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ABSTRACT

When SARS struck Taiwan in the spring of 2003, many people feared that the disease would spread through the health care 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 longtime community residents to recent arrivals, who are less socially connected. We find that people learned from both public and private sources during SARS. A dynamic simulation based on the regressions shows that social learning magnified and lengthened 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, culling 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 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 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 an outbreak. In even the most saturated media markets, public announcements only weakly signal a person's idiosyncratic infection risk. The lack of a precise public signal may cause people to rely on private signals such as the opinions or actions of their peers. This mechanism has the potential to cause an "information cascade" that magnifies the response to an unfamiliar risk (Bikchandani et

al. 1992, Banerjee 1992, Welch 1992). Although this phenomenon has ambiguous welfare implications, social learning may mediate the effectiveness of public announcements or other policies during an emergency.

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 show theoretically that under some conditions, the change in health care utilization among peers (from a pre-SARS baseline) provides a proxy for the risk perception of peers. 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 29,501 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. A difference-in-difference design addresses this concern by comparing the response of longtime community residents to the response of recent arrivals who are less socially connected. We find that social learning has a larger effect than local incidence reports, but a smaller effect than national incidence reports.

Several supplemental results reinforce this finding. Under our definition of a peer group, patients may belong to multiple groups, each of which offers a signal of risk. We regress the patient's visits to the doctor that defines group j on the signal from group j as well as from other groups. This regression shows evidence of social learning from both group j

and other groups. A regression featuring the interaction between the signals from group j and other groups indicates that these signals are substitutes. Both results are unlikely to arise spuriously. A complementary identification strategy controls for common unobservables using the current *level* of peer visits. Estimates from this approach resemble the difference-in-difference results. 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 two literatures. In economic epidemiology, this paper is the first examination of the individual behavioral response to a new outbreak. We demonstrate that under incomplete information, the response elasticity varies by information source. This paper also contributes to the literature on social learning. Several studies have examined how social learning affects technology adoption (Foster and Rosenzweig 1995, Munshi 2004, Conley and Udry 2010) or consumption (Moretti 2010). The few studies that examine social learning in medical utilization (Aizer and Curry 2004, Rao et al. 2007, Deri 2005) do not consider the context of an emergency.

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. First, we present a simple model of individual belief formation about SARS risk. Rather than determine exactly how people form beliefs, our

goal is to offer a plausible structural interpretation for our subsequent empirical estimates. We incorporate the decision to seek health care and illustrate the conditions under which a chance in medical visits (from a risk-free period) proxies for the 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. People may learn about s_{ijt} from a common signal, $s^c_{jt} \in [0,1]$, an individual private signal, $s^p_{ijt} \in [0,1]$, and the private signals of their peers, $\{s^p_{\neg ijt}\}$. We define each signal to be orthogonal to the others. In our application, the common signal represents media coverage of SARS while the private signal represents an individual's idiosyncratic knowledge or assessment of SARS risk. People also learn from other unobservable factors, ε_{ijt}, which may include their Bayesian prior beliefs.

Our empirical objective is to test whether individual risk perceptions depend upon the common signal or peers' private signals. To proceed, we assume that each person's risk perception is a linear weighted average of the common signal and private signals. For tractability, we also assume that these weights are homogeneous across people and that people learn from the average private signal of their peers. The following orthogonal decomposition represents person i's perception of SARS risk.[1]

$$s_{ijt} = \phi_1 s^c_{jt} + \phi_2 s^p_{ijt} + \phi_3 \bar{s}^p_{\neg ijt} + \varepsilon_{ijt} \tag{1}$$

In this expression, $\bar{s}^p_{\neg ijt} = \sum_{l \in I, l \neq i} s^p_{ljt}/(N_j - 1)$ is the average private signal of peers and $\phi_m \geq 0$ for $m \in \{1,2,3\}$ are the weights on the signals. People learn from the common signal if $\phi_1 > 0$, from their own private signal if $\phi_2 > 0$, and from the private signals of peers if

[1] A more general model might include interactions between the common and private signals or allow the weight associated with each signal to depend upon the signal's precision. These embellishments make it more difficult to map from the theory to the empirical estimates.

$\phi_3 > 0$. If each of these elements 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 average private signal of peers, $\bar{s}^p_{\neg ijt}$, by replacing it with the average *overall* risk perception of peers, $\bar{s}_{\neg ijt} = \Sigma_{l \in I, l \neq i} s_{ljt}/(N_j - 1)$, in the hypothetical regression equation. We justify this step by noting that under the assumption that people place common weights on the signals, the average risk perception of peers simplifies to the following expression:

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

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).

Both expressions (1) and (2) become more straightforward if N_j is large.[2] As the group size increases, the influence of person i's private information on the average private information of the group declines: $\lim_{N_j \to \infty} \bar{s}^p_{\neg ijt} = \bar{s}^p_{jt}$. This fact simplifies the expression for individual learning in equation (3) below, and allows us to ignore the informational feedback between person i and the group in equation (4).

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

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

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). After this substitution, coefficients from a regression based on this expression no longer map directly into the structural

[2] A large N_j assumption is reasonable because the median peer group size is 60 in our data.

parameters.

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

Moreover, even this equation cannot be estimated because we do not observe person i's private information.

Instead we propose a hypothetical regression of the individual's risk perception on the common signal and the group risk perception.

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

Person i's private signal, s^p_{ijt}, appears in equation (5) but is an omitted variable in equation (6). Fortunately, it is safe to overlook this variable because the information sources in equation (1) are orthogonal. The orthogonality of the information sources directly ensures that the omitting this variable does not bias α_1. This assumption also ensures that s^p_{ijt} is uncorrelated with $\bar{s}^p_{\neg ijt}$. 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. Thus, omitting the individual private signal does not bias α_2 if groups are large.

The regression in equation (6) yields a system of three equations relating the regression coefficients to the structural parameters of the learning model.

$$E[\hat{\alpha}_0] = \phi_2 \bar{s}^p \tag{7}$$

$$E[\hat{\alpha}_1] = \frac{\phi_1\phi_2}{\phi_2+\phi_3} \tag{8}$$

$$E[\hat{\alpha}_2] = \frac{\phi_3}{\phi_2+\phi_3} \tag{9}$$

In this expression, $\bar{s}^p$ denotes the sample mean of s^p_{ijt}, which is unknown. Because the system includes three equations and four unknowns, we cannot identify the structural parameters directly. However equation (9) shows that a non-zero coefficient on the risk perception of peers is evidence of social learning. This coefficient represents the impact of social learning relative to the combined impact of social learning and the individual private signal. Likewise,

a non-zero coefficient on the common signal is evidence of learning from public information.

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 how 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. Holding illness and other costs constant, a change in visits indicates an increase 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 forces him to pay 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:[3]

$$EU[v_{ijt} = 1] = (1 - s_{ijt})u(h_{ijt}, m_{ijt} - c_t) \tag{10}$$

$$EU[v_{ijt} = 0] = u(h_{ijt} - d_{ijt}, m_{ijt}) \tag{11}$$

A person seeks care if the value of alleviating his illness exceeds the cost of treatment:

[3]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.

$EU[v_{ijt} = 1] > EU[v_{ijt} = 0]$. Taking logs leads 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 incorporate this requirement, we assume that the net utility from a visit is a trend-stationary function of the person's age: $u(h_{ijt} - d_{ijt}, m_{ijt}) - u(h_{ijt}, m_{ijt} - c_t) = \mu_{ij} + g a_{ijt} + e_{ijt}$, where a_{ijt} is the person's age.[4] 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 that 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 that $\ln(1 - s_{ijt}) \approx -s_{ijt}$ for small values of s_{ijt}.

$$pr[v_{ijt} = 1] = F[\ln(1 - s_{ijt}) - \mu_{ij} - g a_{ijt}] \tag{12}$$

$$\approx F(\bar{e}) - f(\bar{e})[s_{ijt} + \mu_{ij} + g a_{ijt} - \bar{e}] \tag{13}$$

The probability of a visit is 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

[4] Under the assumption of a quadratic trend, equations (14) and (15) become non-linear functions of age.

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.

$$E[\Delta v_{ijt}] \approx f(\bar{e})gk - f(\bar{e})s_{ijt} \tag{14}$$

$$E[\Delta\bar{v}_{\neg ijt}] \approx f(\bar{e})gk - f(\bar{e})\bar{s}_{\neg ijt} \tag{15}$$

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 following regression.

$$\Delta v_{ijt} = \beta_0 + \beta_1 s^c_{jt} + \beta_2 \Delta\bar{v}_{\neg ijt} + \eta_{ijt} \tag{16}$$

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.

$$E[\hat{\beta}_1] = -f(\bar{e})\left[\frac{\phi_1\phi_2}{\phi_2+\phi_3}\right] \tag{17}$$

$$E[\hat{\beta}_2] = \frac{\phi_3}{\phi_2+\phi_3} \tag{18}$$

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. As above, $\hat{\beta}_2$ 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 (17) 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.

$$\Delta v_{ijt} = \beta_3 + \beta_4 s^c_{jt} + \omega_{ijt} \tag{19}$$

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 $\hat{\beta}_1$ to measure this response leads to a larger and more accurate estimate of learning from public information. A regression based on equation (19) proves useful for the dynamic simulation in Section 5, where it serves as a predictive model that fully 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 long 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 make 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 health care 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 find it advantageous to visit the doctor (and obtain medicine) for minor illnesses such as sore throats and colds. These conditions, classified broadly as "upper respiratory infections," comprise 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. SARS 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 1 shows the trajectory of the epidemic by plotting the number of reported and probable SARS cases (explained below) by two-week period. 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), "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 and international air travel fell by 30 percent and 58 percent, respectively, 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 2 plots the nationwide volume of outpatient visits by two-week period in 2001, 2002, 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. To quantify the severity of this reaction, we compare the SARS response to the decline in visits that occurred after a 2002 increase in the copayment. Among medical centers (the most advanced facilities), the copayment rose by US$3 and visits fell by 3 percent. Using $2 million as a benchmark for the value of statistical life, SARS mortality risk increased the copayment by US$0.75 in expectation. However, scaling by the 2002 price response, the public reacted as though SARS had increased the copayment by $34.

The self-protective response did not differentiate between townships that did and did not experience actual SARS incidence. Figure 3 distinguishes between townships with and without any SARS cases during the epidemic. The response to SARS is only slightly larger in townships that actually experienced the outbreak. The large response in places where there was zero ex-post SARS risk casts doubt on the premise that people had complete information about SARS risk.

The timing and magnitude of the SARS response also depended on the nature of the visit. Figure 4 categorizes visits as respiratory, critical, chronic, or other.[5] 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 several mechanisms may be at play, the low marginal benefit of a respiratory visit is the likely explanation for this pattern.[6]

[5]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.

[6]Alternatively, patients with minor respiratory ailments may have feared that doctors would place them

3.2 Signals of Risk

Decision makers who lack full information about disease risk may seek out new sources of information and tailor their self-protective response to the credibility and precision of the information source. Common risk signals, such as announcements about disease prevalence or incidence, can accurately convey the average risk in a population. However, these announcements are not necessarily informative about a person's idiosyncratic disease risk, which depends upon personal behavior and social interactions. Objective data are particularly noisy during the outbreak of a new disease, when even experts do not fully understand the disease's severity or mode of transmission. Without sufficient objective information, people may learn from subjective signals such as the actions or opinions of their peers.

SARS incidence reports from the Taiwan Centers for Disease Control (TCDC) provide an objective signal of SARS risk. The agency released these reports daily and to widespread media coverage throughout the epidemic. The newspaper clipping from the Apple Daily News on May 22, 2003 in Figure 5 exemplifies the print coverage of SARS. The lead article describes a SARS-related restriction on travel out of Taiwan. The map on the left, which shows the cumulative number of SARS cases by county, sits above a summary of the number cases and deaths nationwide. In principle, either local or national SARS incidence may provide the most salient objective signal. Taiwan is a small island where an outbreak could easily spread across jurisdictions. The country shares one media market with common television and print outlets. These features suggest that national SARS reports may affect behavior more than local reports.

Without a precise common signal of idiosyncratic risk, people may rely on private signals such as the opinions or behavior of their peers. As the model describes, the change in medical utilization from a previous (risk-free) period signals a person's perception of SARS risk. This signal is noisy as an individual metric because health varies idiosyncratically.

in quarantine (Hsieh et al. 2005). As a respiratory condition, SARS could also have increased respiratory visits among people concerned about possible exposure.

Any individual's decline in visits may merely reflect the absence of an illness that existed previously. Within-group aggregation abates the idiosyncratic noise in this variable and improves its power as a signal of perceived risk.

During SARS, the rate of visitation became more disperse across groups. Figure 6 plots the coefficient of variation (CV) in visits by two-week period and distinguishes between variation within and across peer groups.[7] In the figure, intergroup variation drives the large increase in this statistic during SARS. This finding of "excess variation" motivates our examination of social learning in this setting (Glaeser et al. 1996, Graham 2008). The reader should interpret this increase cautiously since a decline in the mean may raise the coefficient of variation mechanically. 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, which administers Taiwan's health care system. This data set contains all claims since 1997 for a representative sample of one million people (4.3 percent of Taiwan's population). This sample is unmanageably large for our purposes, and we utilize a randomly-selected three-percent subsample of 29,501 people. The outcome variable in our analysis is the number of outpatient visits by patient, provider, and two-week period from 2001 to 2003.

A patient's actual peer group consists of his family, friends, and neighbors. We proxy for peer groups, which we do not observe directly, by constructing cohorts of patients who visit the same doctor and health care facility. The baseline definition of a peer group is the set of people who see a common physician $\times$ facility from 2001 to 2003.[8] The outpatient health care market is highly localized, so that many neighbors frequent the same physicians.

[7]The decline in visits, which are bounded at zero, mechanically reduces the standard deviation in visits. The CV partially overcomes this issue by dividing by the mean.

[8]Under this approach, we construct peer groups and measure behavior using the same raw data, which allows us to observe the visits of movers to their current physicians. The regressions in Table 6, which do not rely on a non-mover difference, are robust to using 1999-2000 activity to define cohorts.

The referral process also connects our proxy to actual peer groups. Many patients select a physician through a friend's referral, so that some people who visit a common physician are directly acquainted (Hoerger and Howard 1995, Tu and Lauer 2008). The empirical definition of a peer group as a patient cohort permits patients to belong to multiple peer groups. Patients in our sample belong to a median of 7 peer groups, which contain a median of 61 people. Regressions below show that results are robust under alternative definitions of the peer group.

Measurement error in the definition of the network is a common issue that does not ordinarily interfere the identification of social interactions. Blume et al. (2011) show that identification is still possible in this setting as long as the true social network overlaps with the proxy. Misidentification of the peer group most likely (though not necessarily) causes attenuation bias through the same mechanism as classical measurement error. We verify that members of a common group are similar by computing the intra-group correlation in income, age, gender, and modal township (a proxy for residential location). Within a common physician $\times$ facility, the correlation coefficients are 0.30, 0.65, 0.42, and 0.34 for income, age, gender, and modal township respectively. These correlations decline as expected if the facility, township or county is used as the peer group proxy instead.

As our model shows, the one-year change in average visits within the peer group provides a proxy for the group's SARS risk perception. $\bar{v}_{\neg ijt}$ denotes the number of visits per person in group j and period t, excluding the index person. The change in peer visits, $\Delta\bar{v}_{\neg ijt}$, is the difference in $\bar{v}_{\neg ijt}$ from the same two-week period in the previous year: $\Delta\bar{v}_{\neg ijt} \equiv \bar{v}_{\neg ijt} - \bar{v}_{\neg ijt-26}$. Because SARS lasted for less than a year, the lagged component of this variable always captures pre-SARS utilization. Although the size of the lag is arbitrary, a one-year difference implicitly removes seasonal variation from $\Delta\bar{v}_{\neg ijt}$. Regressions in which $\Delta\bar{v}_{\neg ijt}$ is constructed with a six month difference yield similar results.

Our identification strategy exploits the distinction between longtime community residents ("non-movers") and people who have recently joined the community ("movers"). Since

people form social connections over time, recent arrivals to the community are less socially connected (Jackson 2009).[9] To distinguish between these two types of people, we first calculate the overlap in patient traffic between all pairwise combinations of townships. Next we determine the modal township by year for each patient and define a move as a transition across townships that have low overlap. A person becomes a mover by joining his or her 2003 township in 2001 or later. According to this definition, 6 percent of the population qualifies as movers. The regression data set oversamples this group (movers are 40 percent of the regression sample) to increase statistical power along this dimension. Probability weights in the regressions below restore the representativeness of the sample.

The Taiwan CDC provides SARS incidence data. SARS cases may be "reported," or "probable." 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). 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. To permit a delay between the development and communication of SARS risk, we construct both s and $\Delta\bar{v}$ as sums over periods $t-2$ to t.

Summary statistics based on the pre-SARS period appear in Table 1. Panel A, which displays patient characteristics, shows that patients average 0.04 visits per two-week period to a particular physician $\times$ facility. The patient is diagnosed with a respiratory infection such as a sore throat or cold in 37-40 percent of these visits. Movers and non-movers appear similar, although movers are younger and more likely to be male. These groups have similar levels of income: movers earn US$ 30 less per month than non-movers.[10] Movers also

[9]This identification strategy relies on heterogeneous exposure to social learning across different subsets of the peer group. Cohen-Cole (2006) and Blume et al. (2011, Theorem 2) show the assumptions under which this approach identifies social learning.

[10]Income data are available for 62% of the sample, and are based on BNHI estimates of earnings by occupational category.

belong to more peer groups than non-movers. Because of the large sample size (there are 72 bi-weekly observations per patient × peer group), many small differences are statistically significant.

Panel B summarizes the characteristics of peer groups. Movers and non-movers belong to cohorts with similar characteristics, although the peers of movers are younger and more likely to be male. Movers comprise a similar proportion of the peer groups of movers and non-movers, and physicians serving these groups have a similar age and gender composition. The peers of movers visit less frequently than the peers of non-movers, however the breakdown by diagnostic category is similar.

4 Estimation

4.1 Empirical Approach

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

$$\begin{aligned} v_{ijkt} &= \beta_1 s^l_{kt} + \beta_2 s^n_t + \beta_3 S_t N_i \Delta\bar{v}_{\neg ijkt} + \beta_4 v_{ijkt-26} \\ &+ [\text{levels and pairwise interactions of } S_t, N_i, \text{ and } \Delta\bar{v}_{\neg ijkt}] \\ &+ \alpha_{jk} + \delta_t + \epsilon_{ijkt} \end{aligned} \tag{20}$$

In this specification, i indexes the patient, j indexes the physician × 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, $v_{ijkt-26}$.[11]

[11]Including $v_{ijkt-26}$ as a control variable is preferable to using Δv_{ijkt} as the dependent variable because it avoids endogeneity that may arise because of 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 $v_{ijkt-26}$. The most direct solution to this problem is to use $v_{ijkt-26}$ as a control

The 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, $\Delta\bar{v}_{\neg ijkt}$. S_t indicates 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.[12] 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 conveyed by 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 reflect the influence of common unobservables that jointly determine these variables (Manski 1993, Manski 2000). The SARS period interaction ensures that any confounder must exhibit a differentially strong influence during SARS to threaten identification. A remaining concern is that heterogeneous supply shocks during SARS may induce a correlation between the visits of group members. Another concern 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. 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 of this regression is that common unobservables with a differential impact during SARS apply equally to movers and

rather than a component of the dependent variable in equation (20).

[12]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 lagged visits with s^l, s^n, and $S_t N_i \Delta\bar{v}_{\neg ijkt}$ are 0.002, 0.007, and -0.021 respectively.

non-movers.

4.2 Baseline Results

Results based on specification (20) appear in Table 2. 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 elasticity with respect to local incidence ranges from -0.0006 for probable cases to -0.0010 for reported cases. The elasticity with respect to national incidence is six to eight times larger: -0.0050 for reported cases and -0.006 for probable cases.[13] Local information contributes little to the response to SARS.

Columns 2 and 4 add subjective peer assessments to the regression with the proxy $S_t N_i \Delta \bar{v}_{\neg ijkt}$ and the associated pairwise interactions. The coefficient estimate is statistically significant and implies a response elasticity of 0.0035. 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 61-65 percent and the national incidence response by 23-30 percent. In Figure 7, which plots these elasticities, national public information has the greatest impact, followed by social learning and local public information. Although the figure facilitates a comparison of effect sizes for the variables in the regression, our model makes clear that these effects do not map directly into structural learning parameters. Column 5 estimates a baseline specification, which we will compare to other results below. In lieu of SARS incidence variables, this model includes a complete set of time fixed effects. This modification slightly increases the social learning estimate.

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

4.3 Robustness

The remainder of this section evaluates the robustness of the social learning result. Table 3 varies the peer group definition under alternative fixed effects specifications. The baseline specification above utilizes $\Delta\bar{v}_{\neg ijkt}$ and $S_t\Delta\bar{v}_{\neg ijkt}$ to control for unobservable shocks. In contrast, the odd columns of Table 3 also utilize a peer group $\times$ SARS fixed effect, $\alpha_{jk}S_t$. This specification controls for arbitrary unobservables that may differ between the pre-SARS and SARS periods. Under the baseline peer group definition, which appears in Column 1, the estimate diminishes by 24 percent but remains significant.

The even columns of the table utilize a peer group $\times$ time fixed effect, $\alpha_{jk}\delta_t$. This specification, which is even more restrictive, absorbs 55-60 percent of the variation in the dependent variable. In Column 2, the estimate diminishes by 50 percent but remains significant at the 10 percent threshold. The remainder of the table replicates these specifications while defining the peer group by facility (Columns 3-4), township (Columns 5-6) or county (Columns 7-8). Results are robust and statistically significant under these alternative formulations.

For the estimates above, we define a mover as anyone who has arrived in his or her 2003 community after 2000. In our data, tenure in the community may range from 1 to 7+ years. To test the sensitivity of our results to this definition, we interact $S_t\Delta\bar{v}_{\neg ijkt}$ with dummies for these tenure values. Figure 8 plots the coefficients and confidence intervals from this regression. In the figure, the response to peers is uniformly higher among non-movers than among recent arrivals.

The preceding analysis examines how a patient's visits to the doctor affiliated with peer group j respond to the private signal of peers from group j. In reality, a patient may also incorporate the private signals of peers from groups other than group j. We construct $S_tN_i\Delta\bar{v}_{\neg ijkt}$ across an individual's other peer groups by dividing the total number of visits to other groups by the total membership of those groups. We also examine the complementarity between group j and other groups by interacting the signals from group j and the signal from other groups.

Estimates based on these specifications appear in Table 4. Column 1 shows the response to the private signals of group j and other peer groups, which are both positive and significant. The coefficient estimate for other peer groups exceeds the estimate for group j. In principle, people may rely more heavily on either source of information: although the signal from group j is more specific to the risk in group j, the signal from all other groups is more precise. Column 2 shows the interaction between the private signals of group j and other groups. The interaction term is negative and significant, suggesting that the signals of different peer groups are substitutes as information sources. Substitution among private signals is a reasonable conjecture if the cost of searching for information is important.

Neither of the results in Table 4 is likely to arise through a spurious correlation. Our primary concern (which the difference-in-difference specification already addresses) is that common unobservables may jointly determine v_{ijkt} and $\Delta\bar{v}_{\neg ijkt}$. For the result in Column 1 result to be spurious, an omitted variable must cause a correlation between a person's visits to group j and the visits of his peers in other groups over and above any correlation with his group j peers. This would require unobservable shocks to occur both at and beyond the particular peer group. Although both types of shocks are plausible, it is unlikely that shocks at groups other than j would more strongly influence an individual's visits to group j than the group-j shocks themselves. In Column 2, it is also unlikely that a negative interaction between the signals in group j and other groups would arise spuriously. To yield a spurious result, shocks of adjacent groups would need to be negatively correlated. The difficulty rationalizing these results as spurious correlations further validates our interpretation of social learning.

Table 5 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. The remainder of the table, which distinguishes among diagnosis categories, may explain this finding. Social learning exerts a particularly strong influence on respiratory visits, with no evidence of an effect on visits for critical or chronic diagnoses. As Figure 4 illustrates, the SARS response among respiratory visits lasted through the end of Quarter 4. Social learning may have contributed to the relatively large and prolonged response in this category.

4.4 A Complementary Identification Strategy

As an alternative to the difference-in-difference identification strategy, a regression may address common unobservables by controlling for $\bar{v}_{\neg ijkt}$. Holding $\bar{v}_{\neg ijkt}$ constant, the patient receives a stronger risk signal from $\Delta\bar{v}_{\neg ijkt}$ if $\bar{v}_{\neg ijkt-26}$ is high. In a specification that includes both $\Delta\bar{v}_{\neg ijkt}$ and $\bar{v}_{\neg ijkt}$, social learning is identified through variation in $\bar{v}_{\neg ijkt-26}$.

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

The $\bar{v}_{\neg ijkt}$ control addresses common unobservables directly. 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. This approach has the advantage that it does not require an assumption that movers are a suitable control group.

Regressions based on this specification appear in Table 6. Column 1, which utilizes peer group and time fixed effects, yields an estimate that is similar to the baseline difference-in-difference result (Table 2, Column 5). The lack of identifying variation within the peer group prevents us from implementing this approach under more restrictive fixed effects specifications. Column 2 distinguishes between the signals from peer group j and other peer groups: estimates conform with earlier results although the effect of other groups is insignificant. Column 3 examines the interaction between the signals of group j and other groups. The

interaction term is negative and significant at the 10 percent threshold with a magnitude that resembles the result in Column 2 of Table 4. The similarity of these estimates despite their reliance on different sources of variation suggests that these approaches have adequately dealt with confounding factors.

4.5 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. Our methodology should not find evidence of social learning as an explanation for this phenomenon. 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 appear in Table 7. The table implements both identification strategies described above and examines the effect of a signal from group j alone (Columns 1 and 2), group j and other groups (Columns 3 and 4), and the interaction between group j and other groups (Columns 5 and 6). In contrast to the baseline finding of a positive and significant social learning coefficient, the results in Columns 1 and 2 are negative but insignificant. In Columns 3 and 4, estimates for group j and other peer groups are insignificant and have opposites signs. Columns 5 and 6 show an insignificant but positive interaction between the signals of group j and other groups, the opposite of the SARS finding. In summary, these results indirectly support our methodology by failing to find spurious evidence of social learning during Chinese New Year.

5 Dynamic Simulation

In this section, we simulate the dynamic response of visits to the SARS epidemic in order to decompose the aggregate response by information source. 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 to SARS risk, we iteratively predict the response to peer behavior in the previous period and then update peer behavior by aggregating these individual responses. This simulation complements the regression analysis from the last section. While our specification cannot identify structural parameters, it serves as a predictive model that captures different sources of information. By manipulating the model and the estimation sample, we selectively close off certain channels of learning and simulate the response path of visits under alternative learning environments.

5.1 Simulation Methodology

The observed response to SARS in Figure 2 consists of a response to public and private information, as well as unobservable shocks. Our simulation is based on a thought experiment in which we sequentially remove social learning, peer group shocks, and learning from public information from the aggregate response. To implement this exercise, we focus on movers rather than non-movers because for non-movers, the response to peers may be correlated with the responses to unobservable shocks and public information.[14] In contrast, it is plausible that movers do not respond to social learning.

The simulation includes four counterfactuals, which we summarize in Table 8.[15] In

[14] $S_t N_i \Delta\bar{v}_{\neg ijkt}$ and $N_i \Delta\bar{v}_{\neg ijkt}$, which reflect social learning by non-movers, are correlated with $S_t \Delta\bar{v}_{\neg ijkt}$ and $\Delta\bar{v}_{\neg ijkt}$. Limiting the sample to movers eliminates this concern.

[15] 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.

the first counterfactual, people respond to public SARS information, peer group shocks, and social learning. As a predictive model, we estimate a variant of equation (20) on the combined sample of movers and non-movers.[16] We simulate the visits of movers by iteratively generating projections using the coefficient estimates from this model. The social learning coefficients (on regressors $N_i\Delta\bar{v}_{\neg ijkt}$ and $S_tN_i\Delta\bar{v}_{\neg ijkt}$), which apply only to non-movers in the regression, also determine the visits of movers in the simulation.

The second counterfactual preserves the response to public information and peer group shocks but shuts down the social learning channel. We construct two versions of this counterfactual to reflect the ambiguous distinction between social learning and other unobservable shocks. Both versions of this counterfactual reproduce the first counterfactual but set the coefficient on $S_tN_i\Delta\bar{v}_{\neg ijkt}$ to zero. In Version 1, the predictive model continues to include $N_i\Delta\bar{v}_{\neg ijkt}$ as a regressor that represents unobservable shocks, although some variation in this variable also reflects social learning. This interpretation reflects a restrictive view of social learning. For Version 2, we drop N_i and all associated interactions and estimate the predictive model with the subsample of movers. Because this version does not treat the effect of $N_i\Delta\bar{v}_{\neg ijkt}$ as an unobservable shock, it allows for a larger contribution of social learning to the overall response. Therefore Version 2 reflects an expansive view of social learning.

Under the third counterfactual, people only respond to public information. As a predictive model for this scenario, we modify Version 2 of Counterfactual 2 above by setting to zero 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 $\Delta\bar{v}_{\neg ijkt}$, social learning does not contaminate 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

[16] Our specification deviates from equation (20) 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.

provides a benchmark for comparison to the other counterfactuals.

5.2 Simulation Results

Figures 9 and 10 show the paths of aggregate visits and respiratory visits under the counterfactuals described above. The simulation focuses on respiratory visits because Figure 4 and Table 5 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 and dash-dotted lines show results for Versions 1 and 2 of the second counterfactual, which excludes the response to social learning. The difference between either of these lines 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 either the dashed or dash-dotted lines represents the response to unobservable peer group shocks.

Our simulation of visits for all diagnoses suggests that SARS incidence (public information) 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 the time visits reached a low point in Period 13 (nearly three months into the epidemic), unobservable shocks and social learning led to nearly half of the continued suppression in visits.[17] By the end of the epidemic in Period 16, visits remained 20 percent below normal.[18] Depending upon whether it is restrictively or expansively defined, social learning contributes from one quarter to one half of the visit suppression that cannot be explained by the response to

[17]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.

[18]This result differs slightly from the finding in Table 5 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.

actual SARS incidence. We find qualitatively similar results for respiratory visits in Figure 10. Visits drop further and social learning plays a larger role, explaining no less than one third of the visit suppression that is not in response to actual SARS incidence.

6 Conclusion

Emergencies such as the 2003 SARS epidemic occur with regularity. During an emerging outbreak, terrorist attack, or other emergency, the private response to risk may affect the severity and duration of the crisis. Facing incomplete information, people may respond to risk signals from both public and subjective private sources. This paper argues that people learned from the private risk assessments of their peers during SARS. Our results indicate that this private signal influenced the response to SARS more than local public information. According to a dynamic simulation based on regression estimates, social learning both exacerbated and extended the response to SARS.

The private response to SARS has important implications for this and other epidemics. Witnesses have suggested that health care avoidance during SARS probably limited the spread of infection and shortened the epidemic. Although SARS may or may not return, other emergencies are likely to exhibit similar dynamics. Policymakers need to understand better the prevalence response mechanism in order to determine the optimal policy response. Under the limited information that is available during an emergency, social learning appears to influence behavior in important ways.

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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

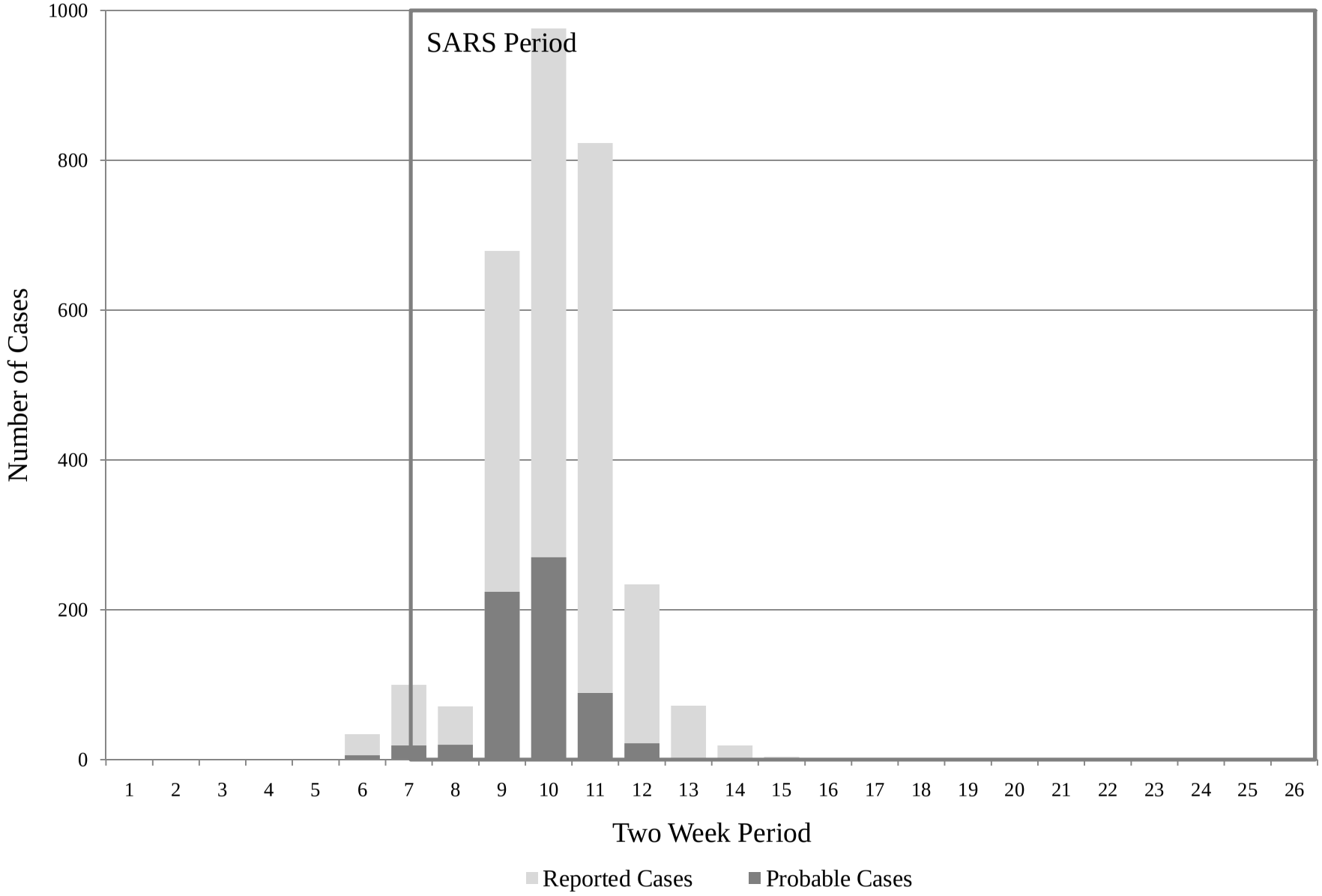

Figure 2: Aggregate Outpatient Visits by Two-Week Period: 2001-2003

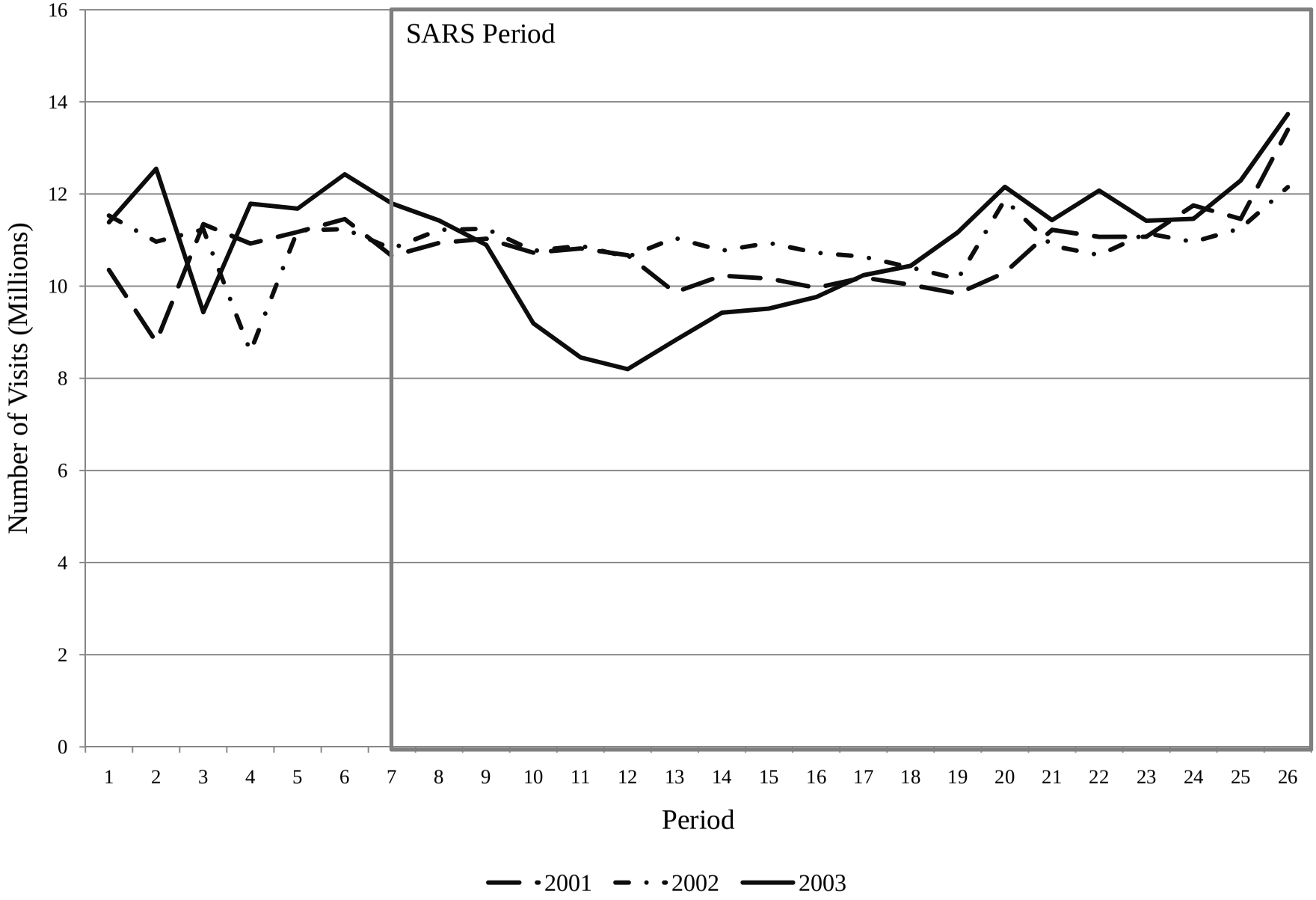

Figure 3: Visits for SARS-Affected and Unaffected Townships During 2003

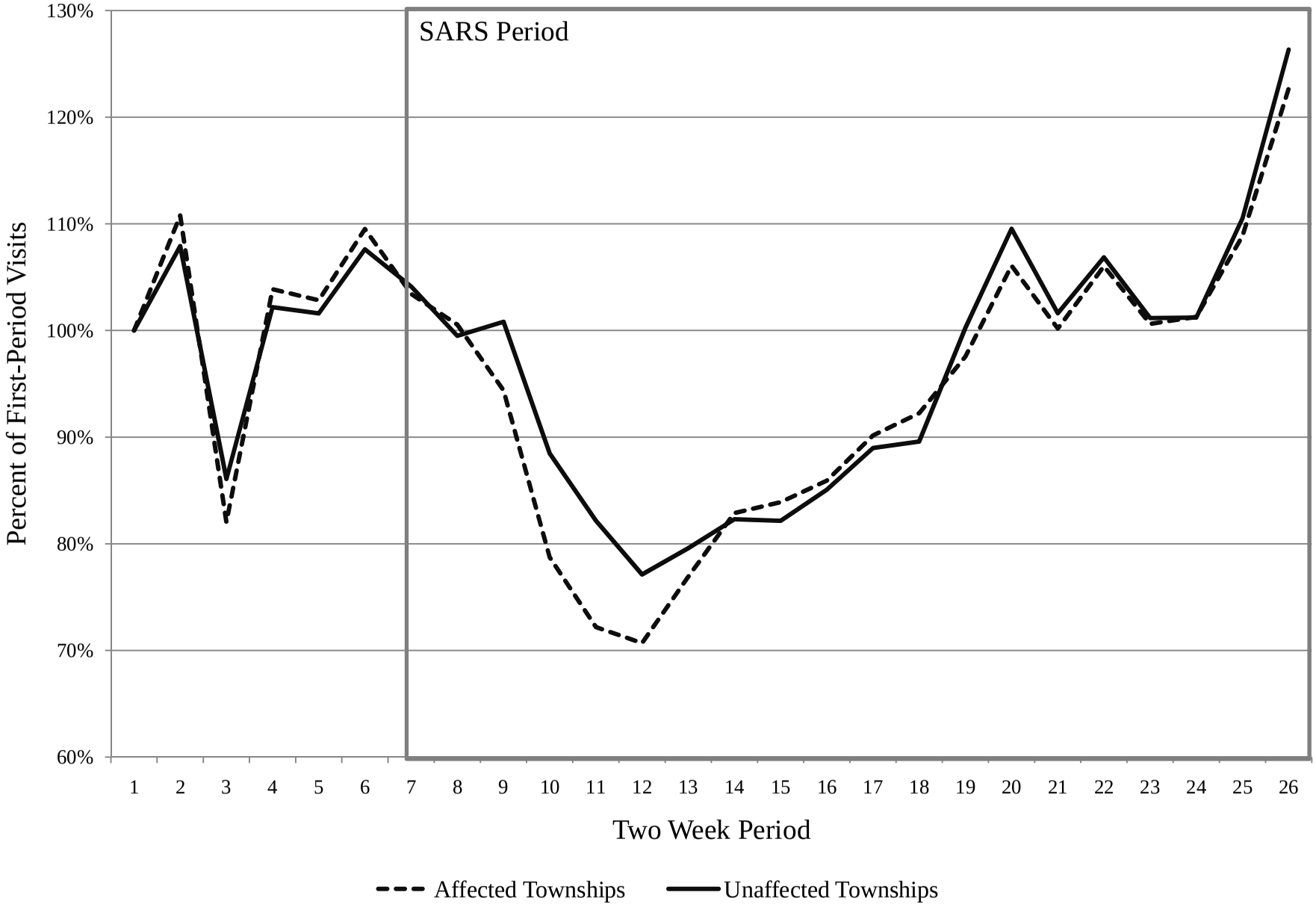

Figure 4: Visits by Diagnosis During 2003

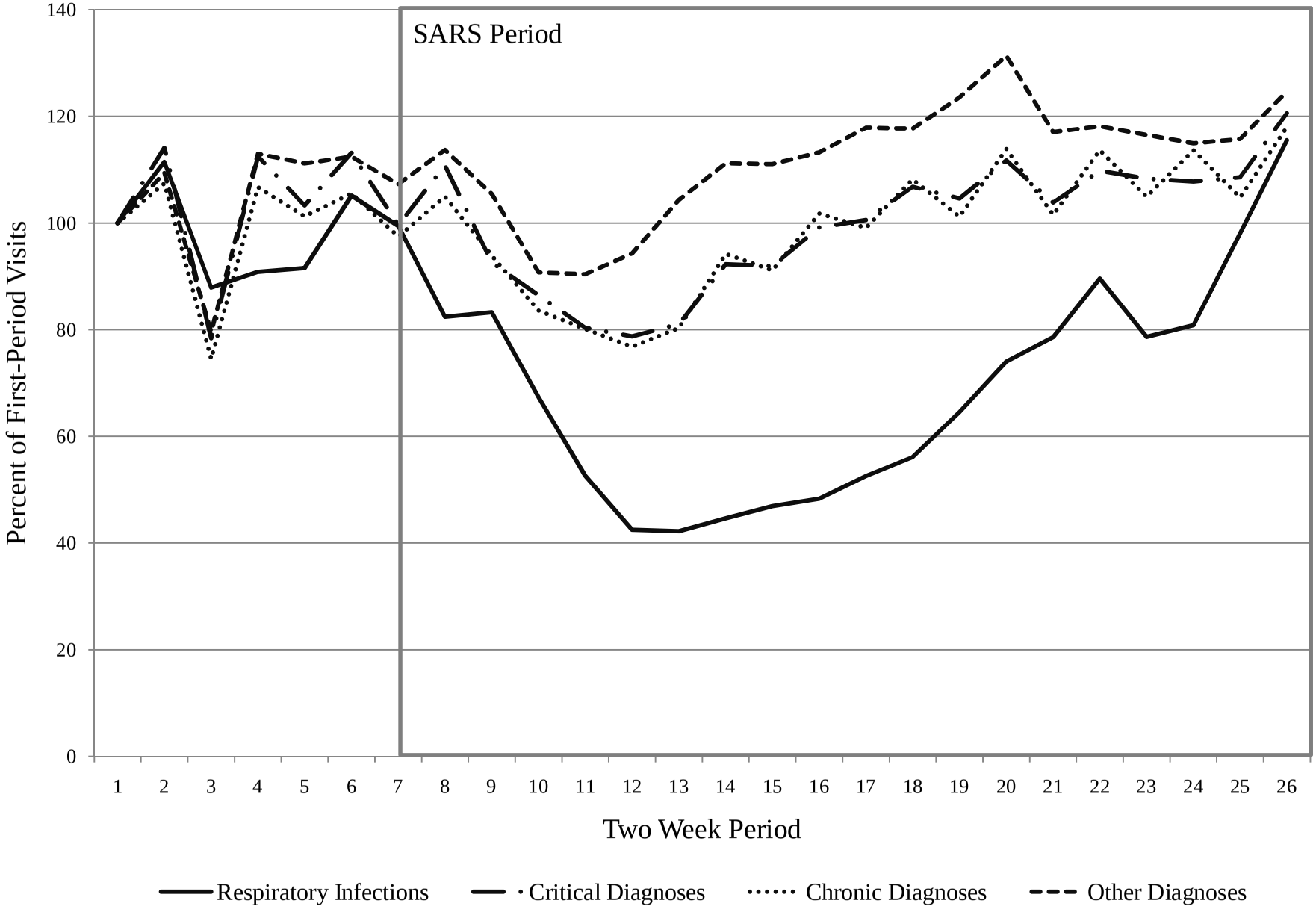

Figure 5: News Coverage of the SARS Epidemic

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

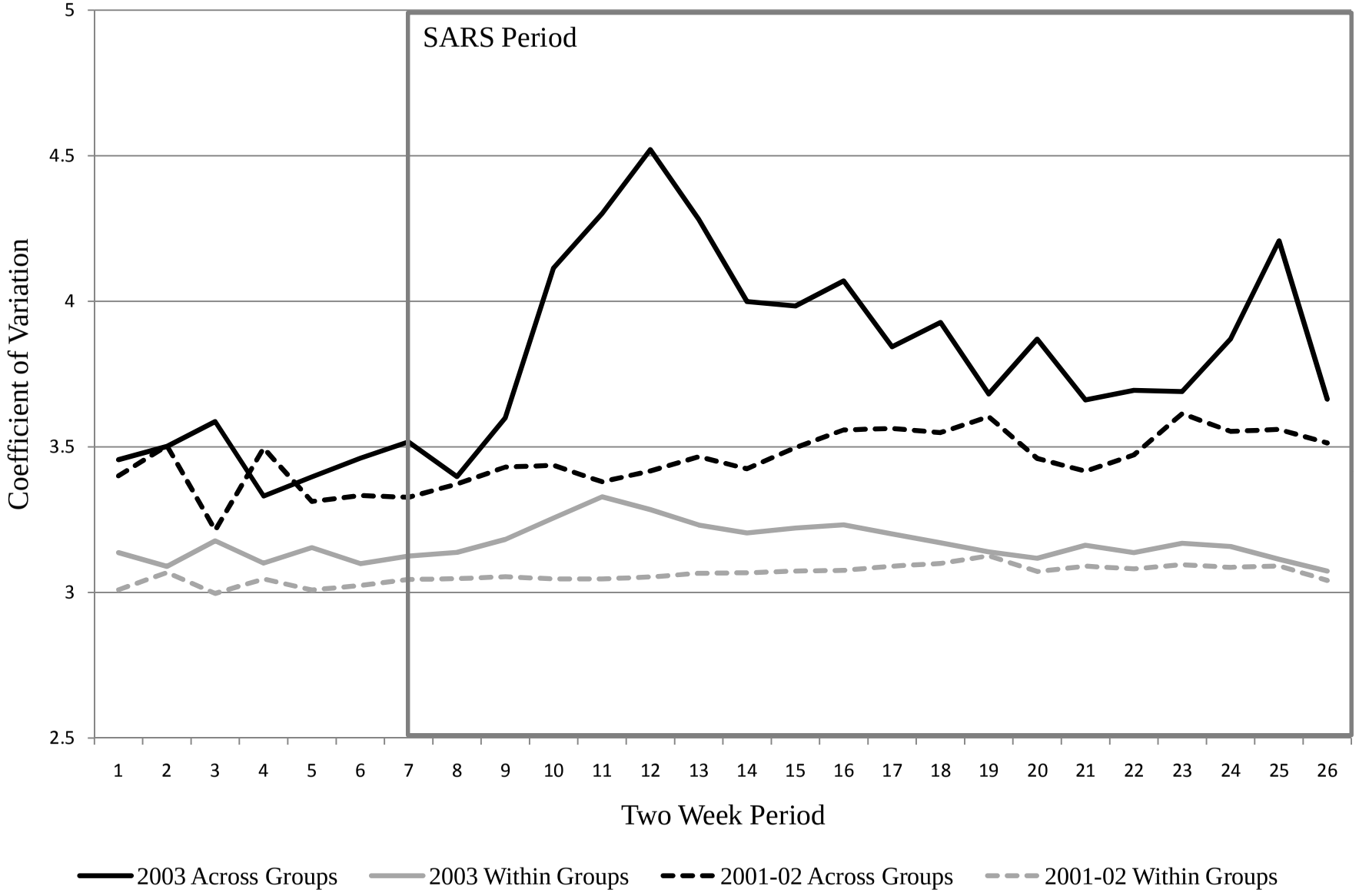

Figure 7: the Prevalence Response Elasticity by Information Source

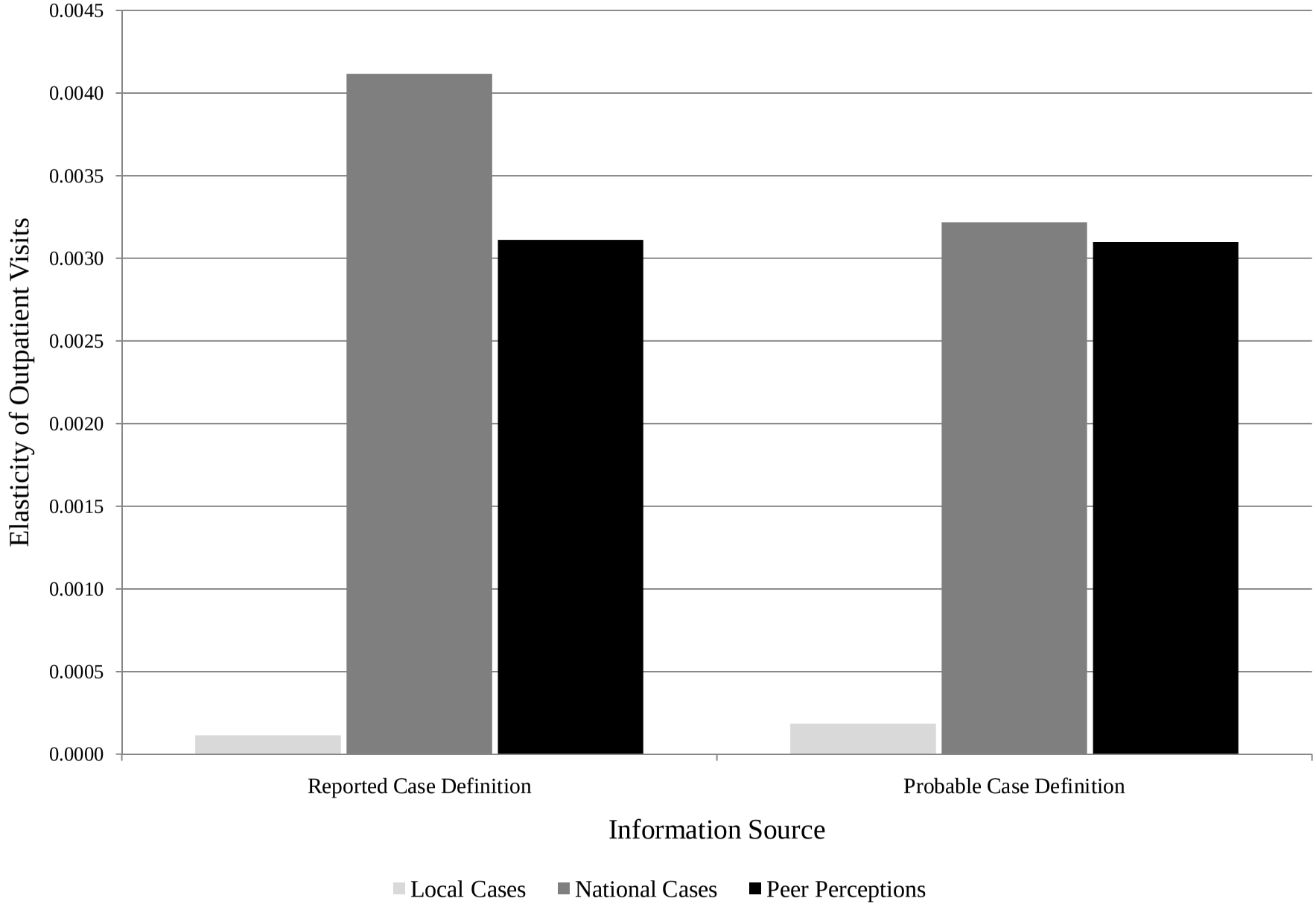

Figure 8: Social Learning by the Patient's Arrival Status

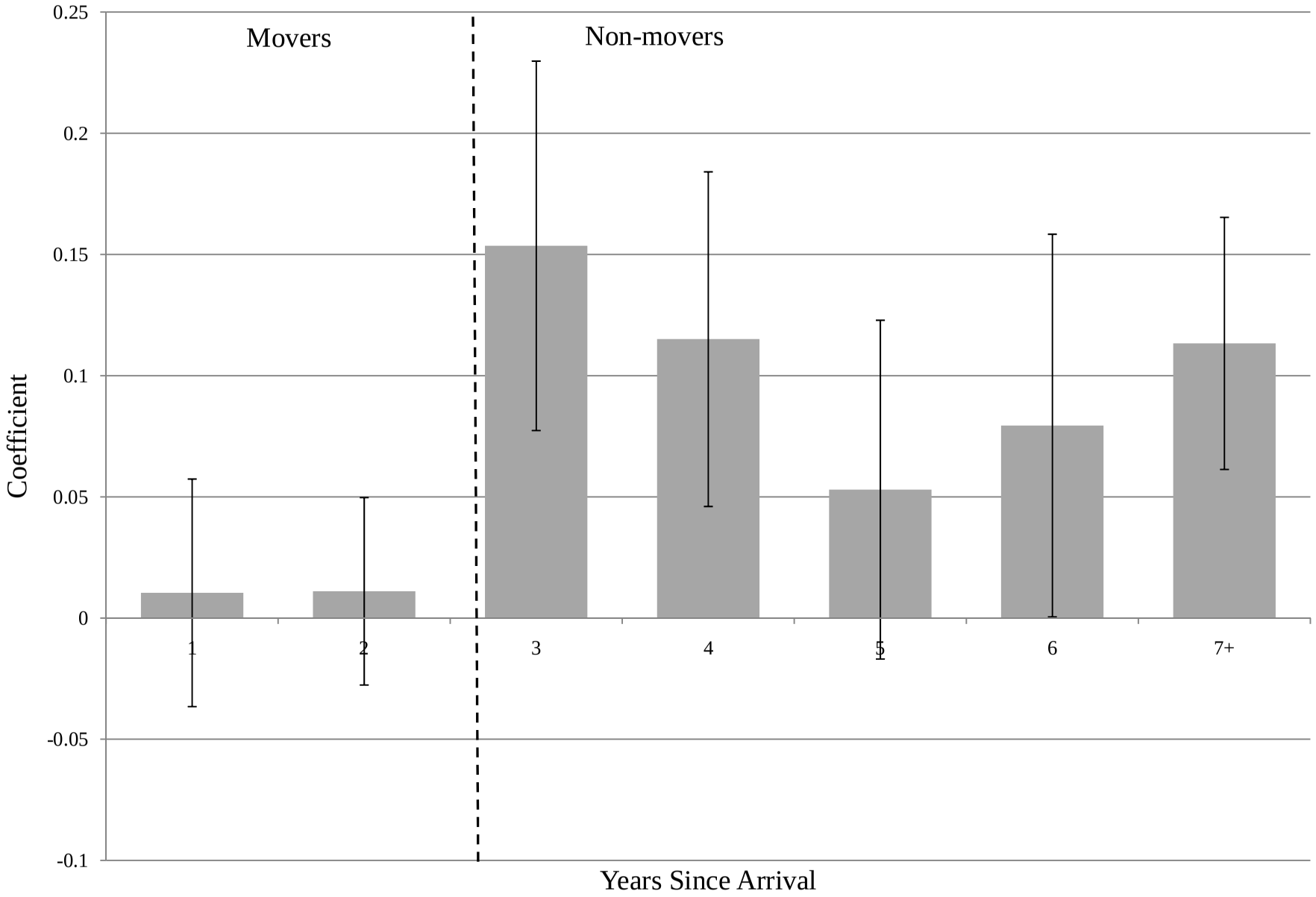

Figure 9: The Simulated Path of Aggregate Visits Under Alternative Scenarios

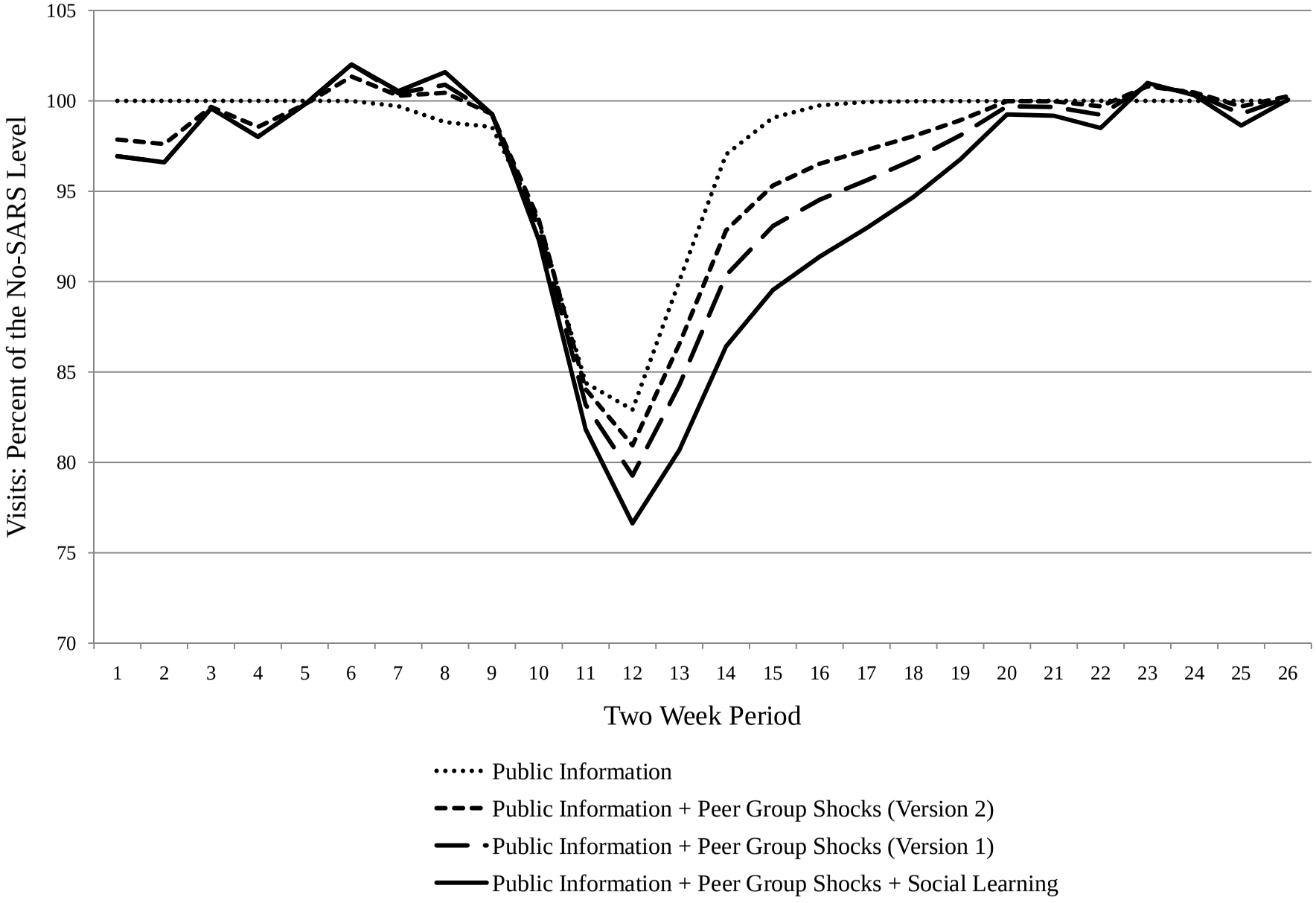

Figure 10: Simulated Path of Respiratory Visits Under Alternative Scenarios