Is SARS a Poor Man's Disease?

The Relationship between Housing Values and Health[†]

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Abstract: This paper investigates the relationship between socioeconomic status (SES) and the spread of SARS. I focus on the SARS infection rates in 295 large-scale Hong Kong housing complexes. Using pre-SARS property values as indicators of SES, I identify a negative SES-SARS link. I also find that density, proximity to the city centers and availability of communal facilities increase the spread of SARS. Building age, flat size, proximity to medical establishments, formation of residents' association or whether the housing complex is public are not significantly associated with the SARS infection rate. The SES-SARS relationship remains robust after controlling for living conditions. I provide evidence that household income is likely to be an important component of the SES-SARS link.

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1. Introduction

A strong positive association between socioeconomic status (SES) and health has been well documented in the literature of economics, sociology and medical science, using data from different time periods and populations. This association has been identified using job rank, education, income and wealth as measures of socioeconomic status, and with a variety of health outcomes, including self-reported general health status, mortality and the incidence of a broad range of infectious, mental and chronic illnesses. The relationship appears to be persistent over time and space.[1] This paper studies the cross-sectional relationship between housing values and the Severe Acute Respiratory Syndrome (SARS) infection rate in different parts of Hong Kong.

Intuitively, the correlation between SES and health might well vary by the measure of health status. For example, Hurd et al. (2003) find that economic status is more highly correlated with reported chronic conditions than with acute conditions. Poterba (2001) argues that one ultimate goal of research in this area is to classify health conditions into a small set of categories by sensitivity to measures of SES, and then investigate determinants of these measures. By studying the link between SES and SARS, I gain insight into the mechanisms by which SES affects health, at least for the case of a communicable disease outbreak.

SARS is the first new illness of the 21st century to threaten international health with global epidemic potentials. It infected 8,422 people worldwide, killing 916 of them, during the outbreak in 2003. The economic cost of the SARS outbreak is estimated to

[1] See Goldman (2001) and Deaton (2002).

range from US$10 billion to US$30 billion.[2] While close interactions of people with livestock and wild animals in low-income countries, notably China, greatly increases the likelihood of a viral disease outbreak, it is unclear whether low-income families are more at risk once an outbreak occurs.[3] It is logical that overcrowding in low-income households increases the risk of contracting SARS, because it is transmitted by respiratory means and person-to-person contact. On the other hand, lower population density in areas further away from the city centers, mostly occupied by lower-income households, might have decreased the spread of SARS. Whether there is any systematic relationship between pre-SARS SES and the spread of SARS, directly causal or otherwise, has direct implications on epidemic control strategies and general public health policies. So far, no epidemic control strategy or preventive measure has been formulated with reference to a possible SES-SARS link.

Hong Kong is of particular interest not only because it was the most severely SARS-hit city in 2003, accounting for more than a third of all SARS deaths in the world, but it also reveals a striking variation in SARS infection rate by district (Figure 1, Table 1A). Column (4) in Table 1B suggests some systematic differences in housing complexes affected by SARS and those that were not. Less than a quarter of all SARS cases in Hong Kong were health care workers, and most of the almost 400 infected residents in the most severe site were strangers to each other. A report by the Hong Kong Department of Health suggests that environmental factors, such as contaminated sewage and communal facilities, might play an important role in the spread of SARS in the most severe site,

[2] Robertson (2003). The 1994 locally-contained outbreak of plague in India is estimated to have cost US$2 billion.
[3] *Nature*, 2003. (http://www.nature.com/nature/focus/sars/sars2.html#why)

Amoy Gardens.[4] There were frequent references in the media to large differentials in the extent to which districts were affected by SARS, as shown in Figure 1. However, it is not obvious whether this classification by district is appropriate or useful, or what factors might have been responsible for this apparent differential in the spread of SARS by district.

The complexity of the SES-health association that has fueled heated debate over health care policy and wealth transfer.[5] The observed SES-health association says nothing about the direct causal effect of wealth (or more generally some correlates of the SES such as income or education) on health; rather, it can be a result of differential access to health care, awareness or health-related behavior, or the impact of health on SES.[6] The relative importance of each of these factors has very different implications on the appropriate public health policy (Deaton 2002). I argue that the association between housing value and the SARS infection rate is at least partly attributable to a direct casual link of SES to health. There are several reasons why the nature of the epidemic helps me disentangle different plausible explanations behind the SES-SARS association.

First, vigorous epidemic control measures were adopted by the government to identify and quarantine suspected and confirmed SARS cases, including enhanced disease surveillance, public education and provision of information, and intensive tracing and home confinements of close contacts of SARS patients. This effort largely eliminated any discrepancies in access to SARS-related health care between the rich and the poor. Because all SARS patients were assigned to specific restrict-access wards, the hospital

[4] Source: *The Standard*; *Oriental Daily*; WHO and Hong Kong Department of Health website.
[5] For example, see Deaton (2002) and Meer, Miller and Rosen (2003).
[6] Variation in the access to health care and health awareness can be expected to be lower within a population in a developed, modern city like Hong Kong, compared to that in a developing country or across developed and developing countries.

population contact rate or the quality of received treatment was likely to be more or less independent of SES.

Second, if the sales of disinfectant and surgical masks were any hint, one might expect high usage of adaptive health behaviors at all levels of the SES given the intense level of anxiety in the society during the epidemic. In general, there was a high level of vigilance against SARS. Precautionary measures believed to be significant protective factors, such as mask wearing, frequent hand washing, avoidance of crowded places, living-quarter disinfecting, were practiced by over 90 percent of the Hong Kong population during the SARS epidemic (Lau et al. 2003).[7]

Thirdly, SARS is a newly identified disease, and there is no clear link between SARS infection and health history. Many young and previously healthy adults were among the infected or deceased.[8] It is impossible for SARS to have affected historical housing prices or pre-SARS housing wealth accumulation, or for self-sorting among different living locations to have been directly caused by SARS. I argue that the significant correlation between pre-SARS housing values and the SARS infection rate found in my data points to a direct impact of SES on the ease of spread of SARS.

Because my study relies on an objective measure of the SARS infection rate, it is free from the potential bias associated with self-reported measures of health status (Baker et al. 2001). Poorer people might either adapt to various inconveniences of life and underreport illnesses, or have a lower sense of general well-being at every given health level due to relative and absolute SES.

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[7] While more educated people 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)

[8] The median age of all SARS patients in Hong Kong is 40. Source: WHO.

This paper focuses on housing values as an indicator of SES. Recent research suggests that wealth bears a stronger relationship with health status than other aspects of socioeconomic status, such as income, schooling or occupation (Duncan et al. 1999; Hurd et al. 2003), and the connection is not likely to be driven by short-run wealth changes (Meer, Miller and Rosen 2003).

Not only does housing value represent a large proportion of household savings, it is crucially related to social status and living conditions.[9] As early as 1872, Friedrich Engels argued that the lower income areas where "workers are crowded together are the breeding places of all those epidemics".[10] This points to living conditions as the source of the observed SES-health gradient. Tam et al. (2003) find that education level and type/ quality of accommodation are stronger predictors of low reporting rates of infectious intestinal disease, compared to other SES measures such as social class, marital status and occupation. Thomson et al. (2001) point out the lack of vigorous analysis on the direct impact of housing conditions on health. In particular, they do not find any published study on the impact of reducing household crowding on the risk of infectious diseases that addresses concerns on direction of causality and selection. As detailed below, I consider how much the variation of the SARS infection rate can be attributed to an array of factors related to the living environment.

There is another reason why housing value appeals as an indicator of SES. While the current and historical market values of other forms of household wealth (such as

[9] According to a report in 1995, home equity of the median US family represents over 70 percent of its net worth, which is the total value of all real and financial assets, including equity in the home, other real estate, vehicles, own businesses, as well as financial assets. (http://www.savingscoalition.org/wealth.html) The Hong Kong Monetary Authority noted that housing remains the "most important form of saving for many households". (http://www.info.gov.hk/hkma/eng/public/qb200403/fa4.pdf)

[10] Engels (1872), p.43.

vehicles) might not both be easily measured, housing value is observed every time a transaction takes place. I have collected transaction prices for 295 housing estates across Hong Kong for years 1993-1998 and 2001-2002.

In absence of individual-level data on SES matched to street addresses, housing estates are the best available unit of observation. Housing estates are large-scale housing complexes, consisting of 1,600 almost identical units on average in my sample. The similarity of the units and the level of facilities sharing imply that most aspects of living environment are constant within an estate. This allows me to assess the impact of specific measures of living conditions, such as the availability of facilities and formation of owners' corporations, on the spread of SARS.

The size of the estates ensures a reasonable transaction frequency and allows for a relatively accurate and up-to-date measure of housing values. Little variation within each of the housing estates allows me to use the median transaction price as an indicator of average housing wealth within an estate, circumventing problems of unadjusted quality differences in cross-sectional housing data at a more aggregate level and of selection bias in repeat sales data.

Upon identifying a negative association between housing values and the SARS infection rate, I explore the channels through which wealth or some underlying related correlates of SES might have a direct impact on the spread of SARS. Possible explanations include living conditions (e.g., sanitation and sharing of facilities), underlying qualities of residents correlated with wealth or SES (such as ability to learn about health and diseases), and a differential in strength of the immune system due to nutrition, stress and life style.

To investigate how much of this causal link comes from housing characteristics, I assess the link between various (time-invariant) estate characteristics and the SARS infection rate, controlling for pre-SARS housing values. A few noteworthy findings emerge: while estate population, travel time to city centers and availability of estate facilities are positively correlated with the SARS infection rate, flat size, proximity of health care facilities or whether it is a public or private housing estate demonstrate no systematic relationship with the SARS infection rate. Smaller estates are more likely to form incorporated residents' associations, which have no significant independent impact on the spread of SARS, controlling for population. Whether the (incorporated) residents' association or an external company is responsible for building management does not seem to affect the SARS infection rate. Nevertheless, these estate characteristics cannot fully explain the relationship between housing wealth and the SARS infection rate.

Although I do not have information on estate population characteristics, I collected information on a wide range of variables at the neighborhood level. Neighborhoods are District Board constituencies, each with a population of about 17,000.[11] After controlling for neighborhood characteristics such as educational attainment, income and homeownership rate, the inverse relationship between housing wealth and the SARS infection rate remains robust. I conclude that not only were lower-price housing estates more affected by SARS, but factors other than observed living conditions, environment or housing characteristics contribute to the link between housing wealth and the SARS infection rate. While this strengthens the case for improving economic conditions because the poor are doubly deprived (Deaton 2002), I find that a

[11] Source: Hong Kong Electoral Board. 15 of the housing estates in my sample have a population exceeding 17,000, and they span two neighborhoods.

linear, instead of proportional, relationship between property values and the SARS infection rate fits the data better. Therefore, my results do not give support to a redistribution of wealth for improving the average SARS infection rate.

This paper is organized as follows: the next section provides an epidemiology of SARS and a timeline of the epidemic in Hong Kong; Section 3 reviews related literature; Section 4 describes the data; Section 5 presents empirical findings and Section 6 concludes.

2. SARS Epidemiology and Timeline

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. The basic case reproduction number of SARS coronavirus is estimated to be between 2 and 4, which implies an average of 2 to 4 people, on average, are infected by each patient in the absence of any control measures. 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.[12] Possible explanations include mutated strains of the virus, differences in modes of transmission and a very skewed population contact rate distribution. For comparability I have excluded the SSEs from my sample.

SARS is transmitted through the deposit of virus through respiratory exudates, fecal-oral contacts and contaminated surfaces on membranes of mouth, nose or eyes. There is also evidence of higher risk of transmission within confined spaces, such as

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

elevators and airplanes. Rodents and cockroaches have been found to be mechanical carriers of the virus. Environmental factors such as sanitation and density are believed to have played a role in outbreaks in Hong Kong hotels and housing estates. (Lipstitch et al., 2003; Riley et al., 2003; Hong Kong Department of Health and WHO websites)

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

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 29 for more than 3 weeks. Residents were infected across the board, including the educated, the young and the previously healthy. 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.[13]

3. Literature on SES-Health Gradient

There is a large literature demonstrating the positive variation in health status by socioeconomic status (SES). Feinstein (1993) and Goldman (2001) provide detailed reviews 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. Marmot (2002) argues that full participation in the society might be as important as the purchasing power derived from income, and that job status or rank plays a large role in explaining the health gradient. The importance of relative income or rank might lead to a gradual health gradient, regardless of the absolute income levels.

Second, the SES-health relationship remains robust regardless of the choice of measurement of the SES, including income, wealth, education and occupation, or the country studied. Moreover, a wide range of health indicators have been investigated, each of which is 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

[13] Source: *The Standard*; *Oriental Daily*; WHO website.

health drift towards the bottom of the SES distribution, or a third factor effect, where factors such as height affect both income and health later in life (Goldman 2001).

Thomson et al. (2001) provide a review of all studies since the 1880s on how housing conditions affect health. The authors argue that existing studies, regardless of the field, are limited by either the vigor of the analyses, particularly the lack of controlling for confounding factors, the size of the sample, or the representativeness of the study due to the geographical area chosen to be studied.

4. Data

A. SARS Infection 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 infection 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 infection 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 infection 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.[14]

B. Pre-SARS Housing Prices

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.[15]

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

[15] 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

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 transaction 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) 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 (Table 1B). 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 the territory total; Census 2001).

To avoid the impact of potential outliers, I use the median transaction prices as an indicator of housing values.[16] Mean prices have a correlation in excess of 0.99 with the median prices in each year for the 295 estates in my sample. Using mean prices as an indicator of housing values produces very similar results.

C. Estate Characteristics

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 child care centers), number of floors, number

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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.

[16] 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.

of flats per floor, and number of blocks.[17] 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.[18] 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 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*.

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[17] 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.

[18] 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].

Information on whether the housing estate is public (either PSPS or HOS) is obtained from the Housing Authority. Whether there is a residents' association (incorporated or otherwise) and whether it is responsible for building management is obtained from the Home Affairs Department.

D. District and Neighborhood Characteristics

Hong Kong is divided into 18 Districts with 400 District Council constituencies (neighborhoods) under the 18 Districts. A district may contain 8 (*Islands* District) to 38 (*Eastern* District) neighborhoods, depending on the district population. Each neighborhood has population of around 17,000. Tables 1A and 2.1C contain summary statistics.

Demographic, education, labor force, household and housing characteristics of all districts and neighborhoods are obtained from the Census and Statistics Department, based on the most recent Census (2001). Each housing estate is matched to a neighborhood by locating it on the electoral boundary maps.[19]

5. Empirical Findings

As a first step, I estimate the following regression:

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

$SARSP_i$ refers to the SARS infection rate of housing estate *i*, α is a constant term, γ_d a district fixed effect, and ε_i is a normally distributed error term with density function $N(0, \sigma_i)$. Because the SARS infection rate is a probability measure bounded between 0 and 1, I estimate a Tobit regression. 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

[19] Available on the Hong Kong District Council Elections website: http://www.elections.gov.hk/elections/dc2003/english/ebmaps/ebmaps.html

the size of the housing estate. If we consider the estate-level SARS infection 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.

Column (1) of Table 2 shows the results. Despite apparent spatial differential in SARS infection rate (Figure 1), simple district dummies alone do not explain variation among housing estates satisfactorily. Because I did not find any evidence towards a simple classification of "high-risk districts" and "low-risk districts", I omit the district dummies from most of the results presented in this paper.[20]

Besides, I do not find any strong support for a spatial correlation of the spread of SARS. Regression of the estate-level SARS infection rate on the self-excluding district-average infection rate does not suggest a significant link, despite the upward bias due to feedback effects (Case 1991, Manski 2000). Regressing the estate-level SARS infection rates *after April 12, 2003* (or other dates during the epidemic) on the self-excluding district average SARS rate *before April 12, 2003* (or the corresponding cutoff date) points to the same conclusion.[21]

A. Pre-SARS Housing Prices

Next, I explore the relationship between the SARS infection rate and 2002 median housing prices, controlling for estate characteristics by regressing:

$$(2) \qquad SARSP_i = \alpha + X_i \beta + \varepsilon_i \qquad (i = 1, \ldots, 295)$$

[20] I add the district dummies to all regressions presented in this paper to check for robustness, and the dummies are never significant as a group.

[21] The lack of evidence towards a dynamic spatial correlation might be because the dynamics are well specified – I find no correlation within an estate in the *before* and *after* periods either.

As before, $SARSP_i$ is the estate-level SARS infection rate, α is a constant term, and ε_i is a normally distributed error term. X_i is a vector of estate characteristics including the pre-SARS median sales price. A grid search on the model likelihood shows that the linear price level fits the data better than log price level. Using log prices instead produces qualitatively similar results with larger standard errors, suggesting that linear price levels fit the data better. In this paper, I present regression results using linear price levels.

Columns (2) show a negative correlation between pre-SARS housing prices and SARS infection rate in the housing estates, which remains robust after controlling for travel time to city centers in Column (3). Travel time is likely to be negatively correlated with income level since the opportunity cost of time increases with hourly wages. It might also correlate with work location and therefore occupation of the residents.

Column (4) reveals that the SARS infection rate increases with the availability of estate facilities. Although the negative correlation between the facilities dummy and pre-SARS price is rather weak (-0.10), controlling for the availability of facilities increases the estimated coefficient for the pre-SARS price. A similar finding is shown in Column (5) when I control for the estate population quadratic, which proxies for estate density and population contact rate within the estate. I experiment with controlling for measures of density (number of flats per block, number of flats per floor and number of floors per building) but they are not significant after I control for the population quadratic.[22]

For a robustness check, I control for travel time, facilities dummy and estate population quadratic in the same regression, along with the pre-SARS price level.

[22] A grid search based on model likelihood reveals that log population is superior to linear population. Using log population instead of quadratic population gives similar results.

Column (6) shows the results. Although the estimated coefficient for pre-SARS housing price level is somewhat reduced in magnitude, it remains significant at the 5 percent confidence level. In Column (7), I show that the point estimate for the impact of housing price on the SARS infection rate remains similar when district dummies are included, although the inclusion of the dummies decreases the precision of the estimates. The district dummies are not significant as a group.

I investigate the robustness of the relationships shown in Table 2 by controlling for additional estate characteristics. Table 3 contains the results. Surprisingly, average building age does not significantly correlate with the SARS infection rate. There is no significant relationship between the average amount of space available in the flat for each person and the spread of SARS either. Controlling for average flat size in the estate leads to the same conclusion. Comparing this finding with the results in Column (5) of Table 2, where the estate population quadratic is significantly and positively correlated with SARS infection rate, it implies that living density in general environment and estate population contact rate are more important determinants of the spread of SARS. It is possible that close family contacts of SARS patients are at high risk regardless of flat size.

Column (3) of Table 3 shows that the proximity to a variety of health care facilities does not demonstrate a statistically significant relationship with the SARS infection rate. I arrive at the same conclusion when 1-0 dummies are used instead of the number of establishments. Intuitively, this could have affected the SARS infection rate in two opposite directions: concentration of SARS patients and high-risk group (healthcare workers and close contacts) around healthcare facilities increases SARS risk, while the

availability of medical assistance and general awareness might be higher in the same areas.

Moreover, I do not find any evidence that public housing estates (PSPS or HOS estates) were differently affected by SARS than private housing estates. 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 estates in my sample are public (Table 1A).

I also experiment with indicators of the formation of residents' associations, whether they are incorporated, and whether the residents' associations are responsible for building management in each estate. Only 2 of the estates in my sample have not formed any building management body. I find a negative correlation between the SARS infection rate and the establishment of incorporated residents' associations alone, not controlling for other covariates, but not other types of residents' associations.[23] This correlation becomes statistically insignificant after I control for estate population. Smaller estates are more like to form incorporated residents' associations and the impact of the associations cannot be separated from the impact of estate population. There is no significant difference in the SARS infection rate between estates where the (incorporated) residents' association is responsible for building management, and those where an external company is commissioned. These results are shown in columns (6) to (8).

[23] Subject to Building Management Ordinance, any resolution passed at the meeting of incorporated residents' associations (with respect to the control, management and administration of the common parts or the renovation, improvement or decoration of those parts) are legally binding on the management committee and all the owners. Other types of residents' associations are not covered by the Ordinance.

Notably, the pre-SARS housing price remains robust and stable regardless of the various estate characteristics I control for, as reflected by comparing the coefficients of the median housing price in the first row in Table 3 to those in Table 2. Moreover, the associations between the SARS infection rate and the estate characteristics (travel time, facilities and population) remain significant and similar to what is presented in Table 2, when I control for other estate characteristics.

As a final check of robustness, I repeat the estimation in Table 2 with alternative measurements of pre-SARS housing values. Table 4 shows results using various median sales prices (1994, 1998 and averaged 1996-2002) and the level-level median monthly rent in 2001.[24] Rent reflects the value of housing services consumed by household(s) occupying the housing unit, while housing price is the net present value of the entire flow of housing services from the present to the end of the useful life of the housing structure (e.g., Poterba 1984). Therefore one might expect rental prices to relate to the SARS infection rate in a way similar to housing prices. The negative correlation between pre-SARS housing values and the SARS infection rate is stable across different measurements. It is interesting that while the estimated coefficient of median sales price in 1998 is somewhat lower than those of housing prices in other years, probably due to inflation around the peak of the 1995-1998 housing boom, there is only a slight reduction in magnitude of the price impact when estate characteristics are controlled for. This contrasts with the more significant fall in the point estimate for prices in 2002 in Table 2.

I conclude that housing values have a negative effect on the spread of SARS, and part of this effect is linked to living conditions such as population contact rate. Contrary

[24] I use the average of median sales prices in years 1996, 1997, 1998, 2001 and 2002. Sales pries are not available for years 1999 and 2000.

to anecdotal evidence, I do not find any evidence in my data that the SARS infection rate is highly district-specific, or correlated with factors such as building age, flat size, proximity to health care facilities, formation of residents' associations or whether the estate is public.

B. Neighborhood Characteristics

To explore how much of the relationship between housing values and the SARS infection rate was due to resident characteristics that correlate with housing prices, I make use of level-level data from the Hong Kong Census 2001 on education, income, demographics, labor force and housing characteristics.

$$(3) \qquad SARSP_i = \alpha + X_i \beta + Z_n \Omega + \varepsilon_i \qquad (i = 1, \ldots, 295)$$

X_i include pre-SARS sales price in 2002, travel time to city centers, the estate facilities dummy and an estate population quadratic. Z_n are level-level indicators. The 295 housing estates in my sample are situated in 156 neighborhoods, out of a total of 400 neighborhoods in Hong Kong (See Table 1C for summary statistics of neighborhood characteristics). Table 6 summarizes the more interesting results.[25] The first two columns show estimated coefficients for the neighborhood characteristic included in the regression, and for the pre-SARS sales price. The third column shows the estimated coefficient for the neighborhood characteristic in a regression the same as (3) above except that the pre-SARS price is excluded. The fourth column contains the weighted correlation coefficient between the neighborhood characteristics and the pre-SARS price level in 2002.

[25] In addition to regressions presented in Table 5, I experiment with indicators of age composition, marriage pattern and labor force participation rate. I do not find statistically significant relationships between these indicators with the SARS infection rate. Results are available upon request.

When the pre-SARS price is controlled for, none of the neighborhood characteristics has a statistically significant correlation with the SARS infection rate. Remarkably, the measures of education and personal income do not seem to affect the spread of SARS. However, as the second column shows, the pre-SARS housing values cease to be statistically significant when household income is included.[26] Moreover, household income is significant at the10 percent level when the pre-SARS housing price is omitted. This suggests that income makes an important contribution to the impact of housing values on the spread of SARS.

Interestingly, I do not find a significant impact of population age on the SARS incidence rate, although it is an important determinant of the outcome of the disease.[27] There is evidence in the literature that homeowners are better citizens (Dipasquale and Glaeser 1997). Incentives to invest in amenities and to improve their community might translate to a healthier living environment. While I do not find any strong evidence that a higher homeownership rate in the neighborhood reduces the SARS infection rate, Row (10) shows that homeownership rate and household income are jointly significant at 10 percent confidence level in a regression omitting the housing price level. Therefore, despite the lack of definitive evidence, I cannot rule out a potential impact of homeownership on the spread of SARS.

Lastly, I should point out that the coefficients of the estate characteristics (travel time, estate facilities and population quadratic) remain statistically significant and similar

[26] The relationship between the 1996-2002 average housing prices and the SARS infection rate is somewhat more robust, but the general pattern is the same as the one presented in this section.
[27] Hong Kong Department of Health. I also experiment with the proportion of different age groups (<15, 15-64, 65+), and find no significant age effect.

in magnitude as in Tables 2 and 2.4, after controlling for the neighborhood characteristics.

C. Changes in Housing Values

After establishing a negative relationship between measures of pre-SARS housing values and SAR infection rate, I look at changes in housing values. Specifically, I explore the link between changes in housing prices from 1998 to 2002, during which period the Hong Kong residential housing market suffered a price fall in excess of 65 percent on average.[28] I find that the impact of the housing price level is more robust than the impact of housing price changes, as Table 6 demonstrates with different measures of housing values.

More surprisingly, my results point to a larger proportional decrease in housing value leads to a *lower* SARS rate, after controlling for pre-SARS price level and other estate characteristics. The negative relationship between pre-SARS price measures and SARS infection rate remains statistically robust. Indeed the estimated coefficients are considerably larger, compared with Tables 2 and 2.4. The relationships between various estate characteristics and the SARS infection rate remain largely similar to those presented earlier in Column (6) of Table 2. While one might expect decreases in housing values to negatively affect health status, factors that determine the size of the housing bubble at each housing estate in 1997 complicate the interpretation. Nevertheless, it is puzzling that a larger price fall is associated with a lower SARS susceptibility.[29]

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[28] Weighted mean [s.d.] of proportional price change 1998-2002 in my sample is -52.49 percent [13.16 percent].

[29] In another paper, I investigate systematic patterns in the size of the historical price fall in terms of pre-bubble price levels, transaction volume, volatility and other estate characteristics.

6. Model Specification and Robustness to Non-normality

In this section, I investigate the robustness of the findings presented in Section 5. Attention is paid to the restrictions imposed by the Tobit model, and the effect of possible heteroskedasticity in the error term.

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:

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

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

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

where $SARSP_i$ is the SARS infection rate. $\mathbf{\Phi}$ refers to the standard normal cumulative density function, and $N(\cdot)$ the normal distribution. X_i are the explanatory variables for estate *i*. (4) can be estimated by a Probit model, and (5) by the truncated regression model. The Tobit model imposes the condition that $\beta_1 = \beta_2/\sigma$ and maximizes the following likelihood function:

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

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

where ϕ is the standard normal probability density function. If this condition is not satisfied, the Tobit model is misspecified. In results not shown here, I estimated the Tobit

model, the truncated regression and the Probit models separately on (4) above (Greene 2000). I did not reject the null hypothesis that the Tobit restriction is valid.

B. Tobit vs. CLAD

I have applied analytical weights equal to the size of the housing estates on all regressions presented so far. Tobit estimators are inconsistent if the normality condition is violated. There is evidence that heteroskedasticity causes a serious problem in a Tobit model, compared to other censored regression models (Arabmazar and Schmidt 1981, Goldberger 1983, Johnston and DiNardo 1997 and Jolliffe 1999).

The censored least absolute deviations (CLAD) estimator based on Powell (1984) is robust to violation of the normality conditions. Deaton (1997) points out that although the CLAD estimator is clearly superior to Tobit estimators only in large samples (n~1000) due to increase in the estimator variance, comparing the CLAD estimators and the Tobit estimators, provides a guide to whether heteroskedasticity has caused a serious bias. Because more than 75 percent of my sample is censored, I perform the CLAD estimations using the 80^{th} percentile, utilizing bootstrap standard errors. The estimates are presented against unweighted and weighted Tobit results in Table 7. Although Columns (1), (3) and (5) suggest no substantial difference between the CLAD estimators and the Tobit estimators, the inclusion of covariates other than price seems to imply the opposite. Also, it is worth pointing out that the iteration process CLAD, during which sample observations where the predicted values are negative are discarded, reduces the number of observations in the final iteration by 31 percent to 193. This accounts for the imprecise estimates shown. Nevertheless, my results give some support for controlling for heteroskedasticity.

C. Heteroskedasticity

One way to correct for heteroskedasticity is to estimate the error term variance, σ_i^2, assuming a functional form such as 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. I estimate:

$$(7) \qquad \sigma_i = \sigma + \omega T_i ,$$

where T_i is the total number of units in estate *i*. A test of heteroskedasticity amounts to a test of $\omega = 0$. Note also that the estate-level SARS infection rate is an average of 1-0 values, defined by whether a resident is infected by SARS or not. This should give rise to an inverse relationship between σ_i^2 and the number of flats (T_i) such that

$$(8) \qquad \sigma_i = \sigma * T_i^{-0.5} .$$

A Tobit regression weighted by the number of flats amounts to fitting the Tobit likelihood function in equation (6) by maximum likelihood, together with equation (8) above. I also allow for a more general relationship in the same form:

$$(9) \qquad \sigma_i = \sigma * T_i^{\delta} .$$

If (8) is the correct specification, δ should be close to –0.5. The ML estimation results, with restrictions (7) – (9), are presented in Table 8. The first two columns are equivalent to weighted Tobit regressions presented before. In Columns (3) and (5), neither δ nor ω is significant, so there is no evidence of heteroskedasticity. However, Columns (4) and (6), which also control for covariates other than housing prices, seem to indicate the opposite. In fact, δ is not significantly different to –0.5 under Column (4), giving support to the specification of Column (2), which is equivalent to the weighted

Tobit model I presented in the previous section. Standard errors of the estimates are large under the more general restriction (9) in Column (4), compared to equation (8) in Column (2), but housing prices are still statistically significant.

7. Conclusion

This paper investigates the association between housing values and the spread of a communicable disease, SARS. Understanding the incidence of the cases by housing values is important for devising epidemic control strategies, and if the link proves to be direct and causal, the relationship also has implications for general public health policies. Given that most believe that SARS will return, and it is unlikely to be the last of the emerging diseases with global health implications, it is worth considering what lessons we can learn from the 2003 SARS epidemic.[30]

Using data of pre-SARS housing values and the cross-sectional variation in the SARS infection rate in Hong Kong, I find a significant and negative association between the two variables. I explore the robustness of the relationship by considering housing values at different points of time, and by controlling for various measured housing characteristics. The nature of the epidemic helps me separate various factors that might have contributed to this association. For example, because SARS is a new and unanticipated disease, it cannot have directly led to sorting among the population into housing estates according to their susceptibility to SARS. The prodigious level of public health effort to combat SARS also makes differences in access to suitable health care an unlikely explanation for this association. Finally, Lau et al. (2003) suggests widespread adoption of precautionary practices in the Hong Kong population, consistent with anecdotal evidence in the media, so the differential in spread of SARS due to health

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

habits is likely to be small. Therefore I argue that my results point to a direct, negative impact of housing values on the risk of SARS.

In addition, my results point to a relationship that is robust after controlling for measures of living conditions. I provide evidence that household income makes an important contribution to the relationship, in addition to environmental factors such as density and availability of facilities. As lower income leads to a higher susceptibility to SARS, there is a stronger case for improving economic conditions. To justify policies of wealth redistribution, however, one needs to look harder at the form of the housing values-SARS relationship. Because the link appears to be linear, a redistribution of wealth would not reduce the average SARS infection rate and my results do not provide strong support for this type of policies.

None of the government measures used to combat SARS during and in the aftermath of the 2003 epidemic was devised with the link between SARS and economic conditions in mind.[31] 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.

An unexpected finding that emerges from my analysis is that the SARS infection rate was higher in housing estates with facilities such as health clubs, shopping arcades and child care centers, after controlling for factors such as estate property value and population. It is unclear whether this result is related to a higher population contact rate in those estates, or some other unobserved lifestyle or estate characteristics. This finding is worth exploring further, by employing more detailed data on both the type and quality of the estate facilities.

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

After the SARS epidemic, the Hong Kong government encouraged the formation of residents' associations, asserting that older buildings without a management structure of some form are more likely to be neglected and less well maintained.[32] However, I do not find any support for this policy based on the relationship between either building age or the formation of residents' associations and the spread of SARS.

While much is still unknown about the disease, this paper contributes to our understanding of the spread of SARS by analyzing the differential infection rate in different parts of Hong Kong. It also provides new evidence on the SES-health link, pointing to an important role of household income, controlling for measures of living conditions. Future research is required to disentangle the effects of unobserved living conditions, resident characteristics and household income to pin down the channels through which housing values are related to the spread of SARS.

[32] Speech by Mr. Michael Suen, the Secretary for Housing, Planning and Lands. October 25, 2003. (http://www.hplb.gov.hk/eng/press/2003/20031025117.htm)

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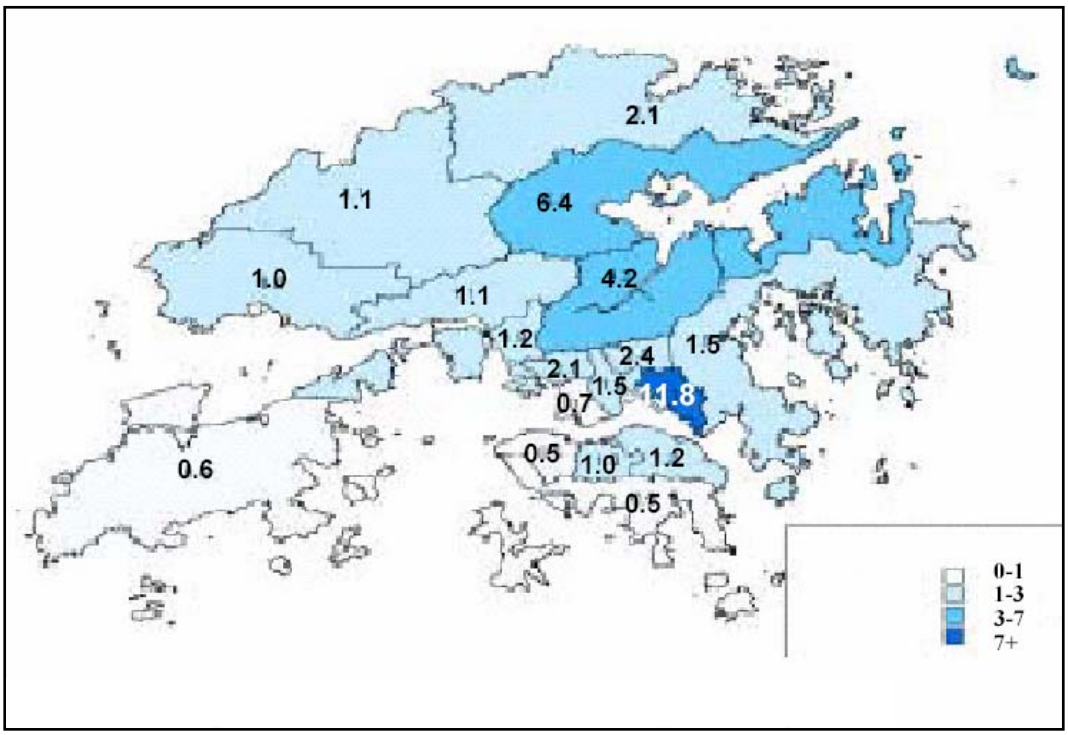
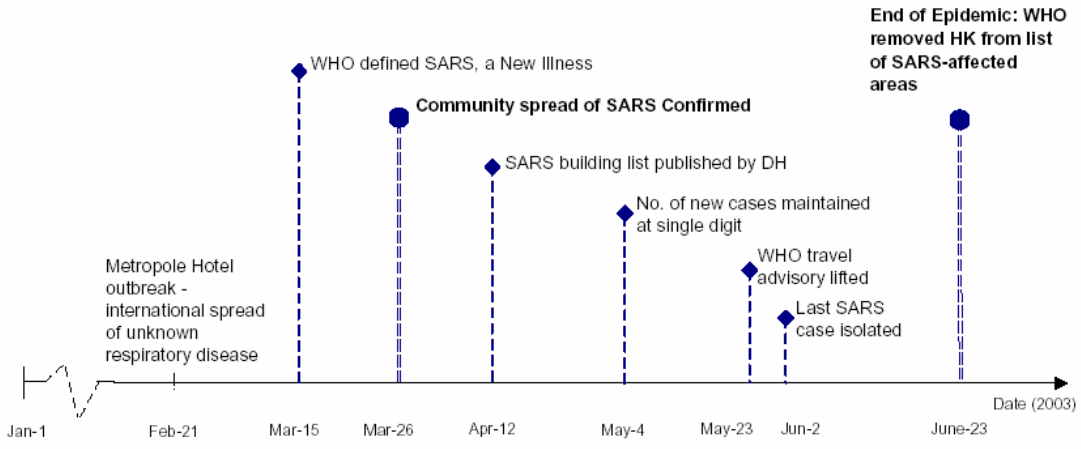
Figure 2.1 SARS Incidence Rates by District



Source: Hong Kong Department of Health. *SARS Bulletin*, 13 June, 2003.

Figure 2.2 2003 Hong Kong SARS Epidemic Timeline



Source: Wong (2004).

Table 2.1A Summary Statistics: District Characteristics

	District Population ('000)	No. of SARS cases per 100,000	% of Tertiary Educated, Aged 20+	Labor Force Participation Rate (%)	Median Monthly Personal Income (USD)	Median Monthly Household Income (USD)	Home ownership rate (%)	Median Household Rent (USD)
1	262	4.69	31.0	66.7	1677	3271	60.0	800
2	167	9.45	34.1	65.9	1677	3355	56.6	968
3	616	12.25	21.7	62.7	1548	3059	61.5	258
4	290	4.84	18.8	62.1	1355	2994	41.4	185
5	282	14.31	16.1	61.2	1290	1897	56.3	387
6	354	16.88	13.3	56.8	1290	1806	38.4	192
7	381	17.49	20.6	60.2	1355	2555	55.4	281
8	445	19.74	9.6	57.0	1290	2077	36.8	194
9	562	96.11	12.1	58.0	1290	2032	38.1	171
10	477	20.60	11.3	60.4	1290	2155	33.0	183
11	276	10.83	17.4	64.2	1419	2710	56.2	219
12	489	10.77	9.9	62.8	1290	2194	53.9	139
13	449	12.90	11.6	61.6	1290	2065	52.1	160
14	299	21.15	10.7	60.2	1290	2220	57.5	168
15	311	64.36	13.6	61.7	1290	2387	61.1	168
16	629	42.99	16.5	62.5	1419	2700	56.3	191
17	328	19.82	16.1	65.2	1419	2710	58.5	207
Weighted Mean		26.45	15.74	61.38	1370.04	2441.18	50.66	246.19
Weighted S.d.		26.01	5.96	2.68	117.93	449.98	10.00	177.97

Table 2.1B Summary Statistics: Estate Characteristics

	(1) **All 295 estates**	(2) **66 listed estates**	(3) **229 non-listed estates**	(4) **Difference (2)-(3)**
Estimated no. of SARS cases	17.20	41.38	--	--
per 100,000 residents	(31.46)	(37.34)		
Median sales price	206.98	189.89	219.40	-30.84
in 2002 ('000 USD)	(171.30)	(104.11)	(206.33)	[20.52]
Proportional price change	-52.47	-51.95	-52.85	0.83
from 1998 to 2002 (%)	(13.23)	(10.31)	(15.03)	[1.59]
Travel time to	0.53	0.56	0.51	0.05*
city centers (hours)	(0.25)	(0.23)	(0.27)	[0.03]
Average flat size	715.11	728.15	705.84	15.81
(Sq. Ft.)	(255.99)	(213.05)	(283.04)	[30.15]
Building age	16.74	17.41	16.26	1.29*
	(6.57)	(7.73)	(5.61)	[0.77]
Estate population ('000)	5.15	9.52	3.85	5.67***
	(6.71)	(10.34)	(4.49)	[0.88]
Availability of facilities	0.68	0.86	0.55	0.32***
(1=yes)	(0.47)	(0.35)	(0.50)	[0.05]
Public housing dummy	0.24	0.18	0.28	-0.11**
(1=yes)	(0.43)	(0.39)	(0.45)	[0.05]
Residents' association (RA)	0.97	0.98	0.96	0.02
dummy (1=yes)	(0.17)	(0.13)	(0.19)	[0.05]
Incorporated RA dummy	0.50	0.41	0.58	-0.16***
(1=yes)	(0.50)	(0.50)	(0.50)	[0.06]
RA Being Responsible for	0.03	0.02	0.04	-0.02
building management (1=yes)	(0.17)	(0.13)	(0.20)	[0.02]
No. of medical establishments	1.01	0.84	1.13	-0.31
nearby	(1.64)	(1.48)	(1.74)	[0.19]
No. of general practitioners	6.95	5.59	7.92	-2.01*
(GP) nearby	(8.76)	(5.86)	(10.24)	[1.03]
No. of other health-related	12.00	10.23	13.26	-2.64*
Facilities nearby	(12.99)	(9.70)	(14.80)	[1.53]

Note: Except for housing estate population, all measures are weighted by total no. of flats in each housing estate. Standard deviations reported in parentheses. Standard errors reported in brackets in the 4th column; *** denotes significance at 1%, ** 5% and * 10%. SARS-list refers to the Department of Health "List of Buildings with Confirmed Cases". Medical Establishments include hospitals, clinics and health care centers. GPs are the community doctors. Other Health-Related Facilities include pharmacies, dental hospitals or Chinese medicine practitioners. The nearby area is defined as a north-facing rectangular area of $0.51km^2$ with the estate in center.

Table 2.1C Summary Statistics: Neighborhood Characteristics

		Mean	S.d.
(1)	Neighborhood Population ('000)	17290	2775
(2)	% of population under 15	16.72	3.22
(3)	% of population over 64	10.54	4.12
(4)	% of Tertiary Educated, Aged 20+ Non-students	19.87	12.76
(5)	Labor Force Participation Rate (%)	63.13	5.96
(6)	Male Labor Force Participation (%)	73.51	6.12
(7)	Female Labor Force Participation (%)	53.65	7.37
(8)	Median Monthly Income from Main Occupation (USD)	1631	525
(9)	Median Monthly Household Income (USD)	3389	2384
(10)	% of Households Owning Quarters Occupied	60.38	24.87
(11)	Median Monthly Household Mortgage/ Loan Payment (USD)	1375	1028
(12)	Mortgage/Loan Payment to Income Ratio (%)	26.96	8.03
(13)	Median Monthly Household Rent (USD)	776	988
(14)	Median Rent to Income Ratio (%)	21.57	8.27
			(N=156)

Note: Rows (2) to (14) are weighted by neighborhood population. Source: Hong Kong Census 2001.

Table 2.2 Pre-SARS Estate Characteristics and SARS Infection Rate

	Dependent Variable: No. of Cases/ 100,000 Estate Residents Tobit Regressions [weighted by no. of flats in each housing estate]						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
District dummies	Yes	No	No	No	No	No	Yes
Median sales price 2002 in USD / 10,000	--	-1.36***	-1.17**	-1.54***	-1.72***	-1.20**	-1.14
		(0.49)	(0.51)	(0.54)	(0.61)	(0.61)	(0.81)
Travel time to city centers (hours)	--	--	26.18	--	--	67.59**	134.50*
			(27.67)			(30.74)	(74.48)
Availability of estate facilities (1 = Yes)	--	--	--	89.99***	--	43.91**	49.65**
				(18.24)		(19.00)	(20.29)
Estate population ('000)	--	--	--	--	10.30***	8.91***	7.97***
					(2.18)	(2.26)	(2.73)
Estate population squared	--	--	--	--	-0.15***	-0.13***	-0.12**
					(0.04)	(0.04)	(0.06)
P-value of F-tests							
District dummies	0.25	--	--	--	--	--	0.37
Population quadratic	--	--	--	--	0.00	0.00	0.00
No. of observations	295	280	280	280	280	280	280

Note: All regressions include a constant term. Standard errors reported in parentheses. *** denotes statistical significance at 1%, ** at 5% and * at 10%. Weighted mean [standard deviation] of the dependent variable is 17.20 [31.46].

Table 2.3 Relationship between SARS Infection Rate and Other Estate Characteristics

	Dependent Variable: No. of Cases/ 100,000 Estate Residents Tobit Regressions [weighted by no. of flats in each housing estate]							
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Median sales price 2002 in USD / 10,000	-1.15* (0.63)	-1.65 (1.10)	-1.18* (0.61)	-1.11* (0.63)	-1.10* (0.63)	-1.11* (0.62)	-1.20** (0.61)	-1.21** (0.62)
Building age	0.34 (1.08)	--	--	--	--	--	--	--
Average flat size per person	--	0.10 (0.21)	--	--	--	--	--	--
No. of medical establishments	--	--	-5.48 (5.07)	--	--	--	--	--
No. of General Practitioners (GP)	--	--	-1.58 (1.63)	--	--	--	--	--
No. of other health-related facilities	--	--	0.45 (1.01)	--	--	--	--	--
PSPS dummy (1 = Yes)	--	--	--	21.21 (32.94)	--	--	--	--
HOS dummy (1 = Yes)	--	--	--	6.38 (20.58)	--	--	--	--
Public housing (1 = Yes)	--	--	--	--	9.89 (18.77)	--	--	--
Incorporated RA (1 = Yes)	--	--	--	--	--	11.68 (15.51)	--	--
RA responsible for for building management	--	--	--	--	--	--	35.14 (43.81)	--
Incorporated RA is for management	--	--	--	--	--	--	--	5.04 (50.14)
P-value of F-tests								
Health facilities	--	--	0.45	--	--	--	--	--
Public housing	--	--	--	0.80	0.60	--	--	--
No. of observations	280	280	280	280	280	279	279	279

Note: RA stands for residents' associations. All regressions also control for a constant term, the travel time to city centres, a facilities dummy and a population quadratic, which are significant at 1 to 10% confidence level. Standard errors reported in parentheses. *** denotes statistical significance at 1%, ** at 5% and * at 10%. Weighted mean [standard deviation] of the dependent variable is 17.20 [31.46].

Table 2.4 Alternative Measurements of Property Values

	Dependent Variable: No. of Cases/ 100,000 Estate Residents Tobit Regressions [weighted by no. of flats in each housing estate]							
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Median sales price 1998 in USD / 10,000	-1.06*** (0.34)	--	--	--	-1.04** (0.42)	--	--	--
Median sales price 1994 in USD / 10,000	--	-1.10*** (0.36)	--	--	--	-1.08** -(1.08)	--	--
Avg. price 1996-02 in USD / 10,000 [2]	--	--	-1.10*** (0.40)	--	--	--	-1.07** (0.51)	--
Neighborhood median rent in USD / 10,000	--	--	--	-1.61* (0.96)	--	--	--	-2.47* (1.28)
Travel time to city centers (hours)	--	--	--	--	59.95** (30.64)	74.76* (38.51)	61.57* (35.44)	74.51** (29.67)
Availability of estate facilities (1 = Yes)	--	--	--	--	47.83** (19.04)	69.97*** (26.15)	80.21*** (25.08)	43.86** (18.62)
Estate population ('000)	--	--	--	--	8.49*** (2.22)	8.88*** (2.59)	8.29*** (2.42)	9.45*** (2.26)
Estate population squared	--	--	--	--	-0.13*** (0.04)	-0.13*** (0.05)	-0.12*** (0.04)	-0.14*** (0.04)
P-value of F-tests Population quadratic	--	--	--	--	0.00	0.00	0.00	0.00
No. of observations	292	236	240	294	292	236	240	294

[1]All regressions include a constant term. Standard errors reported in parentheses. *** denotes statistical significance at 1%, ** at 5% and * at 10%. Weighted mean [standard deviation] of the dependent variable is 17.20 [31.46].
[2]Average pre-SARS baseline price is the mean of median sales prices in years 1996, 1997, 1998, 2001 and 2002. Sale prices are not available for years 1999 and 2000.

Table 2.5 Summary of correlations: SARS Infection rate, Housing Prices & Neighborhood Characteristics

		Weighted Tobit Regressions			
		Controlling for price 2002		Not controlling for price 2002	
	Neighborhood Characteristics	Estimated coefficient on neighborhood characteristic	Estimated coefficient on sales price 2002	Estimated coefficient on neighborhood characteristic	*Correlation between neighborhood characteristic & 2002 price*
(1)	Proportion of non-student aged 20+ with tertiary education (%)	-0.25 (0.67)	-1.11* (0.66)	-0.57 (0.61)	0.47
(2)	School attendance rate of aged 6-18 (%)	-1.48 (3.41)	-1.13* (0.63)	-2.12 (3.23)	0.34
(3)	Median monthly household income ('000 USD)	-5.22 (5.31)	-0.88 (0.69)	-7.51* (4.51)	0.66
(4)	Median monthly personal income from main occupation ('000 USD)	-7.61 (14.12)	-1.11* (0.63)	-10.63 (13.34)	0.31
(5)	Median Age	-4.86 (3.87)	-1.10* (0.61)	-6.05 (3.79)	0.19
(6)	Labor Force Participation Rate (%)	1.05 (1.11)	-1.19** (0.61)	1.09 (1.11)	0.07
(7)	Proportion of households owning units they occupy (%)	0.10 (0.27)	-1.15* (0.61)	0.20 (0.26)	-0.05
(8)	Median mortgage/loan repayment to income ratio (%)	0.77 (0.85)	-1.14* (0.62)	0.70 (0.84)	0.18
(9)	Median rent to income ratio (%)	0.81 (0.88)	-1.34** (0.63)	0.32 (0.84)	0.26
(10)	(3) & (7) above	P-value = 0.36	-0.59 (0.73)	P-value = 0.10	--

[1]All regressions control for a population quadratic, travel time to city centre, availability of estate facilities and a constant term. Standard errors reported in parentheses. *** denotes statistical significance at 1%, ** at 5% and * at 10%. Weighted mean [standard deviation] of the dependent variable is 17.20 [31.46].
[2]Population quadratic, travel time and facilities dummy remain robust at 1 to 10% confidence level in all regressions presented in this table.

Table 2.6 Changes in Property Values and the SARS Infection Rate

	Dependent Variable: No. of Cases/ 100,000 Estate Residents					
	Tobit Regressions [weighted by no. of flats in each estate]					
	(1)	(2)	(3)	(4)	(5)	(6)
Median sales price 2002	-1.60***	--	--	-1.53**	--	--
in USD / 10,000	(0.53)			(0.67)		
Median sales price 1998	--	-1.05***	--	--	-1.05**	--
in USD / 10,000		(0.34)			(0.43)	
Average price 1996-02	--	--	-1.18***	--	--	-1.16**
in USD / 10,000 [2]			(0.41)			(0.50)
Change in sales price,	1.42*	0.78	1.64	1.51*	0.93	2.68**
1998-2002 (%)	(0.83)	(0.80)	(1.17)	(0.91)	(0.86)	(1.35)
Travel time to city	--	--	--	61.39**	59.76*	65.09*
centers (hours)				(30.93)	(30.78)	(35.26)
Availabilities of facilities	--	--	--	52.99***	53.06***	89.75***
				(19.97)	(19.94)	(25.50)
Estate population ('000)	--	--	--	8.19***	8.27***	8.05***
				(2.26)	(2.26)	(2.36)
Estate population squared	--	--	--	-0.12***	-0.12***	-0.12***
				(0.04)	(0.04)	(0.04)
P-value of F-tests						
Population quadratic	--	--	--	0.00	0.00	0.00
Price variables	0.00	0.00	0.01	0.05	0.04	0.02
No. of observations	280	280	240	280	280	240

[1]All regressions include a constant term. Standard errors reported in parentheses. *** denotes statistical significance at 1%, ** at 5% and * at 10%. Weighted mean [standard deviation] of the dependent variable is 17.20 [31.46].
[2]All Average pre-SARS baseline price is the mean of median sales prices in years 1996, 1997, 1998, 2001 and 2002. Sale prices are not available for years 1999 and 2000.

Table 2.7 Tobit vs. CLAD

	Dependent Variable: No. of Cases/ 100,000 Estate Residents					
	Tobit		Weighted Tobit		CLAD	
	(1)	(2)	(3)	(4)	(5)	(6)
Median sales price 2002 in USD / 10,000	-1.39*** (0.61)	-0.70 (0.63)	-1.36*** (0.49)	-1.20** (0.61)	-1.12** (0.52)	-0.14 (0.42)
Travel time to city centers (hours)	--	92.49** (39.97)	--	67.59** (30.74)	--	46.63 (32.95)
Availability of estate facilities (1 = Yes)	--	47.56** (22.74)	--	43.91** (19.00)	--	27.22 (17.53)
Estate population ('000)	--	7.58** (3.63)	--	8.91*** (2.26)	--	2.29 (2.73)
Estate population squared	--	-0.11 (0.08)	--	-0.13*** (0.04)	--	-0.05 (0.06)
No. of observations	280	280	280	280	245	193

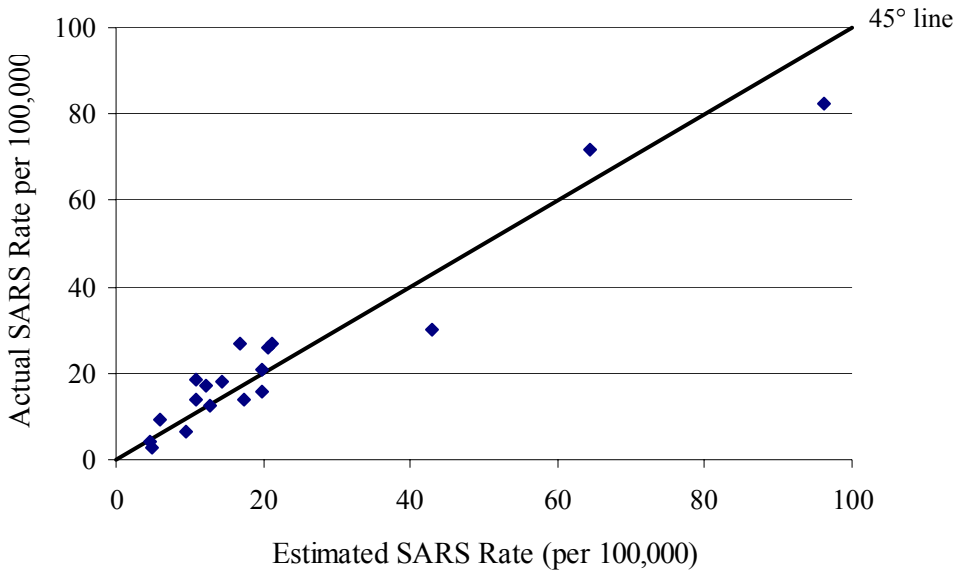
[1]Convergence is achieved in all CLAD estimations, and bootstrapped estimates of the standard errors are reported in Columns (3) and (4). The 80th percentile is used in the quartile regressions in the CLAD procedures, and all bootstraps are with 1000 replications.
[2]Standard errors reported in parentheses. *** denotes statistical significance at 1%, ** at 5% and * at 10%. Weighted mean [standard deviation] of the dependent variable is 17.20 [31.46].

Table 2.8 Heteroskedasticity

(T_i = number of flats in estate *i*)	Maximum Likelihood Regressions with Restrictions on σ_i^2					
	$\sigma_i = \sigma * T_i^{-0.5}$		$\sigma_i = \sigma * T_i^{\delta}$		$\sigma_i = \sigma + \omega T_i$	
	(1)	(2)	(3)	(4)	(5)	(6)
Median sales price 2002 in USD / 10,000	-1.36*** (0.49)	-1.20** (0.61)	-1.48** (0.60)	-1.10* (0.63)	-1.39** (0.60)	-0.86 (0.66)
Travel time to city centers (hours)	--	67.59** (30.74)	--	77.25** (33.33)	--	54.50 (42.52)
Availability of estate facilities (1 = Yes)	--	43.91** (19.00)	--	43.02** (19.56)	--	49.39** (23.09)
Estate population ('000)	--	8.91*** (2.26)	--	9.24*** (2.47)	--	8.81*** (2.80)
Estate population squared	--	-0.13*** (0.04)	--	-0.14*** (0.05)	--	-0.12*** (0.05)
δ	--	--	-0.10 (0.08)	-0.39*** (0.08)	--	--
ω	--	--	--	--	-0.002 (0.002)	-0.01*** (0.001)
P-value of $\chi 2$ test H_0: $\delta = -0.5$	--	--	0.00	0.20	--	--
No. of observations	280	280	280	280	280	280

Note: Standard errors reported in parentheses. *** denotes statistical significance at 1%, ** at 5% and * at 10%. Weighted mean [standard deviation] of the dependent variable is 17.20 [31.46].

Appendix 2.A Estimated and Actual SARS Infection Rates in all 18 Districts



Note: The actual SARS infection rates reported in this graph are the ratio of the number of SARS cases in each district to the Census 2001 district population (also used in deriving the estimated rates). They are therefore slightly different from the infection rates shown in Figure 2.1 using 2003 population estimates.