijt-k}$, proxies for the average risk perception of the peer group.

$$E[\Delta v_{ijt}] \approx f(\bar{e})gk - f(\bar{e})s_{ijt} \tag{11}$$

$$E[\Delta \bar{v}_{\neg ijt}] \approx f(\bar{e})gk - f(\bar{e})\bar{s}_{\neg ijt} \tag{12}$$

A person who is familiar with $f(\bar{e})$ and the effect of age on health, g, can infer s_{ijt} and $\bar{s}_{\neg ijt}$ from the change in individual and group visits, respectively.

Our empirical strategy uses these proxies to estimate a version of the hypothetical regression in (6):

$$\Delta v_{ijt} = \beta_0 + \beta_1 s^c_{jt} + \beta_2 \Delta \bar{v}_{\neg ijt} + \eta_{ijt} \tag{13}$$

By substituting in the expressions for Δv_{ijt}, $\Delta \bar{v}_{ijt}$, s_{ijt} and $\bar{s}_{\neg ijt}$, we find that $\hat{\beta}_1$ and $\hat{\beta}_2$ have the following structural interpretations.

$$E[\hat{\beta}_1] = -f(\bar{e})\left[\frac{\phi_1\phi_2}{\phi_2 + \phi_3}\right] \tag{14}$$

$$E[\hat{\beta}_2] = \frac{\phi_3}{\phi_2 + \phi_3} \tag{15}$$

Although the system (which also includes an expression for $E[\hat{\beta}_0]$) is not identified, the coefficient estimates test whether people learn from public information and from peers. A significant value of $\hat{\beta}_1$ indicates that people learn from public information, while a significant value of $\hat{\beta}_2$ indicates that people learn from their peers. The signs on these coefficients differ because an increase in $\Delta\bar{v}_{\neg ijt}$ indicates less risk while an increase in s^c_{jt} indicates greater risk. In equation (15), $\hat{\beta}_2$ provides the contribution of social learning relative to the combined contribution of social learning and the individual private signal. A coefficient estimate that is significantly less than 1 indicates that people also respond to their own private information.

Equation (14) also shows that $\hat{\beta}_1$ underestimates the response to public information because $\phi_2/(\phi_2 + \phi_3) < 1$. A complementary regression of the change in individual visits on just the common signal, however, eliminates this source of attenuation.

$$\Delta v_{ijt} = \beta_3 + \beta_4 s^c_{jt} + \omega_{ijt} \tag{16}$$

Because information sources are orthogonal in equation (1), excluding $\Delta\bar{v}_{\neg ijt}$ from this regression does not cause omitted variables bias. The coefficient on the common signal has the following structural interpretation:

$$E[\hat{\beta}_4] = -f(\bar{e})\phi_1$$

Using this coefficient estimate rather than $\hat{\beta}_1$ leads to a larger and more accurate estimate of learning from public information. A regression based on equation (16) will prove useful for the dynamic simulation in Section 5, where it serves as a predictive model that better captures the response to public information.

3 Context and Data

3.1 The SARS Epidemic in Taiwan

Taiwan is a densely populated island located near mainland China. The country has a population of 23.1 million and income per capita of around $31,000. Modern highways and railways facilitate intercity travel. Taiwan is made up of 25 counties and cities, which further subdivide into 368 townships and urban districts (hereafter labeled "counties" and "townships" respectively). The population has a median age of 37 and a life expectancy of 78. Chinese New Year, which occurs on a lunar schedule in January or February, is an important holiday that causes a large decline in medical visits. During the two-week holiday, many families travel to visit relatives and some medical offices close. This holiday has a large impact on health care utilization in the figures below.

In 1996, Taiwan implemented a universal fee-for-service health care system (Cheng 2003). Under the system, patients contribute modest copayments of US$5 or less for visits, tests, and prescriptions. The Bureau of National Health Insurance (BNHI) administers the system and reimburses providers for most expenses. People may obtain outpatient care from either hospital outpatient departments or small storefront clinics. Clinics, which are ubiquitous in cities, serve around 70 percent of the outpatient market. With such low copayments, many patients prefer to visit the doctor (and obtain medicine) for minor illnesses such as sore throats and colds. These conditions, classified broadly as "upper respiratory infections," constitute 38 percent of all outpatient visits. The low out-of-pocket cost has led to intense health care utilization, with patients seeking care a median of 10 times per year.

SARS is a respiratory illness that resembles severe pneumonia. The disease is caused by a coronavirus and is transmitted through close contact with an infected person. The SARS epidemic originated in Guandong, China in November of 2002 and soon spread to Hong Kong, Southeast Asia, and Canada. Taiwan's first SARS case occurred in a traveler who became ill on March 14, 2003 after arriving from mainland China. The epidemic escalated

on April 22 when an indigenous outbreak among patients and hospital staff at the Ho-Ping Hospital in Taipei led to several secondary outbreaks in other major cities. Figure **??** plots the number of reported and probable SARS cases (explained below) by two-week period to show the progression of the epidemic. The SARS epidemic lasted through June, leading to a total of 312 confirmed infections and 82 deaths. At the peak of the epidemic, SARS infected 60 and killed 6 people per day. Nevertheless, the overall burden of SARS was only 1.4 confirmed cases and 0.36 deaths per 100,000 people.

The Ho-Ping Outbreak, which took place during Period 9 in the figure, led to widespread panic. According to Ko et al. (2006, p. 398), "People started to hoard all possible protective equipment, and reject people or materials with any risk of infection, including infected patients, the families of patients, subjects quarantined, and even health providers." Domestic air travel fell by 30 percent and international air travel fell by 58 percent from 2002 levels (National Policy Foundation 2003). The price of Isatidis Radix, a traditional Chinese antiviral remedy, rose by 800 percent (Huang 2003).

The SARS epidemic also had a large impact on health care utilization. Figure **??** plots the nationwide volume of outpatient visits by two-week period in 2001, 2002, and 2003. In a sharp deviation from the usual seasonal pattern, visits fell by over 30 percent from March to June of 2003. Visits did not return to the pre-SARS level until September of that year, three months after the last probable SARS case on June 16. Based on the number of SARS deaths and outpatient visits from March-June of 2003, SARS created a mortality risk of at most 0.0000007 deaths per visit. Using an upper-bound estimate of $2.2 million for the value of statistical life (Hammitt and Liu 2004), the risk of SARS death during a medical visit raised the expected price of a visit by $1.93. However the decline in visits during SARS is consistent with a much larger perceived cost. After a copayment increase of $3 in November of 2002, visits to medical centers fell by 3 percent. Benchmarking the SARS response by this copayment response, people behaved as if SARS had increased the price of a visit by

$17.60.[7]

The response to SARS occurred both in townships with and without actual SARS incidence. Figure **??** plots total visits, comparing townships with zero and positive SARS incidence. The response to SARS is only slightly larger in townships that actually experienced the outbreak. The timing and magnitude of the SARS response also depended on the nature of the visit. Figure **??** categorizes visits as respiratory, critical, chronic, or other.[8] Although utilization fell in all categories, the response of respiratory visits was particularly sharp and extended. These visits fell by over 50 percent and remained suppressed through the end of the year. Although respiratory visits are distinct in several aspects, the low marginal benefit of a respiratory visit is the most likely explanation for this pattern.[9]

3.2 Signals of Risk

Under incomplete information, a decision maker may seek new information sources and tailor his response to a signal's credibility and precision. Common signals of risk, such as public announcements of disease incidence, convey the average risk in a population. However these signals may provide little information about idiosyncratic risk, which depends upon a person's behavior and social interactions. Common signals are especially noisy during a new disease outbreak, when even experts do not fully understand the disease's severity or mode of transmission.

Data on SARS incidence from the Taiwan Centers for Disease Control (TCDC) represent a common signal of SARS risk. The agency released these reports daily to intense media coverage. The front page of the Apple Daily News on May 22, 2003 in Figure 1 exemplifies

[7] The mortality risk calculation assumes conservatively that all SARS deaths arise because of outpatient visits. Consistent with the extreme response, Liu et al. (2005) find that the VSL associated with avoiding SARS risk is several times greater than conventional VSL measurements from Taiwan.

[8] Critical visits include visits related to pregnancy, abortion, injury, appendicitis, stroke, heart attack, and internal bleeding. Chronic visits include visits related to dialysis, chemotherapy, diabetes, and liver or kidney failure.

[9] Patients with mild respiratory illnesses may have also feared that doctors would place them in quarantine (Hsieh et al. 2005). As a respiratory condition, SARS could also have increased respiratory visits among people concerned about possible exposure.

the print coverage of SARS. The lead story describes a restriction on travel out of Taiwan. On the left, a map shows the cumulative number of SARS cases by county, and a table summarizes the number of cases and deaths nationwide. Although both local and national incidence contain information, national incidence may provide a more meaningful signal in a small country like Taiwan.

Without precise objective information, people may turn to private signals such as the perceptions or behavior of their peers. As our model shows, the change in visits from an earlier (risk-free) period indicates a person's risk perception. This signal is noisy for a particular individual because health varies idiosyncratically: a decline in visits during SARS could merely indicate the absence of a prior illness. Aggregation within a group reduces the idiosyncratic noise in this signal.

Consistent with an increase in social learning, inter-group variation in the frequency of visits increased during SARS (Glaeser et al. 1996, Graham 2008). Figure **??** plots the coefficient of variation (CV) in visits by two-week period, distinguishing between variation within and across peer groups.[10] In a pattern specific to 2003, inter-group variation rose dramatically during SARS while intra-group variation remained flat. The reader should interpret the increase in dispersion cautiously since a decline in the mean of visits may mechanically inflate the CV. However the CV only increases slightly during Chinese New Year (Period 3 of 2003), despite an even larger decline in visits at that time.

3.3 Data

Our primary data source is a large panel of medical claims furnished by the BNHI. The data set contains all outpatient visits from 1997 to 2003 for a representative sample of one million people (4.3 percent of Taiwan's population). We obtain a manageable regression data set by drawing a random 6 percent subsample through the procedure below. For each individual $\times$ peer group, the regression data set contains 78 biweekly observations from 2001 to 2003.

[10] Because visits are bounded by zero, the decline in visits mechanically reduces the standard deviation. The coefficient of variation partially corrects for this issue.

The dependent variable is the number of outpatient visits by a patient to the doctor who defines a particular peer group.

The patient's actual peer group–his family, friends, and neighbors–is unobservable. We proxy for peer groups using cohorts of patients who visit the same physician and medical facility from 2001 to 2003. Using this measure, 93.1 percent of the population belongs to at least one peer group and the median number of peer groups is seven. Quartiles of the group size distribution occur at 12, 55, and 204 people. A peer group definition based on common health care utilization is sensible for two reasons. People typically seek outpatient care for mild conditions and are unwilling to travel far outside the community. Outpatient health care markets in Taiwan are highly localized and many neighbors visit the same physician. The referral process also leads to social ties among patients of the same doctor. Because most patients select a physician through a friend's referral, patients of a common physician often share a direct or indirect acquaintance (Hoerger and Howard 1995, Tu and Lauer 2008).[11]

Noise in the definition of a peer group is a common issue that does not ordinarily interfere with the identification of social interactions as long as the true social network overlaps with the proxy (Blume et al. 2011). Misspecification of peer groups most likely (though not necessarily) causes attenuation bias through the same mechanism as classical measurement error. In Section 4, we show that results are robust to defining peer groups by facility, township, or county. Results are also similar if a person must visit twice from 2001-2003 rather than just once in order to count as a group member.[12]

The one-year change in average visits of peers proxies for the group's perception of SARS risk. $\bar{v}_{\neg ijt}$ denotes the average number of visits in group j, excluding the index person. This variable is the sum over periods $t-2$ to t, allowing it to reflect SARS risk information from the recent past. The change in peer visits, $\Delta\bar{v}_{\neg ijt}$ is the difference in $\bar{v}_{\neg ijt}$ from the same

[11] Published evidence of this phenomenon from countries other than the US is extremely limited. Anecdotally, referrals are especially important in Taiwan because there few institutional restrictions (such as HMO networks) on the choice of physician.

[12] If group membership requires two rather than one visit, then 85 percent of the population belongs to at least one group and the median number of groups per person is 4. Quartiles of the group size distribution occur at 6, 29, and 115 people.

two-week period in the previous year: $\Delta\bar{v}_{\neg ijt} \equiv \bar{v}_{\neg ijt} - \bar{v}_{\neg ijt-26}$. The lagged component of $\Delta\bar{v}_{\neg ijt}$ always captures pre-SARS utilization because SARS lasted for less than a year. While the duration of the difference is arbitrary, a one-year difference implicitly removes seasonality from the regressor. Regressions in which $\Delta\bar{v}_{\neg ijt}$ is constructed as a six-month difference lead to similar results.

Our identification strategy distinguishes between longtime community residents ("non-movers") and people who have recently joined the community ("movers").[13] Recent arrivals have weaker ties to their peers because people establish social connections over time (Jackson 2009). To identify movers, we first calculate the overlap in outpatient traffic between all pairwise combinations of townships. Next we determine each patient's modal township by year and define a move as a transition across townships with low overlap.[14] This process allows us to classify people by tenure status in the community, which ranges from 1 to ≥ 7 years. Movers are defined as people who join their 2003 township in 2001 or later, so that they either one or two years of tenure. Under this definition, movers make up 5.6 percent of the population, people with tenure of 3-6 years make up 6.6 percent of the population, and people with tenure of ≥ 7 years make up 87.8 percent of the population.

Our sampling procedure is designed to increase statistical power by oversampling people with tenure below 7 years and balancing the sample of movers and non-movers within each peer group. We begin by discarding peer groups that contain only movers or only non-movers (2 percent of all observations). For the remaining peer groups, we draw up to four people each who have tenure of 1, 2, or ≥ 7 years. This step increases the proportion of movers and reduces the proportion of people with tenure of ≥ 7 years in the sample relative to the population. We also increase the representation of people with 3, 4, 5, or 6 years of tenure by

[13] The identification strategy exploits heterogenous exposure to social interactions among subsets of the peer group. Cohen-Cole (2006) and Blume et al. (2011, Theorem 2) derive the conditions under which this approach is valid. Despite the superficial similarity, this strategy is distinct from Gaviria and Raphael (2001), who test the endogeneity of school choice by comparing movers and non-movers.

[14] As a baseline, townships have low overlap if they fall below the fifth percentile of the overlap distribution, so that less than 0.17 percent of patients visit doctors in both townships. As we show below, results are robust to using the tenth percentile of overlap (0.92 percent) as an alternative cutoff.

drawing up to two people from each group. The procedure yields a regression data set with 38.8 percent movers and an adequate sample of people with tenure of 3-6 years. Regressions use probability weights to restore the population proportions of these groups.

Table **??** compares the characteristics of movers and non-movers. The large sample ensures that many small differences between movers and non-movers are statistically significant. In Panel A, non-movers average 0.045 visits per period to a particular physician $\times$ facility, while movers average 0.035 visits per period. Both movers and non-movers are diagnosed with respiratory infections (e.g. a sore throat or cold) in around 40 percent of visits. Movers and non-movers have similar characteristics, although movers are younger and more likely to be male. Movers earn US$30 less per month than non-movers.[15] Panel B summarizes the characteristics of peer groups. Peer characteristics are balanced because movers and non-movers are sampled in approximately fixed proportions within groups.

To investigate homophily within peer groups, Table **??** reports the correlation between individual characteristics and the group means of these characteristics (excluding the index person). Among patients of a common physician $\times$ facility in Column 1, these correlations are 0.42, 0.66, and 0.30 for gender, age, and income respectively. The correlation is also high for the number of peer groups per patient, the annual number of visits per patient, and the location of the peer group in the patient's modal township. In Columns 4-6, the correlation falls monotonically as the peer group broadens to the facility, township, or county. A table of intraclass correlation coefficients (available from the authors) shows the same pattern.

Columns 2 and 3 of Table **??** show that movers and non-movers exhibit a similar degree of homophily with their peers. The correlation with the group mean is comparable across movers and non-movers for income, the number of peer groups per patient, and the location of the peer group in the patient's modal township. Movers have a higher correlation with the group for gender but a lower correlation for age.

The Taiwan CDC provides data on the incidence of "reported" and "probable" SARS

[15] Income data based on BNHI estimates of earnings by occupation category are available for 62% of the sample.

cases. A reported case is any case that the TCDC investigates as a possible SARS infection. A probable case is a reported case that also (1) exhibits high fever and difficulty breathing, (2) an epidemiological link to other SARS cases, and (3) radiographic evidence of pneumonia or respiratory distress syndrome or a positive assay for the SARS coronavirus (WHO 2003).[16] To express SARS incidence, s, as an infection probability, we compute the number of cases per 100 people. Regressions also utilize a SARS period indicator, which equals 1 for Quarters 2-4 of 2003. As with $\Delta\bar{v}_{\neg ijkt}$, SARS incidence is a sum over periods $t-2$ to t.

4 Estimation

4.1 Empirical Approach

In this section, we estimate the response to public and peers' private information about risk. In a difference-in-difference style specification, social learning is the differential effect of $\Delta\bar{v}_{\neg ijkt}$ for non-movers during SARS.

$$\begin{aligned} v_{ijkt} = {} & \beta_1 s^l_{kt} + \beta_2 s^n_t + \beta_3 S_t N_i \Delta\bar{v}_{\neg ijkt} + \beta_4 v_{ijkt-26} \\ & + [\text{levels and pairwise interactions of } S_t, N_i, \text{ and } \Delta\bar{v}_{\neg ijkt}] \\ & + \alpha_{jk} + \delta_t + \epsilon_{ijkt} \end{aligned} \tag{17}$$

In this specification, i indexes the patient, j indexes the physician $\times$ facility, k indexes the township, and t indexes the two-week period. The dependent variable, v_{ijkt} is the number of outpatient visits. Consistent with the interpretation of Δv_{ijkt} as the patient's risk perception, the regression controls for the one-year lag of the dependent variable, $v_{ijkt-26}$.[17] The

[16] Confirmatory diagnostic tests for SARS did not become available until midway through the epidemic. Even once these tests arrived, authorities did not provide immediate confirmation of SARS infection. Therefore, people did not generally have information about confirmed SARS incidence.

[17] We control for $v_{ijkt-26}$ rather than use Δv_{ijkt} as the dependent variable in order to avoid endogeneity due to serial correlation in individual risk perceptions. If perceptions are serially correlated, then lags of v_{ijkt} belong as controls in the specification. However, these lags are functionally dependent upon $v_{ijkt-26}$. Regressing on $v_{ijkt-26}$ is the most direct solution to this problem.

specification includes the number of local and national SARS cases per 100 people, s^l_{kt} and s^n_t, and the one-year change in peer visits, $\Delta\bar{v}_{\neg ijkt}$. Local cases are calculated by township. S_t identifies the SARS period (Quarters 2-4 of 2003) and N_i identifies non-movers.

A peer group fixed effect, α_{jk}, controls for time constant attributes of the peer group, allowing the regression to compare groups with similar levels of utilization.[18] A time fixed effect, δ_t, controls for systematic time variation in visits. Because s^n_t and δ_t are collinear, specifications that include s^n_t utilize separate period and year (rather than period $\times$ year) fixed effects. We estimate the model using OLS and cluster standard errors by the modal townships of patients. The regressions employ probability weights to restore the population proportion of movers and weight patients equally. Negative signs for $\hat{\beta}_1$ and $\hat{\beta}_2$ indicate avoidance of SARS risk based on public information. A positive sign for $\hat{\beta}_3$ indicates a response to the risk perceptions of peers.

The correlation between v_{ijkt} and $\Delta\bar{v}_{\neg ijkt}$ may arise because common unobservables jointly influence individual and group behavior (Manski 1993, Manski 2000). The SARS period interaction ensures that any confounder must exhibit a differentially strong influence during SARS to threaten identification. One specific concern is that heterogeneous supply shocks during SARS may induce a correlation between the visits of group members. Another possibility is that patients and their peers, having self-selected into the same group, may share common traits such as risk aversion that affect the SARS response. Group members may also receive correlated risk signals that cause them to exhibit similar responses.[19]

Our approach addresses these concerns by treating movers as a control group. By exploiting the interaction between S_t, N_i, and $\Delta\bar{v}_{\neg ijkt}$, the regression differences out common unobservables that are constant among movers and non-movers. The identifying assumption

[18] The use of a peer group fixed effect leads to bias in the coefficient on the lagged dependent variable. However, Hsiao (2003, p. 72) notes that the bias vanishes as $T \to \infty$. With 78 time periods, this setting features an unusually long panel. Moreover, bias in β_4 is unlikely to contaminate the other coefficients: the pairwise correlations of $v_{ijkt-26}$ with s^l, s^n, and $S_t N_i \Delta\bar{v}_{\neg ijkt}$ are 0.002, 0.007, and -0.021 respectively.

[19] Our results are consistent with imitation of peers as well as social learning. Imitation of informed people by uninformed people is a form of social learning that is consistent with our model (Apesteguia et al. 2007). People may also imitate their peers because they simply prefer homophily. It is unclear why an emergency would heighten the preference for conformity in the absence of a learning mechanism.

of this regression is that common unobservables with a differential impact during SARS apply equally to movers and non-movers. As part of this analysis, we implement a specification with peer group $\times$ year $\times$ period fixed effects. The fixed effects in this specification control for all group-specific shocks that are common to both movers and non-movers. The difference within a peer group between the responses of movers and non-movers is the only remaining source of identifying variation. For social learning to arise spuriously, non-movers must receive a targeted shock that does not affect movers.

We evaluate the possibility of non-mover specific shocks by implementing a complementary identification strategy. Under this approach, we control for common unobservables by conditioning on $\bar{v}_{\neg ijkt}$ in a regression of v_{ijkt} on $\Delta\bar{v}_{\neg ijkt}$. This regression is identified through the negative correlation between v_{ijkt} and $\bar{v}_{\neg ijkt-26}$, as we explain further below. This framework allows us to control for non-mover specific shocks by controlling separately for the current visits of mover and non-mover peers.

4.2 Baseline Results

Results based on specification (17) appear in Table **??**. Columns 1 and 3 leave aside social learning and show the response to local and national SARS incidence. These estimates show a relatively small response to local SARS incidence. The response elasticity to local incidence ranges from -0.0006 for probable cases to -0.0011 for reported cases. The response elasticity to national incidence is several times larger larger: -0.0050 for reported cases and -0.0066 for probable cases.[20]

Columns 2 and 4 add subjective peer assessments by incorporating $S_t N_i \Delta\bar{v}_{\neg ijkt}$ and the related pairwise interactions. The coefficient estimate is statistically significant and implies a response elasticity of 0.0048. Perceiving a higher SARS risk, patients who observe a decline in peer visits also visit less often. Accounting for social learning and unobservable shocks in this way attenuates the local incidence response by 62-68 percent and the national incidence

[20] Regressions that also include county-level incidence (available from the authors) show a small and insignificant response to county-level information.

response by 27-37 percent. Figure **??** plots the response elasticity by information source. Information from peers and national incidence have comparable effects, while local incidence has almost no effect. Although the figure compares effect sizes, the model makes clear that these effects do not map directly into structural learning parameters.

The specifications in Table **??** evaluate the robustness of the social learning result. We replace the SARS incidence variables with comprehensive time fixed effects. Although the table does not report the coefficients, these regressions also include all levels and pairwise interactions of $S_t N_i \bar{v}_{\neg ijkt}$, as well as the one-year lag of individual visits. Column 1 shows the baseline estimate, which is slightly larger than the estimates in Table **??**. Columns 2 and 3 incorporate peer group $\times$ SARS and peer group $\times$ time fixed effects respectively. These more restrictive specifications only slightly attenuate the social learning estimate. Column 3 is identified exclusively through the difference between the responses of movers and non-movers within a common peer group. For these results to be spurious, non-movers must experience differentially strong unobservable shocks during SARS.

Columns 4-8 of Table **??** show that the social learning estimate is robust under several alternative formulations. In Column 4, people must visit a doctor $\times$ facility twice during 2001-2003, rather than once, in order to belong to a peer group. In Column 5, which broadens the definition of movers, people move if they transition across townships with overlap below the 10th percentile of the overlap distribution, rather than the 5th percentile. Columns 6-8 define peer groups by facility, township, or county. Defining the facility as the peer group leads to the largest social learning estimate. After that point, expanding the peer group to include the entire township or county causes the estimate to decline.[21]

In the preceding estimates, a mover is defined as someone with tenure in the community

[21] Our discussion thus far has analyzed the effect of a signal from peer group j on an individual's visits to the doctor $\times$ facility associated with group j. In reality, people may also learn about SARS risk from their peers in other peer groups. Patients in our data belong to a median of 7 peer groups. To construct $\Delta \bar{v}_{\neg ijkt}$ across an individual's other peer groups, we compute the total number of peer visits in an individual's other groups and divide by the total population of these groups (excluding the index patient). Augmenting the baseline regression to include the signal from other peer groups leads to significant effects of both group j and other peer groups.

of 1 or 2 years in 2003. We test the sensitivity our results to this definition by interacting $S_t \Delta \bar{v}_{\neg ijkt}$ with indicators for each tenure value from 1 to $\geq$ 7 years. Figure **??** plots the coefficients and confidence intervals from this regression. With the exception of years 3 and 4, the response generally rises with tenure in the community. The greatest response occurs among people who have resided in the community for seven or more years.

Table **??** investigates the timing of the SARS response by category of diagnosis. Instead of treating Quarters 2-4 as a common SARS period, these regressions interact $N_i \Delta \bar{v}_{\neg ijkt}$ with quarter-of-2003 dummies. Column 1 shows that across all diagnoses, the social learning effect is greatest in Quarter 2, followed by Quarter 4. While Quarter 2 coincides with the peak of the epidemic, the result for Quarter 4 is initially surprising because visits fully resumed by the end of Quarter 3. Distinguishing among diagnoses helps to explain this finding. In Columns 2-5, the social learning estimate is particularly strong for respiratory infections but is virtually absent for critical or chronic illnesses. As Figure **??** highlights, the SARS response for respiratory visits lasted through the end of Quarter 4.

4.3 A Complementary Identification Strategy

In this section, we corroborate our results through an alternative identification strategy. The critical assumption of the non-mover difference-in-difference strategy is that movers and non-movers experience the same unobservable shocks during SARS. An alternative to using movers as a control group is to control for unobservable shocks by conditioning on the current level of peer visits. The following specification regresses v_{ijkt} on $\Delta \bar{v}_{\neg ijkt}$ and conditions on $\bar{v}_{\neg ijkt}$.

$$v_{ijkt} = \gamma_1 \Delta \bar{v}_{\neg ijkt} + \gamma_2 S_t \Delta \bar{v}_{\neg ijkt} + \gamma_3 \bar{v}_{\neg ijkt} + \gamma_4 S_t \bar{v}_{\neg ijkt} + \gamma_5 v_{ijkt-26} + \alpha_{jk} + \delta_t + \epsilon_{ijkt} \quad (18)$$

This regression controls for peer group and time fixed effects but cannot utilize peer group $\times$ time fixed effects because it relies on variation across groups. The identifying assumption

of this approach is that no omitted variables cause a spurious correlation between v_{ijkt} and $\bar{v}_{\neg ijkt-26}$. Since $\bar{v}_{\neg ijkt-26}$ enters $\Delta\bar{v}_{\neg ijkt}$ negatively, only a *negative* correlation between v_{ijkt} and $\bar{v}_{\neg ijkt-26}$ may generate a spurious positive effect.[22]

Conceptually, this identification strategy fixes the current level of visits and compares the response in peer groups where visits were previously high to peer groups where visits were previously low. The risk signal is stronger in groups where visits were previously high because visits have implicitly declined more to reach the current level.[23] The level of peer visits controls for unobservable shocks because this variable is the first term in $\Delta\bar{v}_{\neg ijkt}$. Any contemporaneous shocks that influence $\Delta\bar{v}_{\neg ijkt}$ must also appear in $\bar{v}_{\neg ijkt}$. The distribution of peer visits is approximately binomial because 99.6 percent of people visit no more than once per period; therefore $\bar{v}_{\neg ijkt}$ is close to a sufficient statistic for the distribution of visits by peers. Conditioning on $\bar{v}^2_{\neg ijkt}$ can control for aspects of the visit distribution that the mean does not capture.

This identification strategy allows us to address directly the concern non-mover specific unobservable shocks. The identifying assumption of the non-mover difference-in-difference in the previous section is that non-movers do not experience targeted shocks during the SARS period. Because non-movers comprise 94 percent of the population, controlling for $\bar{v}_{\neg ijkt}$ primarily absorbs unobservable shocks to non-movers. We refine this approach by calculating the level of peer visits separately for movers and non-movers. Distinguishing between mover and non-mover peers slightly reduces the sample size because some peer groups contain only one mover or non-mover. To address non-mover specific shocks that differentially affect other non-movers (the particular concern in Sub-Section 4.2), we interact

[22] To observe movers' visits to their 2003 peer groups (a necessary aspect of the non-mover difference-in-difference), we must construct peer groups and measure behavior with the same raw data from 2001 to 2003. In regressions that control for $\bar{v}_{\neg ijkt}$, we can construct peer groups based on utilization prior to 2001. Defining peer groups based on 1999-2000 utilization yields similar results.

[23] One mechanism that may induce a negative correlation between v_{ijkt} and $\bar{v}_{\neg ijkt-26}$ is regression toward the mean. Stochastic shocks may elevate visits in period $t-26$ and suppress visits in period t. By conditioning on $v_{ijkt-26}$, Specification (18) controls for the effect of stochastic shocks on on the individual in period $t-26$. In addition, our regression focuses on the interaction between S_t and $\Delta\bar{v}_{\neg ijkt}$. Stochastic shocks would need to become stronger during the SARS period to cause a spurious correlation.

these variables with a non-mover indicator.

Regressions utilizing the complementary identification strategy appear in Table **??**. The coefficient on $S_t \Delta \bar{v}_{\neg ijkt}$ provides the social learning estimate in these regressions. In Column 1, the baseline estimate of 0.128 is nearly identical to the difference-in-difference estimate of 0.126 in Column 1 of Table **??**. Column 2 controls separately for the average visits of peers who are movers and non-movers. This distinction does not affect the social learning estimate, suggesting that shocks specific to non-movers are not an important source of bias in these regressions. Column 3 also controls for the interaction between a non-mover indicator and the visits of mover and non-mover peers. This specification permits non-mover specific shocks to differentially affect non-movers. Interactions with the non-mover dummy are small and insignificant, and the social learning estimate remains unchanged. Columns 4-6 incorporate the square of peer visits into these specifications, which only slightly reduces the social learning estimate.

4.4 Chinese New Year

A falsification test based on Chinese New Year further validates the social learning result. During Chinese New Year, both patients and physicians travel to reunite with family, causing a 20-30 percent decline in health care utilization that is plainly unrelated to social learning. Chinese New Year represents a combination of supply and demand shocks, both of which threaten the identification of the estimates above. This approach specifically addresses the objection that unobservable changes in health care supply or demand may drive the SARS results. Some offices close and others remain open during Chinese New Year, potentially generating a correlation between Δv_{ijkt} and $\Delta \bar{v}_{\neg ijkt}$. If unobservable shocks, either on the part of patients or doctors, spuriously drive our findings, then an interaction with Chinese New Year may generate the same pattern. We proceed by replacing S_t and all related interactions with an indicator for Chinese New Year in the primary specifications. Regressions exclude data from the SARS period (Quarters 2-4 of 2003).

Results for Chinese New Year based on both identification strategies appear in Table **??**. Column 1 replicates the baseline specification, replacing SARS interactions with interactions for Chinese New Year. The coefficient is 13 percent of the size of the SARS estimate in Column 1 of Table **??**. The regression that controls for $\bar{v}_{\neg ijkt}$ yields a coefficient that is 32 percent of the SARS estimate in Column 1 of Table **??**. These findings indirectly validate our methodology by failing to find social learning in this instance.

5 Dynamic Simulation

In this section, we simulate the dynamic response of visits to the SARS epidemic. The response to SARS may have a dynamic component because individuals update their beliefs about SARS risk using information from previous periods, including information from peers. To simulate the dynamic response, we first estimate regressions that allow us to predict visits in the current period based on information from the prior period. Then we simulate the behavior of a hypothetical population of individuals in each period, updating peer behavior in that period by aggregating individual responses before we simulate the next period.

This exercise also allows us to distinguish the relative influence of public information and social learning. Although regression analysis cannot identify the structural parameters associated with different sources of information, zero values of these parameters also imply zero values for certain regression coefficients. To simulate the path of visits without a given source of information, we simply zero out the appropriate regression coefficients when predicting individual visits. As an organizing principle, our simulation follows a thought experiment in which we sequentially remove social learning, peer group shocks, and learning from public information from the aggregate response to SARS.

5.1 Simulation Methodology

When conducting this exercise, we focus on simulating the behavior of movers rather than non-movers. For non-movers, the response to peers may be correlated with the responses to peer group shocks and public information. Therefore, one cannot zero out the influence of social learning by simply setting any one regression coefficient to zero and leaving other coefficients unaltered. Because movers arguably do not respond to social learning, the coefficients associated with their response to peer group shocks and public information are less likely to be contaminated by social learning. Therefore, it is credible to simulate social learning by movers by adding the regression coefficient that captures social learning by non-movers to regression coefficients that capture the influence of other sources of information on movers.

The simulation includes four counterfactuals, which we summarize in Table **??**.[24] In the first counterfactual, people respond to public SARS information, peer group shocks, and social learning. The regression model for the first counterfactual is a variant of equation (17) estimated on a combined sample of movers and non-movers.[25] We simulate the visits of movers by iteratively generating predictions using coefficient estimates that capture the behavior of movers plus coefficients on the regressors $N_i \Delta \bar{v}_{\neg ijkt}$ and $S_t N_i \Delta \bar{v}_{\neg ijkt}$, which capture social learning by non-movers.

The second counterfactual preserves the response to public information and peer group shocks but shuts down the social learning channel. This is accomplished by simulating the migrant behavior using the coefficient estimates employed in the first counterfactual but setting the coefficient on $S_t N_i \Delta \bar{v}_{\neg ijkt}$ to zero. The second counterfactual provides a conser-

[24] This exercise is based on the following algorithm. First, we create a simulation data set with 1000 hypothetical doctor's offices, each populated with 61 patients, the median size of peer groups in the regression sample. The simulation data set spans the period from 2002-2003. For each person, the number of visits during period t in 2002 equals the mean of this variable for movers in the regression sample. Beginning with the first period in 2003, we construct v_{ijt} using $\Delta \bar{v}_{\neg ijt}$ and v_{ijt-26} based on lagged data according to the requirements of each counterfactual.

[25] Our specification deviates from equation (17) in three important ways. First, we construct the regressors as sums over periods $t-2$ and $t-1$ (rather than $t-2$ to t), to avoid the need to determine v_{ijt} and $\Delta \bar{v}_{\neg ijkt}$ jointly in the subsequent simulation. Secondly, our regression eliminates the need to assign simulated people to actual townships by omitting local SARS incidence, for which the effect is small. Thirdly, we add an interaction between N_i and s_t^n to allow movers and non-movers to respond differently to public information.

vative measure of social learning because it does not zero out the coefficient on $N_i \Delta \bar{v}_{\neg ijkt}$, which may capture social learning that occurs independently of the SARS crisis.

For the third counterfactual, in which people only respond to public information, we estimate the regression for Counterfactual 1 on a subsample of movers. The sample restriction caused all regressors continaing N_i to drop out. The simulation uses the coefficient on public information from this regression but zeroes out the coefficients on $\Delta \bar{v}_{\neg ijkt}$ and all associated interactions. National SARS incidence is the only remaining variable that contains information about the epidemic. Because this variable is largely orthogonal to $\Delta \bar{v}_{\neg ijkt}$, neither peer group shocks nor social learning contaminates the effect of national SARS incidence in the modified regression. The fourth counterfactual also excludes the response to national SARS incidence by setting the coefficient on s_t^n to zero. This scenario provides a benchmark for comparison to the other counterfactuals.

5.2 Simulation Results

Figures **??** and **??** show the paths of aggregate visits and respiratory visits under the counterfactuals described above. The simulation isolates respiratory visits because Figure **??** and Table **??** indicate that respiratory visits contribute substantially to the overall decline in visits. In each figure, we calculate the ratio of aggregate visits by period under Counterfactuals 1-3 to aggregate visits under Counterfactual 4. The solid black line presents average visits by movers per period from our first counterfactual in which movers experience the social learning of non-movers. The dashed line shows the result for the second counterfactual, which excludes the response to social learning. The difference between this line and the solid line represents the contribution of social learning to the overall response. Finally the dotted line shows the response under the third counterfactual, which only includes the response to public information. The difference between the dotted line and the dashed line represents the response to unobservable peer group shocks.

Our simulation of visits for all diagnoses suggests that SARS incidence (public informa-

tion) was the sole driver of the initial, sharp decline in visits. Peer group shocks and social learning prolonged the decline beyond the peak in SARS incidence. By Period 13 (just after visits reach their nadir), unobservable shocks and social learning account for nearly half of the continued suppression in visits.[26] By the end of the epidemic in Period 16, visits remained almost 20 percent below normal[27] and social learning accounts for roughly one-third of all visit suppression. We find qualitatively similar results for respiratory visits in Figure **??**. Visits drop further and social learning plays a larger role, explaining no less than one half of the visit suppression that is not attributable to actual SARS incidence.

6 Conclusion

This paper analyzes the behavioral response to the SARS crisis. Our analysis broadens the existing approach to measuring the response to risk by comparing the response to public and private risk signals. Estimates indicate that the response to information from peers and the response to public information have similar elasticities. The social learning mechanism may partially explain why people react more strongly to risks that are novel rather than mundane. Our dynamic simulation indicates that social learning magnified the behavioral response to SARS risk. Future work will consider the collateral health impact of health care avoidance during this episode.

Crises like the SARS epidemic occur with regularity. Past examples include the 9/11 terrorist attacks, the outbreaks of H1N1 and H5N1 flu, and oil spill in the Gulf of Mexico. In 2011, the earthquake and tsunami in Japan in March forced many residents to assess the risk of radiation exposure. Despite reassuring test results, consumption of Japanese seafood fell dramatically because people worried about radiation (Fukue 2011, Kelland 2011). The outbreak of a novel strain of *E. coli* in Europe in June caused an international scare over

[26] Visits in our simulation closely track the actual decline in visits by movers, for whom all visits fell by around 25 percent and respiratory visits fell by around 60 percent.

[27] This result differs slightly from the finding in Table **??** that social learning had the largest impact in Quarters 2 and 4. This difference most likely arises because the simulation uses a single dummy for the SARS period (Quarters 2-4), while regressions in the table use separate dummies for each quarter.

Spanish produce before officials traced the outbreak to Germany (Patterson 2011).

The way people learn from their peers may strongly influence the duration and severity of an emergency. Social learning can cause the perception of risk to deviate from reality in either a positive or negative direction, leading to either an insufficient or excessive private response. By skewing individual risk perceptions, social learning may also influence the demand for public policies related to risk, such as counter-terrorism or nuclear energy initiatives. As a result, authorities may wish to control the extent of social learning about risk. Further research should examine how education campaigns or more precise public signals affect the reliance on information from peers.

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Table 1: Summary Statistics for Movers and Non-Movers during the Non-SARS Period

	Non-Movers		Movers		P Value
	Mean	S.D.	Mean	S.D.	
	(1)	(2)	(3)	(4)	(5)
Panel A: Individual Characteristics					
Male	0.50	0.50	0.53	0.50	0.00
Age	33.1	18.4	31.3	17.1	0.00
Income	828.7	579.6	798.3	565.3	0.00
Group membership	9.6	6.9	10.0	7.0	0.00
Visits					
--All	0.045	0.252	0.035	0.218	0.00
--Respiratory	0.018	0.162	0.014	0.137	0.00
--Critical	0.004	0.075	0.003	0.068	0.00
--Chronic	0.002	0.047	0.001	0.040	0.00
--Other	0.021	0.166	0.017	0.148	0.00
Change in visits					
--All	0.007	0.326	0.006	0.290	0.00
Panel B: Peer Group Characteristics					
Male	0.49	0.50	0.53	0.50	0.00
Age	32.6	18.7	31.1	17.2	0.00
Income	783.8	112.0	781.8	146.3	0.19
Non-mover	0.92	0.12	0.92	0.07	0.01
Group size	589	347	592	335	0.14
Physician male	0.91	0.18	0.93	0.14	0.00
Physician age	44.2	7.8	44.8	6.3	0.00
Visits					
--All	0.153	0.072	0.147	0.066	0.00
--Respiratory	0.073	0.059	0.067	0.052	0.00
--Critical	0.012	0.017	0.012	0.015	0.00
--Chronic	0.006	0.017	0.005	0.010	0.00
--Other	0.063	0.041	0.063	0.037	0.57
Change in visits					
--All	0.015	0.048	0.015	0.047	0.33
Number of patients	17,625	--	11,876	--	--

Note: visit counts are tallied by two-week interval during each period. Peer visits and the change in peer visits are tallied from periods t to t-2 for consistency with subsequent regressors. Income is the approximate monthly earnings in US Dollars.

Table 2: The Response to SARS Information by Information Source

Dependent variable:	Individual visits				
SARS case definition	Reported		Probable		N/A
	(1)	(2)	(3)	(4)	(5)
Local SARS incidence	-0.148	-0.017	-0.351	-0.108	--
	(0.068)	(0.064)	(0.117)	(0.111)	
National SARS incidence	-1.560	-0.962	-5.048	-3.487	--
	(0.337)	(0.339)	(1.344)	(1.330)	
SARS × N × change in peer visits	--	0.090	--	0.090	0.101
		(0.027)		(0.027)	(0.027)
Lagged individual visits	0.075	0.076	0.075	0.076	0.076
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Pairwise interactions	No	Yes	No	Yes	Yes
Fixed effects:					
Peer group	Yes	Yes	Yes	Yes	Yes
Year and period	Yes	Yes	Yes	Yes	No
Year × period	No	No	No	No	Yes
Sample size	17,299,100	17,299,100	17,299,100	17,299,100	17,299,100
R-squared	0.119	0.121	0.119	0.121	0.121

Standard errors appear in parentheses. Standard errors are clustered by the patient's modal township. Individual Visits is observed at time t, Lagged Individual Visits is observed at time t-26, and all other regressors are observed from time t to t-2. SARS is an indicator for quarters 2-4 of 2003. N is an indicator that the person is a non-mover.

Table 3: Alternative Peer Group Definitions and Fixed Effects Specifications

Dependent variable:	Individual visits							
Peer group definition:	Physician × facility		Facility		Township		County	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
SARS × N × change in peer visits	0.076	0.051	0.129	0.119	0.075	0.076	0.043	0.040
	(0.023)	(0.031)	(0.019)	(0.021)	(0.010)	(0.008)	(0.006)	(0.005)
Fixed effects:								
Physician × facility × SARS	Yes	No	Yes	No	Yes	No	Yes	No
Physician × facility × Year × Period	No	Yes	No	Yes	No	Yes	No	Yes
Year × period	Yes	No	Yes	No	Yes	No	Yes	No
Sample size	17,299,100	17,299,100	17,299,100	17,299,100	17,299,100	17,299,100	17,299,100	17,299,100
R-squared	0.144	0.600	0.144	0.591	0.144	0.550	0.144	0.549

Note: standard error appear in parentheses and are clustered by the patient's modal township. The dependent variable is measured at time t, while all regressors are calculated from time t to t-2. SARS is an indicator for the SARS period. N is an indicator that the person is a non-mover. Regressions include all pairwise interactions between SARS, N, and the change in peer visits.

Table 4: Learning from Multiple Peer Groups

Dependent variable:	Individual visits (group j)	
	(1)	(2)
SARS × N × change in peer visits (group j)	0.082	0.055
	(0.027)	(0.027)
SARS × N × change in peer visits (other groups)	0.203	0.162
	(0.041)	(0.046)
SARS × N × change in peer visits (group j × other groups)		-1.177
		(0.555)
Fixed effects:		
Peer group	Yes	Yes
Year × period	Yes	Yes
Sample size	17,239,224	17,239,224
R-squared	0.120	0.120

Note: standard errors appear in parentheses and are clustered by the patient's modal township. The dependent variable is measured at time t while all of the regressors are measured at time t to t-2.

Table 5: Social Learning by Diagnosis and Quarter of 2003

Dependent variable:	Individual visits				
Type of visit:	All	Respiratory	Critical	Chronic	Other
	(1)	(2)	(3)	(4)	(5)
N × change in peer visits:					
× 2003 quarter 1	0.038	0.055	-0.008	-0.008	-0.002
	(0.032)	(0.021)	(0.009)	(0.005)	(0.022)
× 2003 quarter 2	0.117	0.071	0.004	-0.002	0.046
	(0.039)	(0.020)	(0.016)	(0.006)	(0.025)
× 2003 quarter 3	0.064	0.025	0.002	0.007	0.033
	(0.037)	(0.019)	(0.012)	(0.009)	(0.024)
× 2003 quarter 4	0.082	0.037	0.003	0.010	0.037
	(0.036)	(0.020)	(0.009)	(0.008)	(0.028)
Peer group fixed effects	Yes	Yes	Yes	Yes	Yes
Year × period fixed effects	Yes	Yes	Yes	Yes	Yes
Sample size	17,239,224	17,239,224	17,239,224	17,239,224	17,239,224
R-squared	0.121	0.111	0.129	0.214	0.113

Note: standard error appear in parentheses and are clustered by the patient's modal township. The dependent variable is measured at time t, while all regressors are calculated from time t to t-2. Critical visits include visits related to pregnancy, abortion, injury, appendicitis, stroke, heart attack, and internal bleeding. Chronic visits include visits related to dialysis, chemotherapy, diabetes, and liver or kidney failure.

Table 6: Regressions that Utilize the Level of Visits as a Control

Dependent variable:	Individual visits		
Specification:	Group j	Group j and other groups	Interaction of group j and other groups
	(1)	(2)	(3)
SARS × N × change in peer visits	0.076	0.069	0.059
	(0.026)	(0.026)	(0.025)
SARS × N × change in peer visits (other groups)	--	0.096	0.077
		(0.038)	(0.041)
SARS × N × change in peer visits (group j × other groups)	--	--	-0.666
			(0.402)
SARS × N × peer visits	-0.077	-0.081	-0.159
	(0.023)	(0.024)	(0.049)
SARS × N × peer visits (other groups)	--	0.010	-0.104
		(0.035)	(0.048)
SARS × N × peer visits (group j × other groups)	--	--	0.590
			(0.315)
Fixed effects:			
Peer group	Yes	Yes	Yes
Peer group × SARS	No	No	No
Year × period	Yes	Yes	Yes
Sample size	17,239,224	17,239,224	17,239,224
R-squared	0.123	0.121	0.121

Note: standard error appear in parentheses and are clustered by the patient's modal township. The dependent variable is measured at time t, while all regressors are calculated from time t to t-2.

Table 7: A Falsification Test Using Chinese New Year

Dependent variable:	Individual visits					
Identification strategy	Difference in difference			Control for peer visits		
	(1)	(2)	(3)	(4)	(5)	(6)
Social learning estimate	-0.047	-0.062	-0.072	-0.037	-0.042	-0.043
	(0.035)	(0.035)	(0.035)	(0.025)	(0.026)	(0.026)
Social learning estimate (other groups)	--	0.119	0.115	--	0.041	0.039
		(0.071)	(0.075)		(0.057)	(0.059)
Social learning estimate (group j × other groups)	--	--	0.446	--	--	0.013
			(0.639)			(0.430)
Fixed effects:						
Peer group	Yes	Yes	Yes	Yes	Yes	Yes
Year × period	Yes	Yes	Yes	Yes	Yes	Yes
Sample size	12,821,420	12,821,420	12,821,420	12,821,420	12,821,420	12,821,420
R-squared	0.130	0.128	0.128	0.130	0.129	0.129

Note: standard errors appear in parentheses and are clustered by the patient's modal township. The social learning estimate refers to SARS × change in peer visits in Column 4 and to SARS × N × change in peer visits in all other columns.

Table 8: Description of Simulation Counterfactuals

Counterfactual	Description	Regression model and sample	Regression sample	Simulation model
1	Public information + peer group shocks + social learning	$v_{ijkt} = \beta_2 s_{t-1}^n + \beta_3 S_t N_i \Delta\bar{v}_{-ijkt} + \beta_4 v_{ijkt-26} +$ [levels and pairwise interactions of S_t, N_i and $\Delta\bar{v}_{-ijkt}$] $+\alpha_{jk} + \delta_t + \epsilon_{ijkt}$	Movers and non-movers	$\hat{v}_{ijkt} = \hat{\beta}_2 s_{t-1}^n + \hat{\beta}_3 S_t \Delta\bar{v}_{-ijkt-1} + \hat{\beta}_4 v_{ijkt-26} +$ [levels and pairwise interactions of S_t and $\Delta\bar{v}_{-ijkt}$] $+ \hat{\delta}_t + u_{ijkt}$
2 (Version 1)	Public information + peer group shocks	Same as above	Movers and non-movers	$\hat{v}_{ijkt} = \hat{\beta}_2 s_{t-1}^n + \hat{\beta}_4 v_{ijkt-26} +$ [levels and pairwise interactions of S_t and $\Delta\bar{v}_{-ijkt}$] $+ \hat{\delta}_t + u_{ijkt}$
2 (Version 2)	Public information + peer group shocks	$v_{ijkt} = \beta_2 s_{t-1}^n + \beta_4 v_{ijkt-26} +$ [levels and pairwise interactions of S_t and $\Delta\bar{v}_{-ijkt}$] $+\alpha_{jk} + \delta_t + \epsilon_{ijkt}$	Movers	Same as above
3	Public information	$v_{ijkt} = \beta_2 s_{t-1}^n + \beta_4 v_{ijkt-26} + \alpha_{jk} + \delta_t + \epsilon_{ijkt}$	Movers	$\hat{v}_{ijkt} = \hat{\beta}_2 s_{t-1}^n + \hat{\beta}_4 v_{ijkt-26} + \hat{\delta}_t + u_{ijkt}$
4	No information	Same as above	Movers	$\hat{v}_{ijkt} = \hat{\beta}_4 v_{ijkt-26} + \hat{\delta}_t + u_{ijkt}$

Note: u_{ijkt} is an independent draw from a $N(0, \hat{\sigma}_\varepsilon^2)$ distribution, where $\hat{\sigma}_\varepsilon^2$ is the variance of the residual from the regression model in Counterfactual 1. $\hat{v}_{ijkt}$ is the prediction of visits.

Figure 1: SARS Cases by Two-Week Period during 2003

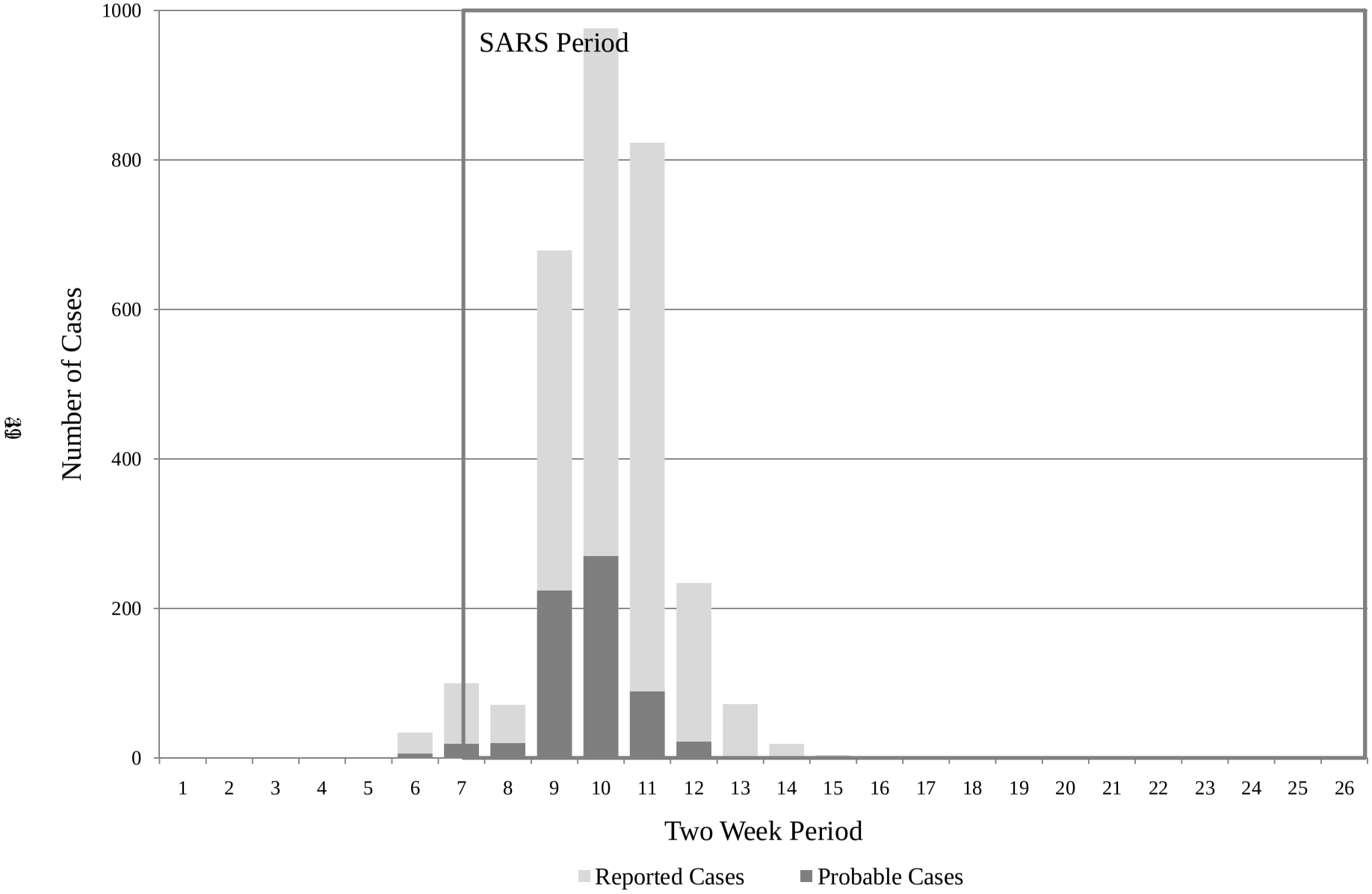

Figure 2: Aggregate Outpatient Visits by Two-Week Period: 2001-2003

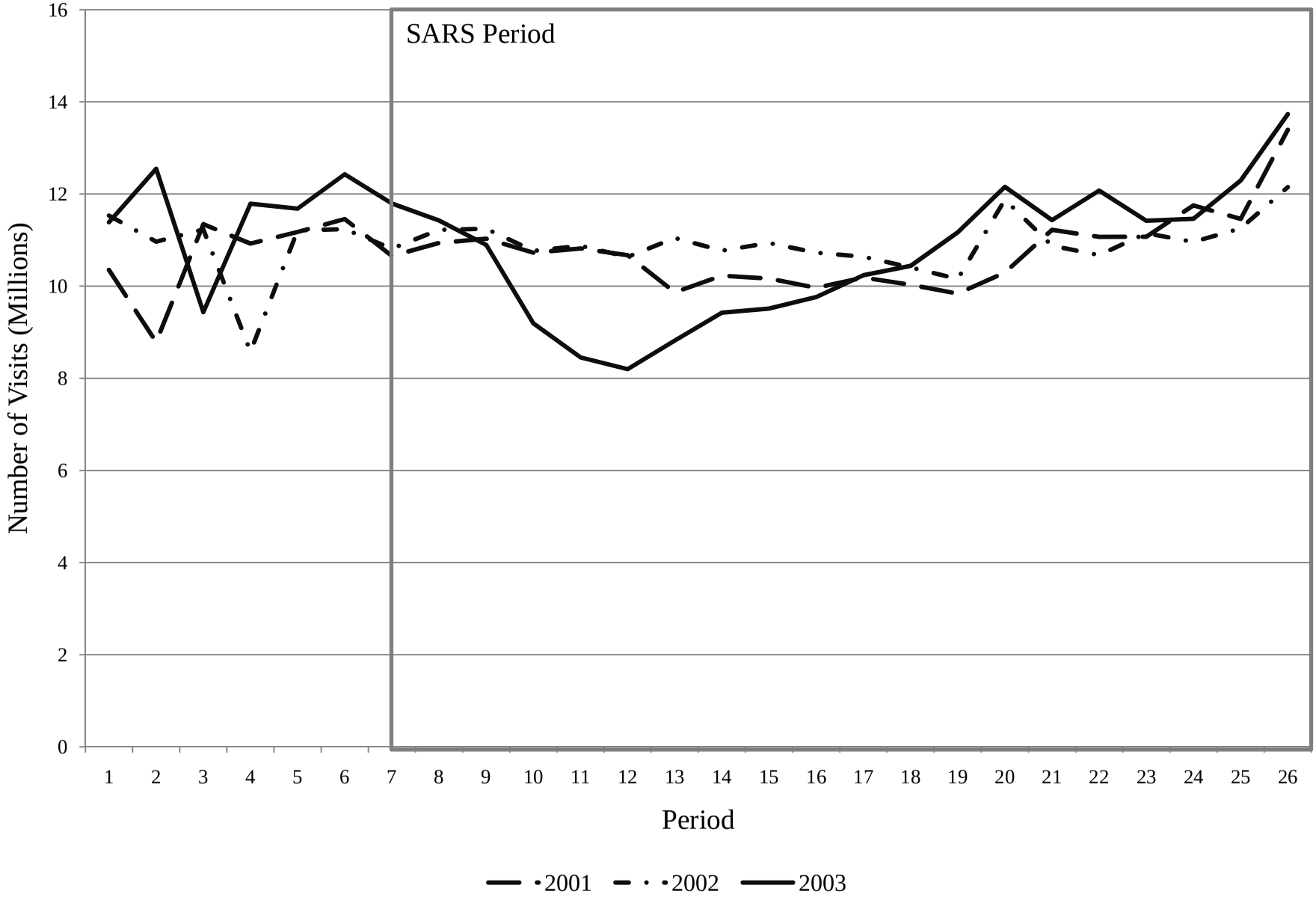

Figure 3: Visits for SARS-Affected and Unaffected Townships During 2003

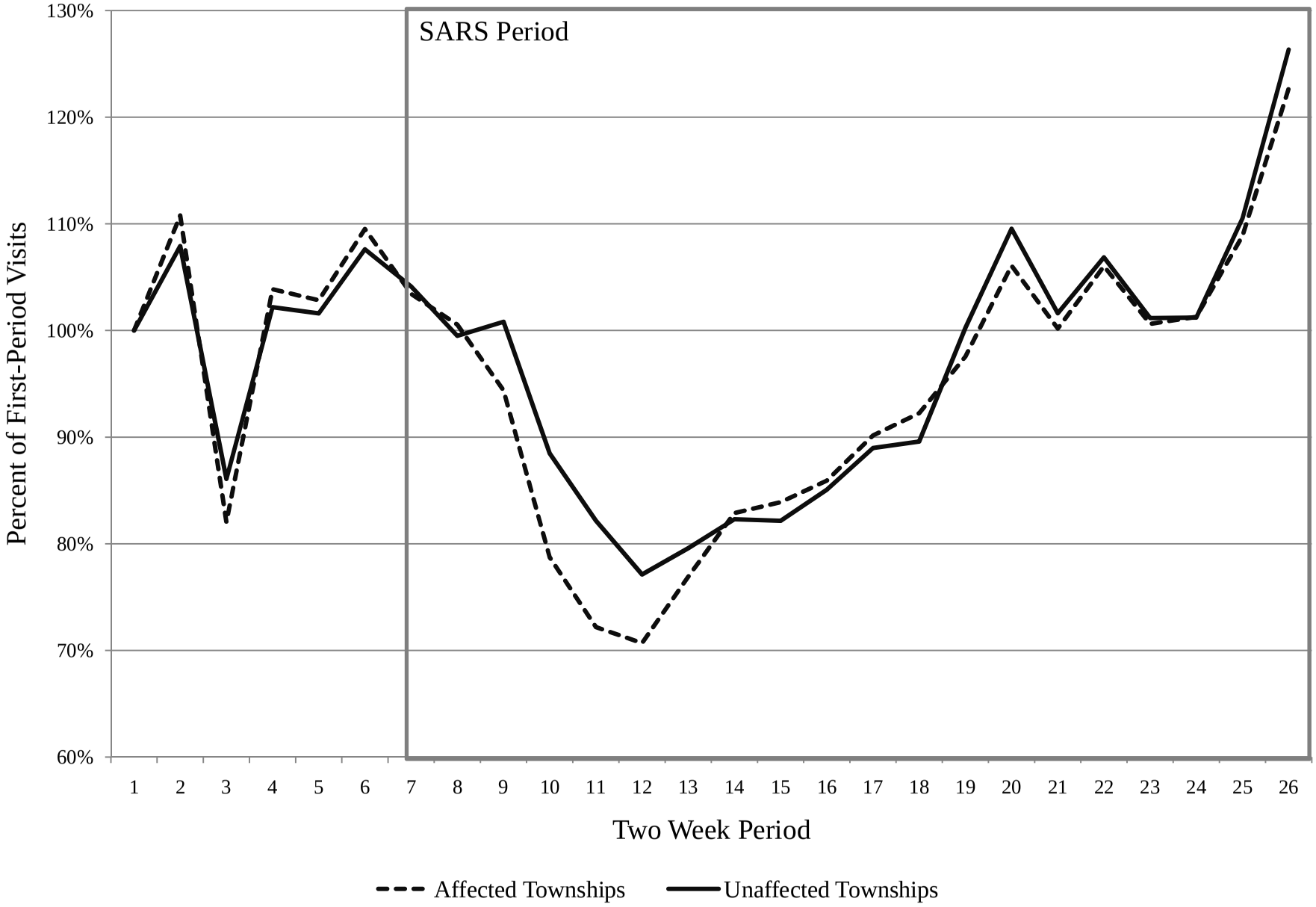

Figure 4: Visits by Diagnosis During 2003

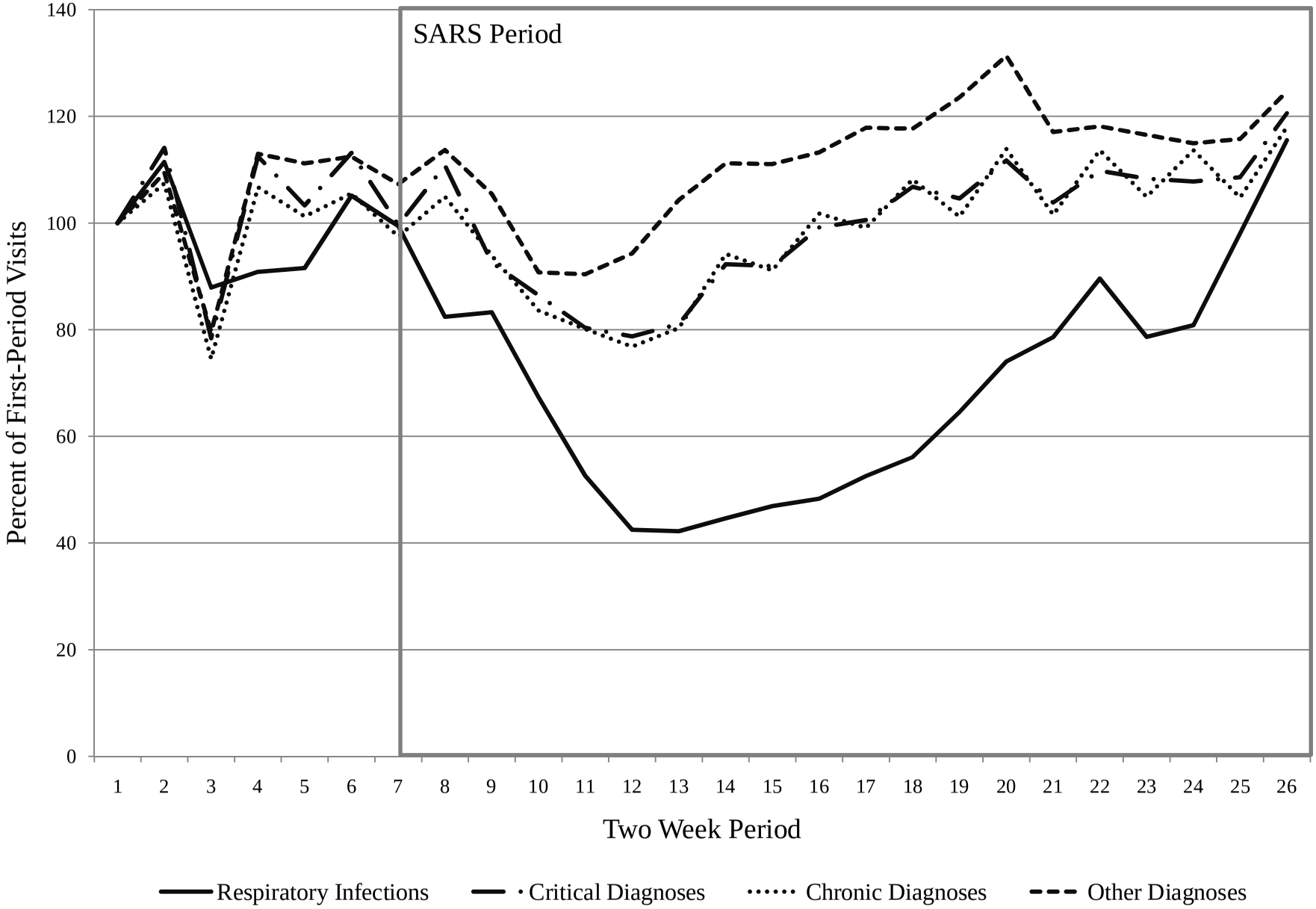

Figure 5: News Coverage of the SARS Epidemic

Figure 5: News Coverage of the SARS Epidemic

Figure 6: The Coefficient of Variation Within and Across Groups by Two-Week Period

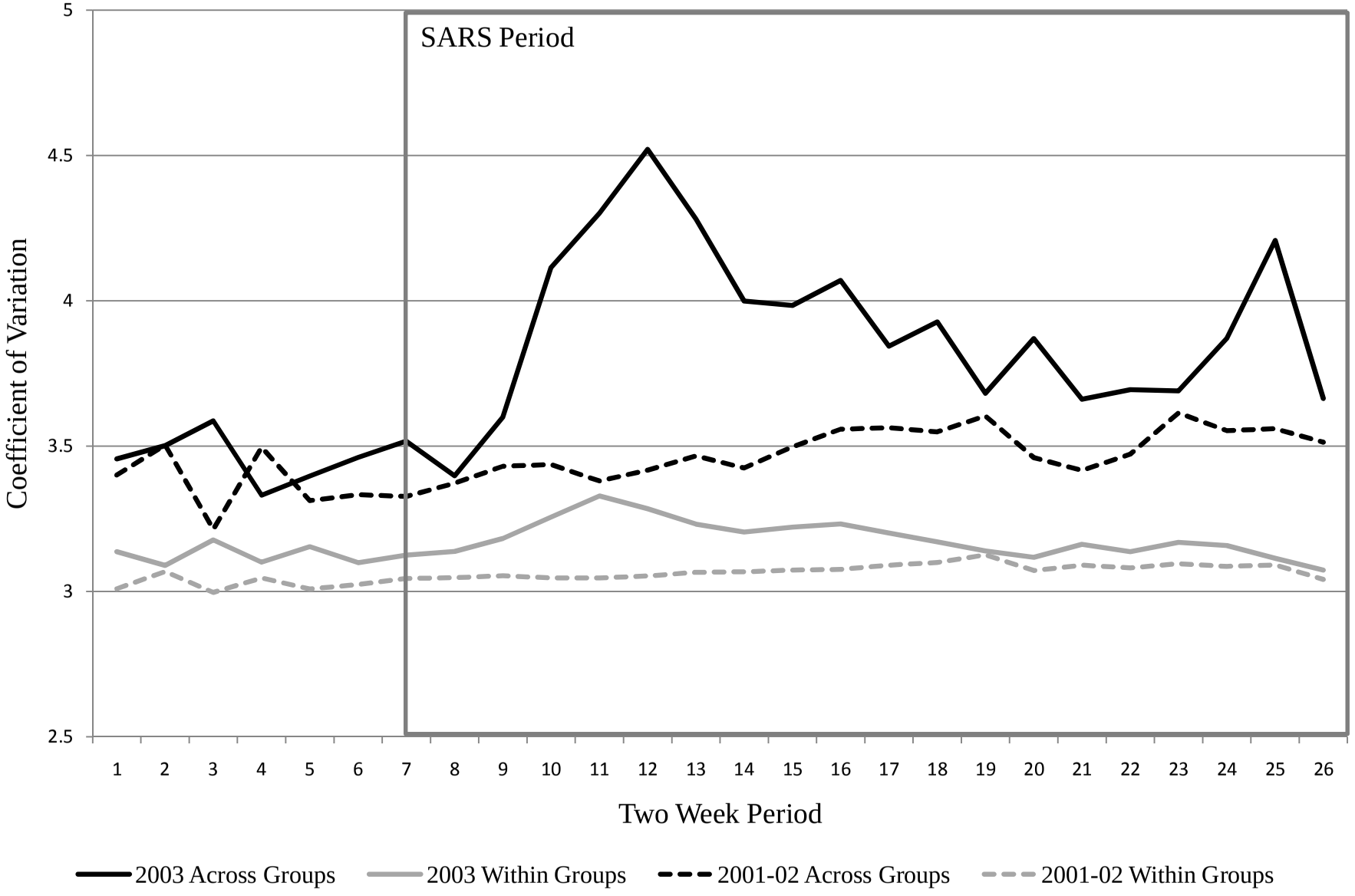

Figure 7: the Prevalence Response Elasticity by Information Source

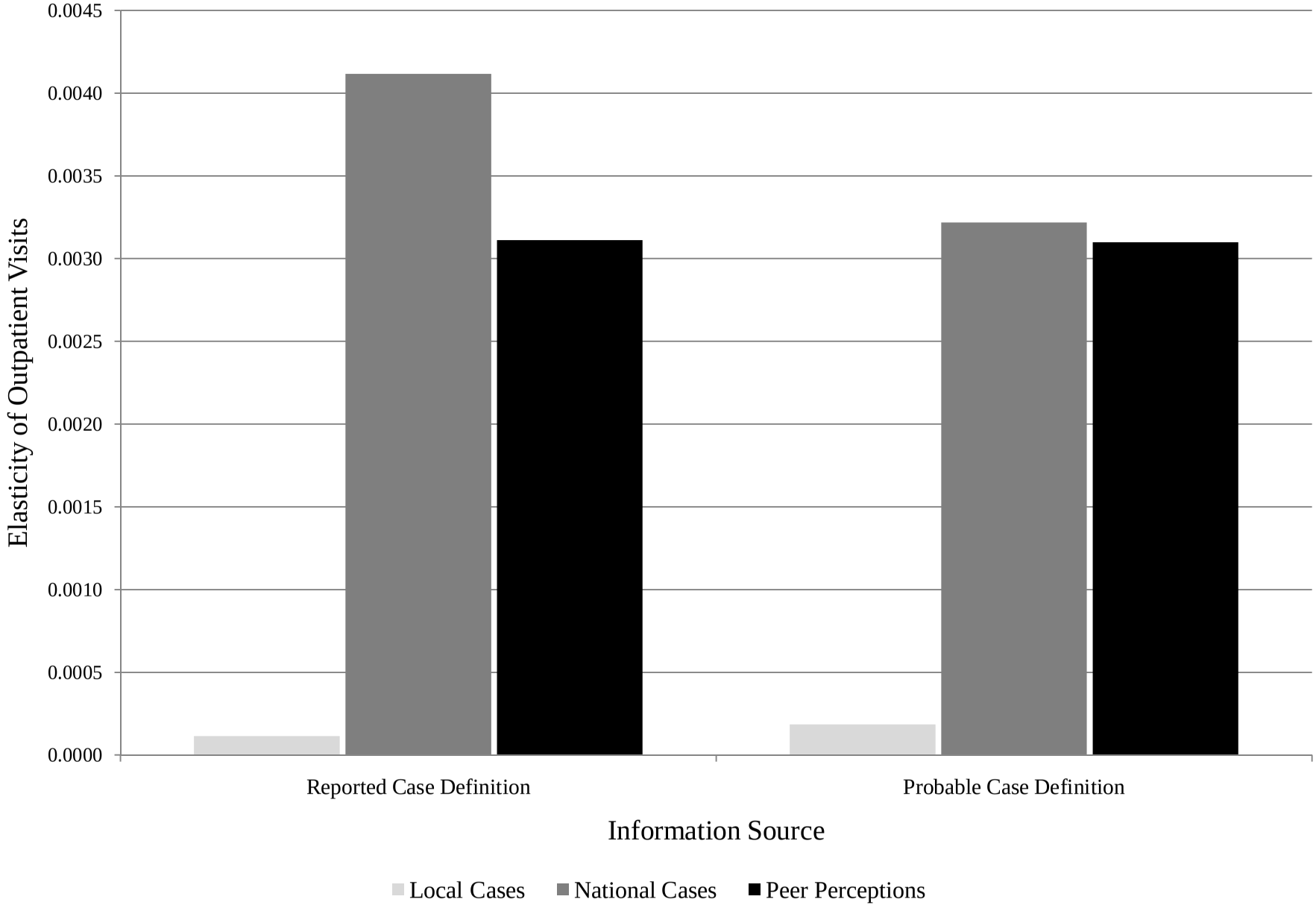

Figure 8: Social Learning by the Patient's Arrival Status

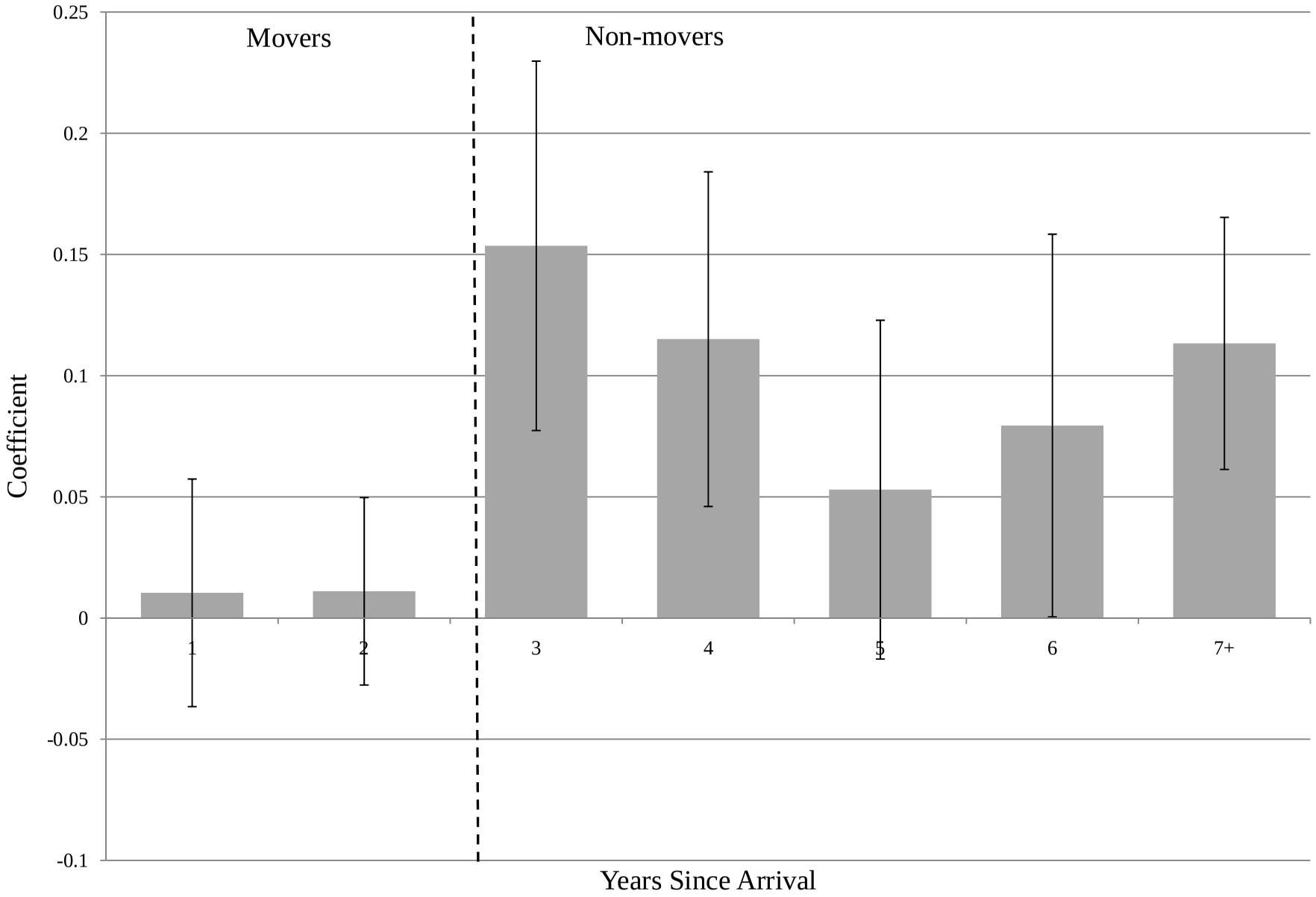

Figure 9: The Simulated Path of Aggregate Visits Under Alternative Scenarios

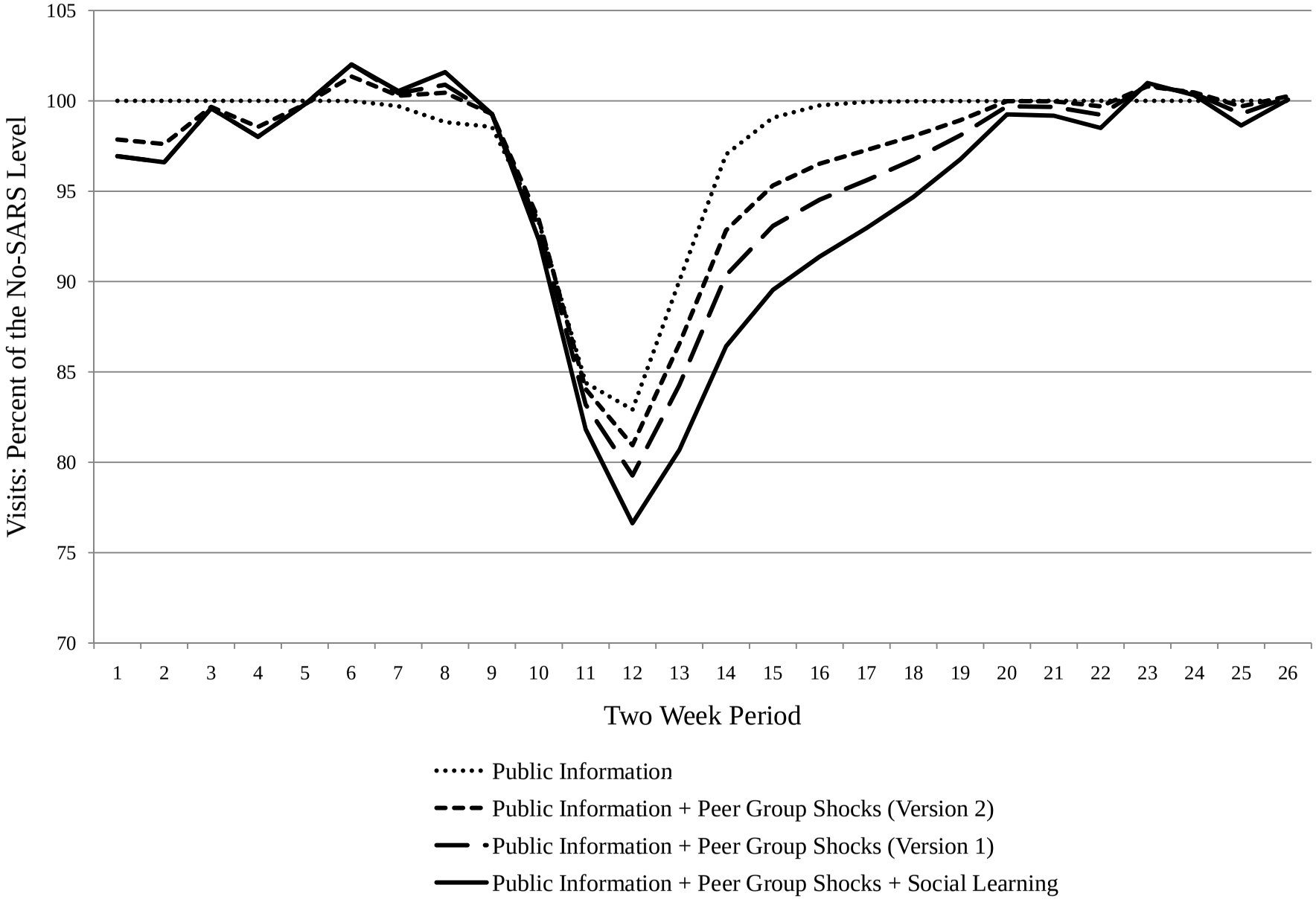

Figure 10: Simulated Path of Respiratory Visits Under Alternative Scenarios