Is SARS a Poor Man's Disease?[†]

Socioeconomic Status and Risk Factors for SARS Transmission

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Abstract

This paper investigates the link between socioeconomic status (SES) and the spread of Severe Acute Respiratory Syndrome (SARS) in Hong Kong. A negative and significant correlation is identified between income and SARS incidence rates, while no similar relationship is found using education level. Areas with more white-collar workers experienced a higher incidence rate, largely driven by the share of service workers and sales personnel. Identifying the income-SARS link is useful for future epidemic-control planning. The unanticipated nature of the epidemic and existing evidence on SARS patient treatment and health behavior adoption suggests that the income-SARS link is likely to be causal. Pre-SARS housing values, a proxy for permanent income and consumption of housing services, accounts for the income-SARS link. Controlling for an array of living conditions does not diminish the income-SARS link. No evidence is found for a correlation between either homeownership or income inequality and SARS incidence.

Introduction

The 2003 Severe Acute Respiratory Syndrome (SARS) epidemic illustrates the type of low-risk, high-cost threat of infectious disease that is expected to become increasingly common in a more integrated world economy. During the 2003 epidemic (March-June), 8,096 people were infected, 774 lives were lost and the economic cost in terms of lost GDP for East and Southeast Asian countries is estimated to be US$18 billion. (Asian Development Bank 2003a) The epidemic threat of outbreaks like SARS is contained by an international effort through information sharing, public education, surveillance, and intensive tracing and home confinement of SARS cases and their close contacts. It involves a large government effort and as in any highly communicable disease, the externalities are high. (World Health Organization, WHO) Knowing which populations are most vulnerable to diseases like SARS should be an important part of the containment strategy. This paper investigates how socioeconomic status (SES) related to the risk of contracting SARS, using data from the most severely hit city in the 2003 epidemic, Hong Kong.[1]

The link between health and SES is robust and well-documented in social sciences, leading to questions about the link's origins and welfare policy implications. (Goldman 2001, Deaton 2002, Currie and Stabile 2003) So far there exists no evidence for or against a SES-SARS link.[2] This paper first provides an estimate of the SARS incidence rate for 295 large-scale housing complexes (estates) in Hong Kong and then extracts evidence for a SES-SARS link from an intra-city analysis. The SARS incidence rate is used as a measure of the spread of SARS,

[1] The SARS incidence rate in Hong Kong, at 0.258 per thousand, was the highest among all SARS-affected cities (World Health Organization). There were 299 SARS deaths in Hong Kong, accounting for more than a third of all SARS deaths.

[2] According to the WHO, SARS risk factors include close contact to SARS cases, a low baseline health status and environmental contamination. There has been no mention of a direct SES-SARS link. No public health measure was taken with explicit consideration of such a link during or after the 2003 epidemic.

considering the process of generating the first SARS case as the same as that of the number of SARS cases conditioned on positive SARS incidence. Section 6 explicitly tests this assumption and provides supporting evidence on this specification.

Significant variation in the SARS incidence rate across the 18 districts in Hong Kong (Figure 1, Table 1A) and its correlation with median income levels are suggestive of a link between SES and the spread of SARS.[3] The SES-SARS link, causal or not, has direct implications on the optimal public health strategies on surveillance and disease containment.

The complexity of the SES-health association has fueled debates over health care policy and wealth transfer (Deaton 2002, Meer, Miller and Rosen 2003). In addition to a direct casual impact of SES on health, a SES-health link can be a result of differential access to health care, variation in awareness or health-related behavior, or the impact of health on SES. The relative significance of each of these factors has varying policy implications (Deaton 2002).

Several characteristics of the SARS epidemic suggest that more can be said about the nature of a SES-SARS link found in the Hong Kong sample. First, vigorous epidemic measures and assignment of all SARS patients to specific restrict-access wards minimizes the difference in quality of epidemic control measures by SES. Second, Lau et al. (2003) find that appropriate precautionary measures were practiced by over 90 percent of the Hong Kong population during the SARS epidemic.[4] This indicates a low level of variation in health behavior adoption. Third, because SARS was a newly discovered disease, a measure of historical SES status before the

[3] Regressing the SARS incidence rate on district fixed effects shows that there was no clear dichotomy of geographical areas into high- or low-risk districts. Section V provides more details.

[4] The measures include mask wearing, frequent hand washing, avoidance of crowded places, living-quarter disinfecting. While the educated or healthcare professionals might be expected to adopt precautionary measures more efficiently, a survey of community doctors (General Practitioners, or GPs) reveals that some clinical practices such as frequent hand washing between patients were not followed. (Wong et al. 2004)

epidemic reflects little or no sorting behavior related to SARS. Therefore a SES-SARS link is unlikely to be due to reverse causality.

This paper explores the origins of the SES-SARS link in two steps. First, three measures of SES are compared: income, education and occupation. Income demonstrates the most robust relationship with the spread of SARS, with an independent, positive effect of employment in one of the occupations with high population contact rate.[5] As early as 1872, Friedrich Engels (1872) argued that the lower income areas where "workers are crowded together are the breeding places of all those epidemics". Income can affect SARS incidence through one of the following: consumption, psychobiological impact, escape. Higher purchasing power or permanent income relates to expenditures on goods that might contribute to the functioning of the immune system or general health status (e.g., living conditions, healthcare and nutrition).[6] There is evidence that socioeconomic circumstances have biological effects on immune functions. (Brunner 1997) Higher-income households presumably also found it easier to leave Hong Kong when the epidemic struck.

Using data on pre-SARS housing sales and rental prices and living conditions, I posit both permanent income and living conditions explain the income-SARS correlation.

When controlling for housing sales or rental prices, the income-SARS link becomes insignificant while the occupation-SARS link remains robust. I further explore the importance of various measurable living conditions and a few noteworthy results emerge. Estates with a higher number of floors per building and facilities such as health clubs or childcare centers experienced

[5] Occupations are divided into five broad categories. The high-contact occupations include professionals/ associate professionals, manager/ administrators, and service workers/ shop sales workers. The other two occupation categories are craft workers/ machine operators, and elementary workers.

[6] Environmental factors were important at least in one super-spreading event. (Hong Kong Department of Health 2003, WHO 2003) Household overcrowding and lower population density in less expensive, more remote residential areas both impact the transmission of SARS, in opposite directions.

a higher incidence rate, possibly through environmental contamination (e.g., elevators) and a meeting-point effect. So did estates with a higher proportion of students and workers using public transportation. On the other hand, household crowding, proximity of health care establishments, building age or whether it is a public or private housing estate demonstrate no systematic relationship with the SARS incidence rate. Even when controlling for all relevant measures of housing conditions, the income-SARS correlation remains robust. Comparing this result and the case when both income and housing price are included suggests that factors aside from living conditions that are related to housing prices, e.g., permanent income and non-housing consumption, contributes to the income-SARS link. I find no significant role of income inequality or homeownership in the income-SARS link.

This paper is organized as follows: the next 2 sections provide an epidemiology of SARS and a timeline of the epidemic in Hong Kong; Section IV reviews related literature; Section V describes the data; Section VI presents empirical findings; Section VII discuses the model specifications and Section VIII concludes.

1. SARS Epidemiology

The causative agent of SARS is a newly identified coronavirus (*SARS-CoV*) that is sufficiently infectious to cause a very large epidemic if unchecked, but controllable with public health measures such as early detection, quarantine and treatment of SARS patients. On average 2 to 4 people are infected by each SARS patient in the absence of any control measures.

Transmission mechanism of SARS is through deposit of virus through respiratory exudates and contaminated surfaces on membranes of mouth, nose or eyes. The risk of transmission increases within confined spaces, such as elevators and airplanes. Environmental

factors such as sanitation and density are likely to have played a role in some outbreaks. (Lipstitch et al., 2003; Riley et al., 2003; Hong Kong Department of Health and WHO websites)

Effective epidemic control measures include reduction of population contact rate, promotion of personal and environment hygiene (frequent handwashing, mask wearing and disinfecting living quarters and shared facilities such as elevators), and detection and isolation of SARS cases.

It is not yet clear why some virus-carriers demonstrated higher-than-normal infectivity in "superspreading events" (SSEs), where single individuals infected as many as 300 others.[7] Possible explanations include mutated strains of the virus, differences in modes of transmission and a much skewed population contact rate distribution. For comparability I have excluded the 3 SSEs from my sample.

2. Timeline of the 2003 SARS Epidemic in Hong Kong

The first SARS cases in Hong Kong are now known to have occurred in February 2003. Figure 2 shows a timeline. At least 125 people were infected around March 3, 2003 in the Prince of Wales Hospital, forming the first SARS cluster (Riley et al., 2003). When 7 residents in Block E of Amoy Gardens, a high-density private housing estate, were diagnosed with SARS on March 26, 2003, the community transmission of the disease – i.e., its spread in the local community outside the group of close medical and family contacts of SARS patients – was confirmed by the government.

After the Amoy outbreak, there was a large-scale shutdown of normal activities. Most people either stayed at home or wore surgical masks, while all schools were suspended on March

[7] *Science**xpress***, May 23, 2003. (http://www.sciencemag.org/cgi/rapidpdf/1086925v1.pdf); *Science*, Lipsitch et al. (2003), Riley et al. (2003).

29 for more than 3 weeks. Residents were infected across the board, including the educated, the young and the previously healthy. A high level of vigilance was displayed by the government and international organizations.[8] Specific restrict-access SARS wards were set up, isolating all known SARS cases.

The epidemic was declared contained after three months on June 23, 2003, 21 days after the last case in the territory was isolated. 1,755 people in Hong Kong were infected and 300 died from the disease. Less than a quarter of the SARS cases in Hong Kong were health care workers and most of the almost 400 infected residents in Amoy Gardens were strangers to each other.[9]

3. Literature on SES-Health Gradient

There is a large body of literature demonstrating the positive variation in health status by socioeconomic status (SES). Feinstein (1993) and Goldman (2001) provide a detailed review of the related studies.

Several main themes about the SES-health link emerge. First, the variation of health status by SES is gradual, and it exists at all levels of SES, not just limited to a poverty effect due to deprivation. This is supported by studies of developed countries, including the Whitehall studies (Marmot et al. 1984, 1991 and 1995) that focus on British civil servants, none of whom is poor, and the proportional income-mortality relationship of the type identified in Rogot et al. (1992), which is constant at all income levels.

Second, the SES-health relationship remains robust regardless of the choice of measurement of the SES, including income, education and occupation, or the country studied.

[8] The Hong Kong government discouraged travel and public gatherings, closed schools, quarantined individuals and encouraged improving personal and environmental hygiene.
[9] Source: *The Standard*; *Oriental Daily*; WHO website.

Moreover, different health indicators are shown to have a relationship of somewhat different strength with SES (e.g., Hurd et al. 2003).

Third, recent studies provide some evidence that the observed SES-health relationship is more than a reverse causal impact of health on SES, i.e., people in poor health drift towards the bottom of the SES distribution, or a third factor effect, where factors such as height affecting both income and health later in life (Goldman 2001).

4. Data

A. SARS Incidence Rate

While disaggregate data on the number of SARS cases below the district level are not recorded, the Hong Kong Department of Health provided the total number of cases in the territory and the number of cases in the 4 SSE sites with the largest clustering of cases. A daily "List of Buildings of Confirmed SARS Cases" (SARS-list henceforth) published by the Department of Health during the epidemic contained addresses (up to the building level) of all SARS-affected sites on that day. I estimate the number of SARS cases in each housing estate by counting the number of times any building within each housing estate was put on the SARS-list, and then multiplying the number by the average number of SARS cases per listing, excluding the 4 most severe sites. The estate-level SARS incidence rate is the ratio of the estimated number of SARS cases to the housing (SARS list henceforth) estate population. Subsection 2B below describes how estate population is calculated.

The reader should be aware that there are several sources of error in my estimate. First, the SARS-list started on April 12, 2003, more than 2 weeks after the Amoy outbreak when community-level transmission of the disease was confirmed. Second, the variation in the number of cases per listing implies that my estimate is at best a crude measure of the relative severity of

the outbreak in the listed buildings. Third, because the SARS-list was published to encourage stringent precautionary measures and self-monitoring of health conditions, especially for residents that might have contact with SARS patients, buildings were only kept on the SARS-list within 10 days of hospitalization of the last SARS patient from that building. If there was more than a 10-day lag between the hospitalization of the patient and the diagnosis of SARS, the incubation period was considered to have passed, and the building in which the patient lived would not be put on the list. Therefore some buildings with SARS cases might have never appeared on the SARS-list.

To assess how accurate the estimation method I have adopted for creating the estate-level SARS incidence rate is, I repeat the same estimation process for all 18 districts, using a district instead of a housing estate as the unit of observation. Next I compare the district-level estimates with the actual district-level SARS incidence rates provided by the Department of Health. The two measures have a correlation of 0.96. The 2 measures are plotted against each other in Appendix A.[10]

B. Measures of Socioeconomic Status and Other Resident Characteristics

Hong Kong is divided into 18 Districts. District-level population density is calculated using population data from the 2001 Hong Kong Census and land area data from the National Bureau of Statistics of China.

Demographic and socioeconomic profiles of estate residents are proxied by Census 2001 data at the building-group level. Building groups (a total of 2,817 in Hong Kong, covering all 39,028 residential buildings) are divided according to building characteristics such as location,

[10] Both the estimated district-level SARS rate and the actual SARS incidence rate are derived using the Census 2001 population.

type, age and height. (Fung 2005) Population-weighted averages are taken for each estate across building groups to which at least one building of the estate belongs. Tables 1A and 1C contain summary statistics.

C. Pre-SARS Housing Sales and Rental Price Data

To measure pre-SARS housing values, I have obtained access to transaction records of all sales and purchases of housing units in Hong Kong during the years 1993-1998 and 2001-2002.[11]

Housing estates are large-scale housing complexes, consisting of many almost-identical blocks of housing units. The substantial similarity of units within each housing estate ensures that the average price level will be a reasonable reflection of housing values within that estate. Only estates with at least 2 transactions per month on average during the period 1993-1996 are included in my sample, for a more accurate measurement of price levels. A site of super-spreading event (Amoy Gardens), suspected to have been struck by a particularly virulent strain, is excluded. Together the 295 housing estates in my sample encompass more than 1.5 million people, about 23 percent of the Hong Kong population. They are situated in 17 of all 18 districts in Hong Kong, except for the *Islands* district that contains the outlying islands with a population of 86,667 (1.3 percent of territory total; Census 2001). There are 58 public housing estates in my sample; excluding them does not change the results quantitatively or qualitatively.

Because of potential outliers, I use the median transaction prices as an indicator of housing values.[12] Mean prices have a correlation in excess of 0.99 with the median prices in each

[11] Data for years 1993-1998 are kindly shared by Tsur Sommerville. Purchase of data for years 2001-2002 was generously supported by a grant from the Andrew M. Mellon Foundation through the Research Program in Development Studies at Princeton University. Both data sets are based on Memorial Day Book of the Hong Kong Land Registry that records all sales and purchase instruments registered with the Registry, subject to the provisions of the Land Registration Ordinance, which prevent a loss of priority to any subsequent registered transactions.

[12] For example, it is not uncommon for housing units on the top two floors to be duplex units or penthouses. These units usually cost more than double most other housing units in that housing estate.

year for the 295 estates in my sample. Using mean prices as an indicator of housing values produces very similar results. The housing price data is supplemented with data on monthly mortgage and rental payment from the 2001 Census.

D. Estate Characteristics and Living Conditions

I compiled data on the characteristics of the housing estates that might be related to the spread of SARS including: age, average flat size, availability of estate facilities (such as health clubs, shopping arcades or childcare centers), number of floors, number of flats per floor, and number of blocks.[13] To generate an estimate of each estate's -population, I multiplied the total number of flats (blocks X floors X flats per floor) by the number of households in each housing unit, and the number of persons in each household. The last two measures are district-level averages from the Hong Kong Census 2001.[14] I define the average flat space per person as the ratio of the estate-average flat size to the district-average of persons per housing unit.

I measure the travel time to city center from a housing estate, defined as the amount of time spent on the most prevalent form of public transport to the closer of the two main commercial/ financial centers in Hong Kong, Tsim Sha Tsui and Central.
Information on travel time to city center was collected from real estate agents and transportation companies.

Availability of health care facilities is checked on the website of Centaline Ltd., a leading property agent in Hong Kong. Under the map function, the numbers of three types of health care facilities can be searched within a north-facing 64m X 80m ($0.51km^2$) rectangle with each

[13] These data were compiled by research on the internet, phone calls to real estate agents and property developers, and visits to some of the estates. Age and the number of floors and flats per floor are averages across the housing estate; number of blocks are often counted from site plans of the estates.
[14] There is not a lot of variation across districts. Mean [s.d.] of the number of households per quarter is 1.02 [0.03]; Mean [s.d.] of the number of persons per household is 3.16 [0.19].

housing estate in the center: medical establishments (general hospitals and clinics, dental hospitals and a variety of health care facilities, both private and public), community doctor/ GP clinics and all other health-related facilities (such as pharmacies, dental clinics and Chinese medicine practitioners). Medical centers apparently unrelated to SARS, such as dental hospitals or optical care centers, are excluded from the *medical establishment* variable and added to the number of *all other health-related facilities*. Information on whether the housing estate is public is obtained from the Housing Authority.

5. Empirical Findings

As a first step, I estimate the following Tobit model:

$$(1) \qquad SARSP_i = \alpha + \gamma_d + \varepsilon_i \qquad (i = 1, \ldots, 295)$$

$SARSP_i$ refers to the SARS incidence rate of housing estate *i* bounded between 0 and 1, α is a constant term, γ_d a district fixed effect, and ε_i is a normally distributed error term with density function $N(0, \sigma_i)$. I weight the regression by the total number of flats in each estate to adjust for heteroskedasticity, assuming that σ_i^2 is inversely proportional to the size of the housing estate. If we consider the estate-level SARS incidence rate to be the average of 1-0 outcomes (infected/ not infected) of all estate residents, then the variance of the error term will be of the form σ^2/N_i where N_i is the number of residents. Section 6 assesses whether this assumption is appropriate.

District fixed effects are not significant as a group (p-value = 0.25), nor is a district-level population density measure (p-value = 0.35). Because the lack of evidence towards a simple

classification of districts into "high-risk type" and "low-risk type", the district-level variables are omitted from results presented.[15]

Besides, I do not find any strong support for a spatial correlation of the spread of SARS. Regression of the estate-level SARS incidence rate on the self-excluding district-average incidence rate does not suggest a significant link, despite the upward bias due to feedback effects (Case 1991, Manski 2000). Therefore in the rest of the paper I focus on the SARS incidence rate of each individual housing estate as independent.

A. Socioeconomic Status and SARS

As discussed in Section V, the SES variables are measured at the building-group level in three dimensions: income, education and occupation. Table 2 shows the regression results of the following Tobit model weighted by the total number of units:

$$(2) \qquad SARSP_i = \alpha + \beta\, SES_i + \varepsilon_i \,.$$

Higher household income levels at either the upper quartile, median or lower quartile correlate with a lower SARS incidence rate (columns 1-3). Measures of personal income levels produce similar results. As the rest of the empirical results will show, the SES-SARS link found is the most stable along the dimension of income.

Column 4 shows the link between SARS and the share of working population in different occupations. The SARS incidence increases most significantly with the share of workers employed in service industries/ shops and secondly for professionals, against a baseline category of elementary, agricultural and fishery and other unclassified workers. The share of managers, administrators and craft workers in the workforce does not correlate significantly with the SARS

[15] For robustness checks, district dummies (or district-level population density) are added to all regressions presented in this paper but neither control ever has statistical significance at 10%.

incidence rate. Likely explanations include the workers' high contact rate with the general population (and thus SARS cases) and the likely proximity of shops and other places where the public convene and the disease spreads. Part of the link between SARS incidence and the share of the workforce who are (associate) professionals can be due to the inclusion of healthcare workers and other client-based professionals (such as consultants) in the category. Similar results are derived in column 5 using two broad occupation groups.

One hypothesis that supports an SES-SARS link is that the more educated adopt appropriate health habits more efficiently. Columns 6 and 7 show no significant relationship between SARS incidence and the education level, despite the high correlation (0.7-0.8) between the education attainment measures and median household income level. This discounts the differential health behavior story, consistent with findings in Lau et al. (2003).

Marmot (2002) argues that full participation in the society might be as important as the purchasing power derived from income. Columns 8 and 9 in Table 2 do not offer much support that this mechanism was at work in the case of SARS. The last two columns in Table 2 demonstrate the income-SARS link and the occupation-SARS link are more or less independent.

B. Possible Channels of the Income-SARS Link

Housing service consumption is expected to increase with current income level, permanent income and the quality of housing services (which can in turn reflect preferences related or unrelated with SES). To explore the income-SARS correlation identified in the previous section I regress:

$$(3) \qquad SARSP_i = \alpha + \mathbf{Income}_i \beta + \mathbf{H}_i \gamma + \varepsilon_i$$

where H_i is a measure of value of housing service consumption of the households and other variables defined as before. Four separate indicators are used: building group-level

monthly rental price, estate-level average sales price in years 1995-98, 2001-2002, estate-level average sales price in year 2002, and building group-level monthly mortgage payment. While rental price can be expected to best reflect the user cost of housing without influence of price expectations and other macroeconomic factors, it is derived from Census 2001 at the building-group level only. No data closer to the 2003 epidemic or at a more disaggregate level is available. Mortgage payment is likely to have the least accurate indicator out of the four, being both measured by the building group level and affected by factors such as when the mortgage was taken out and structure of the mortgage. Nevertheless, Table 3 demonstrate that different housing service value indicators produce results that point in the same direction. The main conclusion from this analysis is that the income-SARS link is reduced in both size and statistical significance when housing service value is controlled for, while the occupation-SARS link remains robust.

C. Measurable Living Conditions

One can think of the rental or sales price of a housing unit as the market value of a vector of living amenities and neighborhood qualities, some of them (such as social capital) unobservable. In this section I explore numerous measurable aspects of living patterns and conditions (***L***) that might have been proxied by housing service value:

$$(4) \qquad SARSP_i = \alpha + \mathbf{Income}_i\,\beta + \mathbf{H}_i\,\gamma + \mathbf{L}_i\,\tau + \varepsilon_i\,.$$

One notable result in Table 4 is that while some living condition indicators have a significant correlation with SARS incidence, the income-SARS link remains robust throughout. Also, comparing column 1 with the rest of the table, neither the magnitude of the income-SARS link or that of the occupation-SARS link experiences any noticeable reduction. Results using the other 3 income level indicators are similar and available upon request.

The proportion of students and workers who use public transport relates a higher SARS incidence rate (column 2). This is consistent with WHO (2003) which recommends special consideration to be given to confined spaces including aircrafts and vehicles. A higher incidence rate in estates with facilities such as health clubs and childcare centers (column 3) might either be due to a higher usage rate during the epidemic (as compared to residents in other estates who stopped using similar facilities outside their estates because of the general wariness of transportation) or a lower level of environmental hygiene. The number of floors per building correlates with the sharing of elevators with a sawtooth pattern. As column 4 shows, it is significant at 10%.[16] These are the risk factors that demonstrate a consistent relationship with SARS incidence using different income measures.

On the other hand, the proximity to health-related establishments (public and private hospitals, general practitioner clinics and pharmacies) does not correlate with the spread of SARS. Similarly, three indicators that potentially capture population and living density – travel time to city centers, space per person in the average unit and the average number of units per floor – do not have a strong relationship with SARS incidence (columns 5-8).[17] The average age of buildings within the estate generally relate to more depreciation of less modern facilities, but it does not show any significant impact in column 9. Column 10 shows that any difference in building management and the level of general building maintenance between private and public estates does not create a gap between the two types of estates in terms of SARS incidence.[18]

[16] If, say, there is an additional elevator bank for every 15 floors, elevator-sharing increases with the number of floors up to 15 floors, but the amount of elevator-sharing in a 16-story building is only as much as that in an 8-story building, and it keeps increasing until the number of floors reaches 30.
[17] Travel time is significant at 10% but this result is not robust using other income indicators.
[18] One potential bias is that many public housing estates are rental only, and they are excluded from my sample because I do not observe any open market transactions for them. While half of the Hong Kong population live in public housing estates, only about a quarter of my sample are public (Table 1A).

D. Testing Other Related Hypotheses

This section examines whether the homeownership rate and income inequality at the building group level affect the SARS incidence. There is evidence in the literature that homeowners have more incentives to invest in local amenities and social capital due to low mobility (e.g., DiPasquale and Glaeser 1999). Because environmental contamination is a risk factor for SARS and there are clear externalities of maintaining a hygienic environment during the epidemic, it is interesting to explore the role of homeownership in this setting. An editorial in the British Medical Journal (1996) proposes that income distribution can play an important role in health, while Waldman (1992) draws a link between income inequality and infant mortality. Because the disease in concern is an acute condition that might affect people with lower baseline health status, an income inequality-SARS link will be of second-order.

I explore the impact of homeownership and income inequality on the SARS incidence rate, controlling for the income level, share of workers in high-contact occupations and living conditions that are significant in the previous section. Columns 2-4 of Table 5 show the results and Column 1 is for comparison. The percentage of homeowners correlates with SARS incidence only at 15% significance. Experimenting with different functional forms or restricting the sample to private estates leads to similar results. There is no strong evidence for homeownership being an important determinant. Similarly, any second-order effect of income inequality is not apparent in the data.

6. Model Specification and Heteroskedasticity

A. Restrictions of the Tobit Model

A more general approach to study the impact of various factors on the spread of SARS is to distinguish between the impact of those factors on whether a housing estate is affected by

SARS at all, and on how severely it is affected, conditional on it being affected. Following Cragg (1971) and Lin and Schmidt (1984), these two relationships can be expressed as follows:

(5) $\Pr(SARSP_i \leq 0) = 1-\mathbf{\Phi}(X_i\beta_1)$

$\Pr(SARSP_i > 0) = \mathbf{\Phi}(X_i\beta_1)$

(6) $\Pr(SARSP_i = y_i \mid SARSP_i > 0) \sim N(X_i\beta_2, \sigma^2)$,

where $SARSP_i$ is the SARS incidence rate. $\mathbf{\Phi}$ refers to the standard normal cumulative density function, and $N(\cdot)$ the normal distribution. X_i are the explanatory variables. (5) can be estimated by the Probit model, and (6) by the truncated regression model. The Tobit model imposes the condition that $\beta_1 = \beta_2/\sigma$ and maximizes the following likelihood function:

(7) $\Pr(SARSP_i=0) = 1-\mathbf{\Phi}(X_i\beta/\sigma_i)$

$\Pr(SARSP_i = y_i \mid SARS_i=1) = 1/\sigma_i * \phi(y_i-X_i\beta/\sigma_i) / \mathbf{\Phi}(y_i\beta/\sigma_i)$,

where ϕ the standard normal probability density function. If this condition is not satisfied, the Tobit model is misspecified. In results not shown here, the truncated regression and the Probit models are estimated separately and a log-likelihood test is performed following Greene (2000). For all regressions presented in this paper the null hypothesis that the Tobit restriction is valid is not rejected at 1% level. Results are available upon request.

B. Heteroskedasticity

One way to correct for heteroskedasticity is to estimate and test some kind of assumption on the error term variance, σ_i^2. Note that the estate-level SARS incidence rate is an average of 1-0 values, defined by whether a resident is infected by SARS or not. This gives rise to an inverse relationship between σ_i^2 and the number of flats (T_i):

(8) $\sigma_i = \sigma * T_i^{\delta}$

The weighted Tobit regressions presented in this paper restricts δ to be -0.5 (columns 1 and 2). In columns 3 and 4 I relax this assumption.

Lastly, one can model a linear relationship between σ_i and all or some of the explanatory variables (Maddala 1983, Rutemiller and Bowers 1968). I experiment with various specifications and the total number of flats seems to have the most robust relationship with σ_i:

$$(9) \qquad \sigma_i = \sigma + \omega T_i ,$$

A test of heteroskedasticity amounts to a test of $\omega = 0$. Note that columns 3 and 5 suggest the adsence of heteroskedasticity because neither δ nor ω is significant. However, Columns 4 and 6 indicate the opposite. δ is estimated at 0.20 under Column 4, giving support to the specification of Column 2, which is equivalent to the weighted Tobit model. All regressions discussed in this paper are replicated without restricting the value of δ in equation (8) and results similar to those presented earlier are obtained.

7. Conclusion

This paper investigates the association between socioeconomic status and the spread of a communicable disease, SARS. Understanding SARS incidence is important for devising epidemic control strategies and public health policies. Given that SARS is unlikely to be the last of the emerging diseases posing a global epidemic threat, it is worth considering what lessons we can learn from the 2003 SARS epidemic.[19]

A significant and negative association between SARS incidence and income is identified, after controlling for the share of population in high-contact occupations. The nature of the identified SES-SARS link is likely to be largely causal. Because SARS is a new and unanticipated disease, it cannot have directly led to sorting among the population into housing

[19] *Science*, Dec 2003. (http://www.sciencemag.org/cgi/reprint/302/5653/2045.pdf)

estates according to their susceptibility to SARS. The prodigious level of public health effort to combat SARS makes differences in access to suitable health care an unlikely explanation. Moreover, widespread adoption of precautionary practices implies that differential adoption of health habits is likely to be small.

Analysis using measures of housing sales and rental values as a proxy for quality of living environments suggests that an important channel of the income-SARS link is through living conditions. Estates with higher usage of public transportation, estate facilities and a higher number of floors (perhaps related to elevator-sharing) experienced a higher SARS incidence rate. Proximity to health-related establishments, household crowding, distance from city centers and the average age of the buildings do not show similar correlations, neither do homeownership rate and income inequality. The robustness of the income-SARS link despite the living condition controls suggests that permanent income plays an important role.

While much is still unknown about SARS, partly due to data limitations, this paper contributes to our understanding of the spread of SARS. It also provides new evidence on the SES-health link in the setting of a low-risk but high-cost event. None of the government measures used to combat SARS during and in the aftermath of the 2003 epidemic was devised with a link between SARS and economic conditions in mind.[20] Given the findings in this paper, it is worth taking the SES-SARS gradient into account when formulating the optimal strategy of surveillance and control of the disease.

[20] http://www.info.gov.hk/info/sars/pdf/checklist-e.pdf

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