ARTICLE | Article in Press

The impact of public health events on individual physical health: taking the SARS epidemic as an example

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Received: 27 November 2025 / Accepted: 8 September 2026

Abstract
This paper examines whether a short-lived public health shock affects subsequent physical health. We combine China Health and Nutrition Survey panel data from 1991 to 2009 with province-level SARS surveillance records and exploit cross-provincial variation in exposure to the 2003 SARS outbreak. The empirical design is a difference-in-differences specification with individual and survey-wave fixed effects. The estimates show that individuals in provinces more exposed to SARS experienced a decline in physical health after the outbreak. This result is robust to propensity-score adjusted DID specifications, alternative exposure measures, a city-population-based exposure denominator, alternative outcome coding, adult-sample restrictions, province-clustered inference, and leave-one-province-out checks. Event-study estimates show no clear pre-outbreak differential trend. Heterogeneity analysis suggests that the adverse effect is present for both women and men and is mainly concentrated among individuals with lower educational attainment. Channel evidence indicates that SARS exposure was associated with changes in drinking behavior and other health-related responses. The findings suggest that the health costs of public health emergencies extend beyond infection itself and may persist through behavioral and access-related adjustments.

Introduction

Public health emergencies are not only epidemiological events but also economic and social shocks. They affect health directly through infection risk and indirectly through uncertainty, delayed medical care, disrupted mobility, and changes in social interaction. These indirect effects are important because standard crisis indicators, such as confirmed cases and deaths, may understate the broader welfare cost of an epidemic. The 2003 SARS outbreak provides a useful historical setting for studying this issue. China reported more than 5000 clinically diagnosed SARS cases in mainland provinces in 2003, but the intensity of the outbreak varied sharply across regions. This variation allows us to examine whether exposure to a sudden public health shock left measurable traces in individual physical health.

A growing literature studies the economic and social consequences of public health crises, including effects on growth, labor supply, household behavior, mental health, and public policy responses (Almond and Mazumder 2005; Kelly 2011; Baker et al. 2020). Related public health research emphasizes infection control and immediate medical consequences. However, less is known about whether a short-lived epidemic affects subsequent physical health among the broader population, especially when many affected individuals were not themselves infected. This gap matters for health economics because physical health is both a welfare outcome and a form of human capital. It also

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 https://doi.org/10.1057/s41599-026-09133-4

Published online: 24 September 2026

matters for policy because post-crisis recovery should address not only infection control but also the health losses generated through behavioral adjustment, reduced access to care, and unequal vulnerability.

This paper uses the SARS outbreak as a quasi-natural experiment and combines province-level SARS surveillance data with individual-level data from the China Health and Nutrition Survey (CHNS) from 1991 to 2009. The CHNS provides repeated observations before and after the outbreak, which makes it possible to compare health changes across individuals living in provinces with different SARS exposure intensities. The dependent variable measures whether a respondent reported being sick or injured during the four weeks preceding the survey. SARS case data are drawn from the 2003 epidemic dynamic surveillance records. Because SARS transmission was concentrated in urban areas, the analysis constructs a city-population-based exposure measure using built-up urban area and city population density from the China Statistical Yearbook, while alternative exposure definitions are used for robustness checks.

The empirical strategy is a difference-in-differences design that interacts provincial SARS exposure with a post-2003 indicator. The baseline specification sequentially adds individual controls, household socioeconomic controls, and provincial economic and healthcare-resource controls. We assess the identifying assumption using an event-study specification with the 2000 CHNS wave as the pre-SARS reference period. We also conduct a set of robustness checks following applied economics practice, including propensity-score matched DID, alternative exposure definitions, alternative outcome coding, adult-sample restrictions, province-clustered inference, and leave-one-province-out tests. These checks are designed to show that the estimated effect is not driven by observable pre-SARS differences, denominator choice, outcome coding, or any single province.

The results show that higher SARS exposure is associated with worse subsequent physical health. The estimated effect remains stable as individual, household, and provincial controls are added, and it is robust across the main alternative specifications. The event-study estimates show no clear pre-treatment differential trend before SARS and a deterioration in health after the outbreak. Heterogeneity analysis shows negative effects for both women and men, while the education results are mainly driven by individuals with lower educational attainment. The mechanism analysis is interpreted as channel evidence rather than formal causal mediation: SARS exposure is associated with changes in drinking behavior and other response channels, suggesting that behavioral adjustment, routine disruption, and healthcare-use responses may help explain the observed health decline.

This paper contributes to several related strands of literature in a unified empirical setting. First, it complements research on the long-run consequences of major health shocks, such as studies of the 1918 influenza pandemic and in-utero or early-life exposure (Almond and Mazumder 2005; Kelly 2011), by shifting attention to the broader population-level physical health consequences of a short-lived epidemic shock. Second, it differs from studies that emphasize the macroeconomic and sectoral costs of SARS, including output losses, trade disruption, and cross-border economic effects (Hai et al. 2004; Lee and McKibbin 2004; Keogh-Brown and Smith 2008), by using micro-level panel data to examine individual physical health. Third, it speaks to work on the economic and behavioral consequences of epidemics and health crises (Agüero and Beleche 2017; Baker et al. 2020; Fang and Feng 2021) by linking province-level SARS surveillance records to a long micro panel and by measuring exposure in a way that better reflects the urban concentration of SARS transmission. Fourth, it connects the health economics literature on health production and medical access with sociological accounts of social capital, stress, and lifestyle behavior (Coleman 1990; Cockerham and Scambler 2021) by examining heterogeneous effects and observable response channels. In doing so, the paper provides evidence on how a public health emergency can affect physical health not only through infection risk but also through changes in behavior, routines, and access to care.

The remainder of the paper is organized as follows. Section 2 develops the hypotheses. Section 3 describes the data and empirical strategy. Section 4 presents the results. Section 5 concludes.

Theoretical Analysis and Research Hypotheses

Severe Acute Respiratory Syndrome (SARS) may be regarded as an exogenous public health shock characterized by sudden onset, rapid transmission, and relatively high case-fatality risk. In this study, we assume that such a shock affects individual health through both direct and indirect channels. The direct channel operates through infection risk and disease exposure, while the indirect channel works through behavioral change, psychological stress, mobility restriction, and disruption of access to healthcare and social support. We further assume that individuals are not perfectly insulated from these consequences and that the health effects of a public health emergency may persist beyond the immediate outbreak period. Under these assumptions, epidemics differ from ordinary health fluctuations because they are large-scale, externally imposed, and capable of altering both biological and social conditions in a relatively short time frame (Chen and Zhou 2007; Baker et al. 2020; Coibion et al. 2020).

More specifically, the mechanism can be understood as a sequential process. First, a higher local SARS exposure intensity raises perceived infection risk and uncertainty. Second, this risk shock changes the constraints and incentives faced by individuals: social contact is reduced, medical visits may be delayed or reallocated, routine physical activity and work intensity may change, and some individuals may adopt coping behaviors such as drinking or smoking. Third, these behavioral and access-related adjustments may affect short-run physical health, either by increasing physiological stress and unhealthy habits or by reducing timely prevention and treatment. The empirical mechanism analysis below is therefore designed to examine whether SARS exposure is associated with observable changes in these intermediate channels, rather than to claim that any single channel fully explains the total health effect.

A second assumption underlying our framework is that the consequences of public health crises are not limited to individuals directly infected by the pathogen. Even those who are not diagnosed with the disease may experience substantial health deterioration due to fear, quarantine, disruption of routine care, reduced physical activity, social isolation, and increased psychological distress. Prior studies have shown that epidemic-related stress can generate trauma, anxiety, and depressive symptoms, all of which may undermine physical health over time (Cui et al. 2020). In addition, the adverse effects of public health emergencies may continue after the crisis has subsided. For example, healthcare workers exposed to SARS exhibited elevated burnout and post-traumatic stress reactions long after the outbreak ended (Maunder et al. 2006), while individuals subjected to quarantine experienced persistent emotional distress and reduced well-being even months later (Jeong et al. 2016). These findings suggest that public health events should be understood as shocks with both immediate and lagged health consequences.

A third assumption is that the health consequences of such shocks are heterogeneous across populations. In particular, individuals with fewer material, social, and informational resources are assumed to be less capable of buffering the negative effects of the crisis. This implies that the same public health event may generate more severe consequences for socially vulnerable groups, including women and individuals with lower educational attainment. Empirical evidence from the SARS outbreak supports this view, showing that the epidemic was associated with increased mortality and greater psychological strain, especially among older adults, women, and less educated individuals (Fang and Feng 2021). More broadly, early-life exposure to environmental and health shocks has been linked to lasting harms in physical development and well-being, suggesting that public health crises can produce durable effects across the life course (Hertzman and Power 2020).

Taken together, the theoretical framework rests on three key assumptions: first, that public health events constitute exogenous shocks with direct and indirect health effects; second, that these effects extend beyond the infected population and persist over time; and third, that the magnitude of harm varies systematically across social groups according to their vulnerability and adaptive capacity. On the basis of these assumptions, we posit that major public health events are likely to generate adverse effects on individual health outcomes. Accordingly, we propose the following hypothesis:

Hypothesis 1: Major public health events are likely to generate adverse effects on individual health outcomes.

The outbreak of SARS generated widespread public anxiety and uncertainty, which in turn altered individuals' social environments and behavioral responses (Coleman 1990). Although SARS affected a broad set of regions, including Guangdong, Beijing, Shanxi, and Hong Kong, its social consequences were not uniform. Regions that experienced higher infection intensity or more visible exposure to the epidemic were more likely to face heightened public attention, risk labeling, and temporary social distancing by outsiders. In this context, the key mechanism is not necessarily large-scale overt stigmatization alone, but rather the broader disruption of social identity, perceived social recognition, and interpersonal trust. According to social identity theory (Tajfel 1978), when group membership becomes associated with risk or vulnerability, individuals may experience a weakening of belongingness and selfworth, which can induce stress-related behavioral responses. Even where direct stigmatization is limited, the perception of being socially disadvantaged or less trusted may still alter individuals' psychological state and behavioral choices.

In addition to identity-related stress, the SARS epidemic substantially weakened social capital by restricting face-to-face interaction and disrupting routine social exchange. Quarantine measures, mobility restrictions, and infection fears reduced the frequency of community contact, particularly in high-incidence areas, thereby limiting access to both instrumental support and emotional reassurance. From the perspective of health sociology, lifestyle behaviors are shaped not only by personal preferences but also by available social and economic opportunities (Cockerham and Scambler 2021). When social networks are weakened, individuals may face greater uncertainty and fewer resources for coping with crisis-induced stress. Under such conditions, some residents may resort to maladaptive strategies, including increased smoking or alcohol consumption, as a means of emotional regulation or stress relief. This mechanism is especially plausible in settings where unhealthy behaviors are embedded in everyday social interaction and can serve as accessible coping devices during periods of instability.

Moreover, in China's relation-centered social context, social integration often depends on repeated interpersonal exchange, and practices such as drinking and smoking may function as important social tools for maintaining reciprocity and trust. When public health crises disturb these relational structures, individuals may attempt to restore social embeddedness through participation in collective rituals or networking activities that involve unhealthy behaviors. Thus, the behavioral consequences of SARS are not driven solely by external stigma in the narrow sense, but also by the combined effects of social disruption, reduced social capital, and compensatory coping. For some individuals, especially those whose social and occupational opportunities depend heavily on interpersonal ties, engaging in smoking or drinking may become a strategy to reduce psychological discomfort or to rebuild social acceptance. Consequently, even though SARS was geographically widespread, the social and psychological impact of the epidemic could still differ across individuals and regions, thereby generating meaningful variation in health-related behaviors.

Taken together, these considerations suggest that public health crises may encourage unhealthy behaviors through multiple channels. On the one hand, crisis-induced stress, weakened social identity, and reduced social capital may increase the likelihood of maladaptive coping. On the other hand, attempts to rebuild social connection and regain interpersonal trust may reinforce participation in unhealthy social practices. Therefore, we propose the following hypothesis:

Hypothesis 2: Public health events may encourage the adoption of unhealthy behaviors, thereby adversely affecting individual health outcomes.

Public health events, including pandemics, environmental pollution crises, and health threats arising from natural disasters, operate as exogenous shocks whose health consequences are rarely distributed evenly across the population. Prior research in economics and sociology consistently shows that the burden of such shocks is shaped by pre-existing social stratification, with women and individuals with lower educational attainment often experiencing greater health vulnerability (Marmot 2005; Cutler et al. 2006; Case and Deaton 2015). From a household production perspective, individuals allocate limited time and resources among market work, domestic production, and leisure, and these allocation decisions are central to health production (Becker 1965; Grossman 1972). Women typically bear a disproportionate share of unpaid household labor and caregiving responsibilities, including caring for sick family members and maintaining sanitary conditions (Bianchi et al. 2000; Folbre 2009). During a public health crisis, the demand for such domestic health-related activities increases sharply, crowding out the time and energy women can devote to their own health investments, such as rest, exercise, and timely medical care. This time constraint mechanism implies that women may experience greater deterioration in health outcomes when confronted with public health shocks.

In addition to time constraints, intra-household resource allocation may further exacerbate women's health disadvantage. Existing studies show that household bargaining power often affects how income, nutrition, and access to medical services are distributed among family members (Thomas 1990; Duflo 2012). In crisis periods, when public health events trigger income loss, unemployment, or shortages of essential resources such as medicines and clean water, women may be less able to secure adequate health-supporting inputs if household decision-making privileges male members as primary earners. Gender norms may also intensify these inequalities. In many settings, women are expected to conform to caregiving roles and family-centered identities, which can limit autonomy in seeking external medical services and increase exposure to infection within the household (Ridgeway 2011). As a result, women may be more likely to sacrifice their own health needs in favor of household demands, thereby amplifying the adverse health consequences of public health shocks.

Educational attainment is likewise a critical determinant of health and becomes even more consequential during public health emergencies. Human capital theory suggests that education enhances individuals' ability to convert health inputs into better health outcomes by improving information processing, decision-making, and resource utilization (Grossman 1972; Cutler and Lleras-Muney 2010). More educated individuals are generally better able to understand health information, evaluate risks, and adopt effective preventive behaviors. By contrast, those with lower educational attainment may have more difficulty interpreting complex guidance, such as quarantine rules, vaccination instructions, or treatment protocols, which can delay appropriate responses and worsen health outcomes (Berkman et al. 2011; Nutbeam 2000). During public health crises, when information is abundant but often inconsistent or difficult to interpret, limited education can increase the likelihood of misinformation exposure, low compliance with preventive measures, and inefficient use of scarce health resources.

Moreover, education expands an individual's capability set and access to adaptive resources, making it easier to buffer shocks and recover from adverse events. Individuals with lower educational attainment often face multiple disadvantages simultaneously, including lower income, less secure employment, poorer housing conditions, and weaker social support networks (Marmot et al. 2012). When public health events disrupt livelihoods and essential services, these compounding vulnerabilities can limit their ability to access alternative healthcare, relocate to safer environments, or obtain timely treatment. Consequently, the health losses associated with public health shocks are likely to be more severe and more persistent among individuals with lower education. Taken together, these mechanisms suggest that the adverse effects of public health events on physical health are likely to be amplified for women and for individuals with lower educational attainment. Therefore, we propose the following hypothesis:

Hypothesis 3: The adverse impact of public health events on individual physical health is disproportionately exacerbated among women and individuals with lower educational attainment.

Methodology

Data sources

The SARS exposure data are obtained from the 2003 SARS epidemic dynamic surveillance dataset, which records the cumulative number of clinically diagnosed SARS cases by province during the 2003 outbreak in mainland China. This dataset provides province-level variation in epidemic intensity and is used to construct the key SARS exposure measure in this study. Specifically, we use the maximum cumulative number of clinically diagnosed SARS cases reported in each province in 2003 to capture interprovincial differences in exposure severity. The data are aggregated at the provincial level and merged with the individual survey data using province identifiers.

The empirical analysis in this study utilizes data from the China Health and Nutrition Survey (CHNS) covering the period 1991–2009. The CHNS is a large-scale longitudinal household survey jointly administered by the Chinese Center for Disease Control and Prevention (formerly the Chinese Academy of Preventive Medicine) and the Carolina Population Center at the University of North Carolina. The survey employed a multi-stage, stratified random cluster sampling design, encompassing urban and rural areas across nine diverse provinces in eastern, central, and western China, rendering the dataset broadly representative of the country's population. To ensure consistency in variable availability across waves, the 1989 wave is excluded due to the absence of individual health indicators. Similarly, post-2009 waves are omitted as the survey did not cover Beijing and certain other municipalities until 2011—areas notably affected by the SARS outbreak. After removing observations with missing values in core variables such as health status, age, and gender, the final analytical sample consists of 66,049 individual-year observations.

Table 1 Number of People Infected with SARS Virus in Different Provinces of China.

Province	Number of infected persons	Province	Number of infected persons	Province	Number of infected persons
Beijing	2523	Anhui	10	Shandong	1
Guangdong	1515	Gansu	8	Jiangxi	1
Shanxi	450	Shanghai	8	Guizhou	0
Inner Mongolia	290	Jiangsu	7	Tibet	0
Hebei	228	Hunan	6	Hainan	0
Tianjin	177	Hubei	7	Yunnan	0
Jilin	35	Ningxia	6	Xinjiang	0
Guangxi	22	Zhejiang	4	Qinghai	0
Sichuan	20	Chongqing	3	Heilongjiang	0
Henan	15	Liaoning	7	Total	5330
Shaanxi	12	Fujian	3		

This table reports the maximum cumulative number of clinically diagnosed SARS cases by province in 2003. Source: 2003 SARS epidemic dynamic surveillance dataset.

The empirical strategy of this study relies on data from the China Health and Nutrition Survey (CHNS). Regarding the choice of the temporal scope, we restrict our analysis to survey waves from 1991 to 2009. This restriction is not an incidental outcome of data availability but is closely tied to the identification strategy of the paper. Specifically, our aim is to estimate the long-term effects of the SARS epidemic on individual physical health. This requires constructing a relatively "clean" observation window that captures both pre-exposure health baselines and sufficiently long post-exposure health trajectories, while avoiding contamination from other major public health events or policy shifts. Following the 2003 SARS outbreak, China did not experience another nationwide public health crisis of comparable scale in the immediate years. However, extending the sample beyond 2010—especially to waves from 2015 onward—would introduce confounding factors such as subsequent emerging infectious diseases (e.g., H1N1 influenza, H7N9 avian influenza) and reforms in the healthcare system, making it difficult to attribute observed health effects solely to the SARS shock.

Moreover, in later CHNS waves (e.g., 2015, 2018), several key health-related variables (e.g., the measurement of self-rated health trajectories and the phrasing of questions on specific chronic conditions) underwent changes in questionnaire design and coding consistency. Directly incorporating these later waves would compromise the cross-wave comparability of core variables and the internal validity of the analysis. Therefore, we retain the 1991–2009 period. This window ensures at least two pre-epidemic waves (1991, 1993, 1997, 2000, etc.) and multiple post-epidemic waves (2004, 2006, 2009), while maintaining stability in variable definitions. Although this temporal restriction limits direct extrapolation to more recent contexts, it is a justified choice given the priority of internal validity and data consistency.

Variable selection

Explained variable

The dependent variable in this study is individual health status. Following the approach established by Lei and Lin (2009), we measure an individual's physical health level based on their self-reported morbidity experience during the four weeks preceding the interview. The CHNS questionnaire assesses this dimension through the following item: "Have you been ill or injured in the past four weeks? Have you suffered from chronic diseases or acute diseases?" To construct a binary health indicator, the variable is coded as 1 if the respondent reports no illness or injury ("None"), indicating good health. Conversely, any affirmative response leads to a value of 0, reflecting the presence of health impairment. This measurement strategy effectively captures short-term variations in health conditions while maintaining comparability with prior research.

Table 2 Definition of variables and descriptive statistical analysis.

variable	Definition	Mean	SD	Min	Max
Health	If you are not sick four weeks before the interview, assign it to 1, otherwise assign it to 0	0.895	0.306	0.000	1.000
SARS	Province-level SARS exposure interacted with Post; measured as ln(1 + cumulative confirmed SARS cases) x Post.	0.649	1.068	0.000	3.135
Female	Female respondent assigned to 1, male respondent assigned to 0	0.503	0.500	0.000	1.000
Age	Individual age	41.465	18.514	4.000	81.000
Education	Individual years of education	6.771	4.614	0.000	9.000
Marry	Married assigned to 1, otherwise assigned to 0	0.702	0.457	0.000	1.000
Hukou	Urban household registration (hukou) is assigned to 1, otherwise it is assigned to 0	0.391	0.488	0.000	1.000
Insurance	Enrolled in medical insurance assigned to 1, otherwise it is assigned to 0	0.373	0.484	0.000	1.000
Income	Annual household income (logarithmic)	9.184	1.293	0.000	9.304
Expense	Annual household consumption (logarithmic)	5.134	3.855	0.000	7.103
Size	Household size	4.024	1.546	1.000	12.000
Gdp	Level of economic development	8.773	0.887	6.941	18.830
Population	Total population of the province where the individual is located	8.660	0.341	8.120	8.700
Bed	Number of beds per 10,000 people	21.633	10.680	1.910	22.000
Doctor	Number of doctors per 10,000 people	13.308	5.531	1.100	14.100
Hospital	Number of hospitals per 10,000 people	0.404	0.109	0.092	2.368

Descriptive statistics are reported for the variables used in the empirical analysis. SARS denotes the interaction between province-level SARS exposure and the post-SARS indicator.

Core explanatory variables

The SARS exposure data are obtained from the 2003 SARS epidemic dynamic surveillance dataset, which reports province-level cumulative clinically diagnosed SARS cases, deaths, suspected cases, and related indicators during the outbreak. For each province, we use the maximum cumulative number of clinically diagnosed SARS cases reported in 2003 and match it to the CHNS province identifiers. Table 1 reports the provincial distribution of SARS cases. Because SARS mainly affected urban areas, standardizing cases by total provincial population may understate urban exposure intensity. The analysis therefore constructs an urban-exposure denominator by multiplying built-up urban area by city population density from the China Statistical Yearbook. Total-population normalization is retained as an alternative specification.

Control variables

Following the original specification, we control for individual, household, and provincial characteristics where applicable, as shown in Table 2. Individual-level controls include age, age squared, gender, years of education, marital status, and hukou type. We also control for medical insurance enrollment, household income, household expenditure, and household size. Provincial controls include log GDP per capita, log total population, hospital beds per 10,000 persons, licensed physicians per 10,000 persons, and medical institutions per 10,000 persons. In specifications with individual fixed effects, time-invariant individual characteristics and time-invariant provincial controls are absorbed by the individual fixed effects; these variables are therefore controlled for through the fixed-effects structure rather than separately reported as coefficients.

Table 3 Estimation Results of the Impact of SARS Epidemic on Individual Health.

Variable	(1)	(2)	(3)	(4)
SARS	−0.014**	−0.016***	−0.019***	−0.014***
	(0.006)	(0.003)	(0.004)	(0.004)
Age		0.200***	0.216***	0.202***
		(0.027)	(0.032)	(0.032)
Age squared		−0.000***	−0.000***	−0.000***
		(0.000)	(0.000)	(0.000)
Education		−0.000	−0.001	−0.001
		(0.001)	(0.001)	(0.001)
Married		0.004	0.008	0.008
		(0.010)	(0.008)	(0.007)
Urban hukou		0.002	0.004	0.005
		(0.007)	(0.007)	(0.008)
Insurance			−0.018**	−0.020**
			(0.008)	(0.009)
Income			−0.002	−0.002
			(0.002)	(0.002)
Expense			−0.000	−0.000
			(0.001)	(0.001)
Size			0.006*	0.006*
			(0.003)	(0.003)
Gdp				−0.007
				(0.080)
Population				0.080
				(0.201)
Bed				0.001
				(0.003)
Doctor				−0.009
				(0.006)
Hospital				0.012
				(0.009)
Individual FE	YES	YES	YES	YES
Year FE	YES	YES	YES	YES
Observations	93331	64420	61740	61740
R2	0.020	0.035	0.036	0.037

Robust standard errors are reported in parentheses. ***$p<0.01$, **$p<0.05$, *$p<0.10$.

Econometric model

Given the substantial variation in SARS exposure intensity across Chinese provinces, this study follows the original continuous difference-in-differences design to estimate the effect of the epidemic on individual physical health. The identifying variation comes from differences in provincial SARS exposure before and after the 2003 outbreak. The baseline specification is written as follows:

$$Health_{ipt} = \beta_0 + \beta_1(Infected_p \times Post_t) + \gamma X_{ipt} + \alpha_i + \lambda_t + \varepsilon_{ipt} \tag{1}$$

In Eq. (1), *Health* denotes the health status of individual i living in province p in survey wave t. *Infected* is the province-level intensity of SARS exposure, and *Post* equals one for CHNS waves after the SARS outbreak and zero for pre-SARS waves. The interaction between Infected and Post is the core explanatory variable. The coefficient β_1

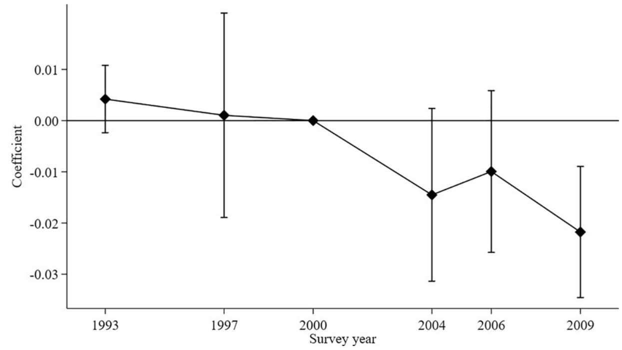

Fig. 1 Parallel Trend Test Plot.

captures whether individuals in provinces with greater SARS exposure experienced larger post-outbreak changes in health. A negative β_1 indicates that higher SARS exposure is associated with a decline in individual physical health. X denotes the vector of individual-, household-, and province-level controls. Individual fixed effects α_i absorb time-invariant individual heterogeneity, including fixed provincial differences, and survey-wave fixed effects λ_t absorb common shocks across waves.

To examine the parallel-trends assumption, we further estimate an event-study specification by replacing the single post-SARS interaction with interactions between provincial SARS exposure and survey-wave indicators:

$$Health_{ipt} = \beta_0 + \sum\nolimits_k \beta_k (Infected_p \times D_{kt}) + \gamma X_{ipt} + \alpha_i + \lambda_t + \varepsilon_{ipt} \tag{2}$$

In Eq. (2), D is a survey-wave indicator, and the 2000 CHNS wave is omitted as the reference period because it is the last pre-SARS survey wave. The coefficients β_k trace the relative evolution of health outcomes across provinces with different SARS exposure intensities. If the pre-2003 coefficients are close to zero, the data support the parallel-trends assumption; the post-2003 coefficients then describe the dynamic health response after the SARS shock.

Empirical Analysis

Baseline regression

Table 3 reports stepwise individual fixed-effects estimates and reports the coefficients on the control variables included in each specification. Column (1) includes the SARS exposure variable, individual fixed effects, and survey-wave effects. Column (2) adds time-varying individual characteristics, Column (3) further adds household socio-economic variables, and Column (4) adds province-year economic and healthcare-resource controls constructed from the China Statistical Yearbook. Across the four specifications, the estimated SARS coefficient remains negative and statistically significant. The preferred specification in Column (4) yields a coefficient of −0.014, indicating that individuals in provinces with greater SARS exposure experienced a larger decline in health after the outbreak. Insurance is negatively associated with health status, likely reflecting adverse selection into coverage among individuals with greater expected medical needs, while income and expenditure have limited additional explanatory power after household and provincial conditions are jointly controlled.

Event-study evidence

We next examine the parallel-trends assumption using Eq. (2). In this specification, the 2000 CHNS wave is taken as the omitted base period, and provincial SARS exposure is interacted separately with each survey-wave indicator. This event-study design makes it possible to compare the pre-outbreak evolution of health outcomes across provinces with

Table 4 Propensity Score Matched DID Robustness Tests.

Variable	(1)	(2)	(3)
SARS	−0.041*	−0.041*	−0.080***
	(0.022)	(0.022)	(0.028)
Controls	YES	YES	YES
Individual FE	YES	YES	YES
Year FE	YES	YES	YES
Observations	37,296	37,296	37,296
R2	0.040	0.040	0.039

The propensity score is estimated using the 2000 pre-SARS wave. Columns (1)-(3) report the common-support sample, propensity-score stratification, and inverse-probability-weighted DID estimates, respectively. All specifications include individual fixed effects and survey-wave fixed effects. Robust standard errors are reported in parentheses. ***$p < 0.01$, **$p < 0.05$, *$p < 0.10$.

different exposure intensities before interpreting the post-outbreak coefficients as the dynamic effect of SARS exposure.

Figure 1 reports the estimated event-study coefficients. The pre-SARS coefficients lie close to zero and do not display a systematic upward or downward pattern before 2003, which is consistent with the parallel-trends condition required by the DID design. After the outbreak, the coefficients turn negative, especially in the later post-SARS waves, indicating that the deterioration in health occurred after the public health shock rather than reflecting pre-existing differential trends.

Robustness checks

Propensity-score-matched DID

As a first robustness exercise, we employ a propensity-score matched difference-in-differences (DID) design, where the propensity score is derived from the 2000 pre-SARS wave conditional on a vector of predetermined individual and household characteristics. We then compare the baseline DID estimate against three commonly adopted propensity-score adjustments—namely, estimation on the common-support sample, propensity-score stratification, and inverse-probability weighting. As shown in Table 4, the estimated SARS effect across all these specifications remains consistently negative. This evidence suggests that our baseline finding is unlikely to be confounded by observable differences in pre-SARS characteristics across individuals residing in high- versus low-exposure provinces.

Concurrent policy environment

A second robustness check considers contemporaneous institutional changes that may confound the estimated SARS effect. A prominent example is the nationwide housing system reform, which could affect household wealth and individual health through changes in housing assets and living conditions. In the individual fixed-effects specification, time-invariant personal and provincial differences related to such institutional background are absorbed by α_i. We therefore examine whether the SARS coefficient remains stable when alternative exposure definitions are used under the individual fixed-effects framework. As shown in Table 5, the estimated effect remains negative across these checks, suggesting that the main conclusion is not driven by fixed provincial or individual differences associated with concurrent policy environments.

Table 5 Estimation Results of Robustness Test.

Variable	(1)	(2)	(3)	(4)	(5)
SARS	−0.014***	−0.002***	−0.060***	0.014***	−0.013***
	(0.004)	(0.000)	(0.011)	(0.004)	(0.004)
Controls	YES	YES	YES	YES	YES
Individual FE	YES	YES	YES	YES	YES
Year FE	YES	YES	YES	YES	YES
Observations	61740	61740	61740	61740	54,652
R2	0.037	0.036	0.036	0.037	0.038

Robust standard errors are reported in parentheses. ***$p < 0.01$, **$p < 0.05$, *$p < 0.10$.

Alternative SARS exposure measures

Table 5 reports robustness checks tied to the discussion above. In the baseline regression, the SARS exposure variable is operationalized as an interaction term between a post-SARS period dummy variable and the natural logarithm of provincial cumulative confirmed cases plus one. For robustness checks, this study further modifies the measurement of the SARS exposure variable: Column (2) directly uses the raw provincial cumulative confirmed case counts; Column (3) adopts a city population-standardized exposure indicator, where the urban population proxy is constructed by multiplying built-up area and population density, with data sourced from the *China Statistical Yearbook*.

Alternative outcome definition

Column (4) of Table 5 replaces the dependent variable with the reverse outcome, namely whether the respondent reported sickness or injury during the previous four weeks. This check is useful because the baseline dependent variable is coded as good health, so a negative SARS coefficient in the main regression should become positive when the outcome is recoded as illness or injury. The result follows this expected direction. Therefore, the baseline finding is not an artifact of how the health indicator is coded, but reflects a deterioration in respondents' physical health after higher SARS exposure.

Adult-sample restriction

Column (5) of Table 5 restricts the sample to adult respondents. This restriction addresses the concern that children and adults may differ in both health reporting and exposure responses, and it also makes the sample more comparable with the behavioral variables used later in the mechanism analysis. The estimated SARS effect remains negative in the adult sample. This suggests that the main conclusion is not driven by age composition, by proxy reporting for children, or by systematic differences in how younger and older respondents report recent illness and injury.

Province-level clustered inference

Column (1) of Table 6 re-estimates the baseline model using province-clustered inference. Because SARS exposure varies at the provincial level, inference may be sensitive to the level at which residual correlation is allowed. Clustering at the province level applies a more conservative inference rule that is aligned with the level of treatment variation. The SARS coefficient remains negative under this specification, indicating that the baseline conclusion is not merely a consequence of using a less conservative standard-error structure.

Table 6 Additional Robustness Checks.

Variable	(1)	(2)
SARS	−0.013***	−0.016***
	(0.004)	(0.005)
Controls	YES	YES
Individual FE	YES	YES
Year FE	YES	YES
Observations	54652	48033
R2	0.038	0.035

Column (1) clusters standard errors at the province level. Columns (2) and (3) report the most negative and least negative SARS coefficients from the leave-one-province-out exercise. Standard errors are reported in parentheses. ***$p < 0.01$, **$p < 0.05$, *$p < 0.10$.

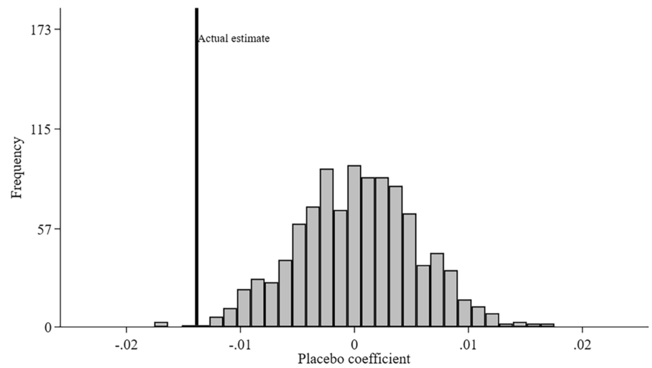

Fig. 2 Placebo Test Plot.

Leave-one-province-out estimates

Columns (2) of Table 6 summarizes the leave-one-province-out exercise by reporting the most negative SARS coefficients across all re-estimations. This exercise repeatedly drops one CHNS province and re-estimates the individual fixed-effects specification, thereby checking whether the result is driven by any single high-exposure province. The estimates remain negative throughout these exercises. This result indicates that the baseline estimate is not driven by any single CHNS province but reflects the broader cross-provincial exposure pattern.

Placebo test

To further examine whether the estimated effect is driven by random assignment of treatment status, we conduct a placebo test by randomly assigning pseudo-treated status to respondents while preserving the treated share in the sample. For each of 1000 repetitions, we construct Pseudo Treat × Post, estimate the same DID specification with the baseline controls and fixed effects, and store the coefficient on the pseudo-treatment interaction. Figure 2 plots the resulting distribution. The placebo coefficients are centered close to zero, whereas the actual treatment estimate lies far in the left tail of the distribution. This pattern indicates that the baseline finding is unlikely to be generated by random treatment assignment.

Table 7 Estimation Results of Heterogeneous Impact of SARS on Physical Health.

Group	(1) Female	(2) Male	(3) Highly educated	(4) Low level of education
SARS	−0.015***	−0.013***	−0.010	−0.014***
	(0.006)	(0.003)	(0.011)	(0.005)
Controls	YES	YES	YES	YES
Individual FE	YES	YES	YES	YES
Year FE	YES	YES	YES	YES
Observations	31,291	30,449	13,127	48,613
R2	0.040	0.034	0.024	0.040

Robust standard errors are reported in parentheses. ***$p < 0.01$, **$p < 0.05$, *$p < 0.10$.

Heterogeneity analysis

Heterogeneity by gender

Table 7 reports heterogeneous effects by gender and educational attainment. The SARS exposure coefficient is negative for both women and men, with a somewhat larger absolute magnitude among women. This pattern is consistent with the heavier care burden and greater disruption of daily health investment faced by women during public health emergencies.

Within resource-scarce crisis contexts, the allocation of limited household resources—such as nutrition and medical care—often reflects deeply entrenched gender biases. Consequently, women, particularly girls and older women, may be disproportionately disadvantaged, experiencing the systematic neglect of their fundamental health needs. Concurrently, incidents of gender-based violence (GBV) frequently escalate during crises (e.g., during lockdowns or within disaster shelters), inflicting direct physical harm and increasing the prevalence of post-traumatic stress disorder (PTSD) among women, thereby constituting a severe public health concern. Economically, women are often positioned in relatively vulnerable labor market segments, frequently concentrated in informal, low-wage, temporary, or precarious employment (e.g., in service and retail sectors). These sectors are typically the most severely impacted by the economic shocks triggered by PHEs, facing mass layoffs or sharp income reductions. This erosion of economic capacity directly undermines women's ability to afford basic necessities, healthcare expenses, and essential medications. Compounding this vulnerability, women often experience lower rates of health insurance coverage compared to men, particularly those employed in the informal economy. This lack of coverage heightens the risk that women will forgo necessary medical care due to prohibitive costs during a crisis.

Gender differences in exposure responses may arise because public health shocks alter caregiving burdens, access to routine services, and household allocation of time and resources. To examine this possibility, we stratify the sample by gender and estimate the individual fixed-effects DID specification separately for women and men. Columns (1) and (2) of Table 7 show negative SARS coefficients for both groups. The point estimate is larger in absolute value among women, which is consistent with the theoretical discussion.

Heterogeneity by educational attainment

Relative to their counterparts with lower educational attainment, individuals who have achieved higher levels of education tend to exhibit superior cognitive abilities, enhanced self-regulation, and a stronger stronger capacity to mitigate the adverse effects associated with public health crises such as the SARS epidemic. Consequently, it can be hypothesized that the negative impact of SARS on health outcomes is likely to be less pronounced among more highly educated individuals compared to those with less schooling. To examine potential heterogeneity in the effect of SARS across educational strata, this study categorizes the sample into two subgroups: one comprising individuals with high educational achievement and the other consisting of those with relatively low educational attainment. The

Table 8 Mechanism analysis.

Variable	(1)	(2)
SARS	0.055***	0.067**
	(0.014)	(0.027)
Controls	NO	YES
Individual FE	YES	YES
Year FE	YES	YES
Observations	61479	48635
R2	0.005	0.016

Robust standard errors are reported in parentheses. ***$p < 0.01$, **$p < 0.05$, *$p < 0.10$.

classification is based on whether an individual's education level exceeds the average educational level observed in the overall population—those above this threshold are classified as having a high education level, while those below it are categorized as having a low education level.

As reported in Column (3) of Table 7, the estimated effect for highly educated individuals is negative under the individual fixed-effects specification. In contrast, Column (4) shows that the coefficient for individuals with lower educational attainment is negative and statistically significant. These findings indicate that the detrimental health impact of SARS is mainly driven by individuals with lower levels of education, supporting the view that educational attainment may serve as a protective resource during public health shocks.

Mechanism evidence

The mechanism analysis is designed to provide channel evidence rather than a formal causal mediation estimate. A plausible channel should satisfy three empirical patterns: SARS exposure should affect health; SARS exposure should affect the proposed channel variable; and the channel variable should be systematically associated with health when included in the health equation. Because the channel variables are observational rather than randomly assigned, these tests are interpreted as stepwise evidence on plausible mechanisms under the maintained DID design.

The theoretical rationale is that public health shocks can affect health through both behavioral and access-related channels. During SARS, uncertainty, mobility restrictions, reduced social contact, and disruption of routine services may have changed individuals' incentives to drink, smoke, seek medical care, or adjust daily activity. The CHNS contains information on alcohol consumption and smoking behavior; Table 8 focuses on drinking behavior because it provides the most stable observable behavioral response in the available data.

As shown in Table 8, the coefficient on SARS exposure remains positive and statistically significant after controlling for individual fixed effects, both without and with the full set of controls, indicating that SARS exposure was accompanied by a significant change in drinking behavior. Taken together, these results provide broader evidence that the SARS shock affected health through a bundle of behavioral adjustment, routine disruption, and healthcare-use responses.

The interpretation is therefore deliberately cautious. The evidence supports the view that health behavior, activity adjustment, and healthcare-use responses are plausible channels, but the paper does not claim that any single variable fully mediates the SARS effect. The empirical tests show that the proposed channels move with SARS exposure and are related to health outcomes, while a definitive causal decomposition would require richer experimental or quasi-experimental variation in the channel variables themselves.

Conclusions

Conclusion

Using CHNS data from 1991 to 2009 and province-level SARS surveillance data, this study examines the effect of the 2003 SARS outbreak on individual physical health. The individual fixed-effects results show that SARS exposure significantly reduced physical health, and this conclusion remains robust after propensity-score adjustments, alternative exposure measures, a city-population-proxy denominator, reverse outcome coding, adult-sample restrictions, province-clustered inference, and leave-one-province-out checks. The event-study estimates using 2000 as the pre-SARS base year support the parallel-trends assumption. Heterogeneity analysis shows negative effects for both women and men, while the education results are mainly driven by individuals with lower education. Finally, the mechanism analysis is interpreted as evidence of health-behavior response rather than formal causal mediation, showing that SARS exposure was associated with a significant increase in drinking behavior.

Policy recommendations

First, the evidence that sustained improvements in health behaviors after the SARS crisis contributed to better COVID-19 mitigation outcomes in previously affected regions highlights the importance of long-term behavioral resilience in epidemic preparedness. This implies that public health policy should not treat health education as a short-term emergency response, but rather as a continuous institutional investment. Governments should therefore strengthen routine public health education campaigns, embed health promotion into community-level outreach, and maintain consistent risk communication before, during, and after crises. In particular, policy design should account for the persistence of behavioral effects over time: interventions that successfully improve health awareness and self-protective habits in the aftermath of one health shock may generate externalities that improve resilience to future outbreaks. This finding underscores the value of integrating behavioral change objectives into broader healthcare policy frameworks, rather than relying solely on clinical or infrastructural measures.

Second, our results indicate that information availability plays a significant role in shaping health-related behavior during public health emergencies. This suggests that governments can improve crisis response by expanding low-cost, timely, and credible information delivery channels. Digital tools such as mobile messaging systems, internet platforms, and social media can be used to disseminate clear guidance on infection prevention, vaccination, and health service use. However, such communication strategies should be designed carefully to avoid misinformation and unequal access. Policymakers may therefore need to combine digital dissemination with community-based outreach, especially in populations with lower digital literacy or weaker access to online resources. In this sense, the empirical link between information access and behavioral response supports a policy approach that prioritizes rapid, transparent, and targeted communication as a core component of emergency preparedness.

Finally, from a broader policy perspective, our results suggest that the interaction between long-term behavioral shifts and healthcare policy design is central to epidemic resilience. Health behavior is not merely an individual choice variable; it is also shaped by institutional incentives, information environments, and the accessibility of healthcare services. Policies that reinforce positive behavioral change after a crisis can generate lasting public health benefits, whereas policies that neglect behavioral heterogeneity may allow temporary shocks to produce persistent welfare losses. Accordingly, an effective public health strategy should combine education, communication, financial protection, and targeted outreach, with special attention to the groups and mechanisms identified in our empirical findings. Such an integrated approach would enhance both the effectiveness and the equity of healthcare policy in the face of future health emergencies.

Discussions

Both SARS-CoV-2, the virus responsible for COVID-19, and the SARS virus belong to the coronavirus family and share similarities in transmission routes and clinical manifestations. However, the COVID-19 pandemic differs from the 2003 SARS outbreak in at least two important respects. First, COVID-19 rapidly evolved into a truly global shock, generating synchronized economic contractions across major advanced and emerging economies and markedly deteriorating the international trade environment. This feature is consistent with studies documenting substantial disruptions to aggregate output, labor markets, supply chains, and economic uncertainty during COVID-19 (Baker et al. 2020; Coibion et al. 2020). Second, owing to its higher transmissibility and longer duration, COVID-19 prompted more extensive and persistent containment policies, which in turn caused more severe restrictions on population mobility, business operations, and local economic activity.

While the primary empirical focus of this paper is on the SARS epidemic as a specific public health event, we recognise that understanding its long-term health consequences requires placing the epidemic within a broader historical and institutional context. The early 2000s in China were not only marked by the SARS outbreak but also characterised by ongoing transitions in household registration (hukou), labour markets, social welfare, and the healthcare system. These structural transformations shaped the environment in which individuals experienced the epidemic and responded to it over time. Viewed from this longer-term perspective, SARS can be understood as a critical public health shock embedded in China's transitional period—a shock that interacted with existing institutional arrangements and individual resources to produce persistent health inequalities.

One of the key insights from our micro-level analysis is that the long-run health effects of SARS were highly heterogeneous across social groups. We find that factors such as education and gender play significant mediating roles. Specifically, individuals with lower educational attainment experienced more pronounced and lasting declines in physical health following the epidemic, suggesting that education served as a buffer—potentially through better health knowledge, more flexible coping strategies, or greater access to information and post-epidemic care. Similarly, gender differences emerged, with female respondents exhibiting longer-lasting health disadvantages, which may reflect differential exposure to caregiving burdens, psychosocial stress, or health-seeking behaviours during and after the crisis. These findings underscore that the legacy of a public health event is never uniform; it is filtered through the lens of social stratification and individual endowments.

By highlighting these heterogeneous effects, the paper contributes not only to the epidemiology of SARS but also to a broader historical understanding of how public health crises can entrench or exacerbate existing health disparities. The lessons drawn from SARS are therefore not limited to the management of respiratory epidemics; they speak to the importance of considering institutional context and social vulnerability when designing post-crisis recovery policies. Future public health strategies should go beyond acute containment and attend to the differential long-term needs of disadvantaged subgroups—a lesson that has become even more salient in the wake of the COVID-19 pandemic. In this sense, our event-specific analysis offers a framework for interpreting the long-run health implications of public health emergencies more generally, while remaining firmly grounded in the empirical identification made possible by the SARS natural experiment.

This study has two main limitations. First, the measurement of key variables remains imperfect. The health outcome is based on self-reported morbidity during the previous four weeks, which captures short-run physical health but cannot distinguish direct SARS infection from indirect health consequences. SARS exposure is also measured at the province level; although the analysis uses a city-population proxy and reports alternative exposure measures, individual-level exposure intensity and within-province spatial variation cannot be fully observed.

Second, the mechanism results should be interpreted as channel evidence rather than a definitive causal decomposition. The analysis supplements the original behavior regressions with a clearer theoretical pathway and stepwise channel checks, but the available survey variables do not fully isolate formal causal mediation. Future research with richer data on psychological stress, healthcare access, mobility, and individual behavioral adjustment could more directly estimate dynamic mediation effects.

Author contributions L investigates how large-scale health crises impact individual and community resilience. His work integrates clinical perspectives with socioeconomic frameworks to inform evidence-based public health interventions. Z specializes in applied econometrics, data visualization, and health economics. F employs empirical methods to analyze how institutional and socioeconomic factors mediate the impact of external shocks on vulnerable populations. He has contributed to studies on health financing, behavioral economics, and sustainable development policy.

Declarations

Competing interests The authors declare no competing interests.

Ethics approval and consent to participate This article does not contain any studies with human participants performed by any of the authors.

Informed consent This article does not contain any studies with human participants performed by any of the authors.

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