

Article

# The COVID-19 Epidemic Spreading Effects

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**Abstract:** Cities are hotbeds for the outbreak and spread of infectious diseases. In the process of urban development, frequent interpersonal interactions are conducive to the spread of viruses. After the outbreak of COVID-19 in Wuhan, China in 2019, it quickly spread to Europe, North America, and Asia. This paper collects data on the number of COVID-19-infected cases per 100,000 people in Taiwan from 1 January to 4 May 2022 and the researcher uses the spatial regression model to analyze the spatial effect of the COVID-19 epidemic. The results of the study find that the hot zones of COVID-19-infected cases per 100,000 people are distributed in Taipei City, New Taipei City, Keelung City, Yilan County, and Taoyuan City, and the cold zones are distributed in Changhua County, Yunlin County, Chiayi County, Chiayi City, Tainan City, and Kaohsiung City. There are three types of urban development indicators: density, urbanization, and transportation system and means of transport, all of which can significantly affect the spatial spread of COVID-19. There is a negative correlation between the area of the “urban planning” district, the “road area” per person, the current status of the urban planning district population “density”, and the number of infected cases of “COVID19”. There is a negative correlation between “urban planning”, “road area”, “urbanization”, and “density” of neighboring cities and “COVID19” in a certain city.

**Keywords:** COVID-19; spatial effect; spatial regression model; hot zones; urbanization



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## 1. Introduction

As of today, much of the literature on urbanization and globalization focuses on the movement of economies and populations within and between cities [1–3]. In recent years, more and more of the literature has begun to focus on academic research and policy analysis of urban health and disease by urbanization [4–7]. Urban epidemiology theory argues that changes in geography affect urban health and disease, and a recent paper further argues that the future of global health depends on urban health [8,9]. The process of spreading infectious diseases should be closely related to the expansion of urbanization [2]. The flow and destruction of social ecology leads to an increase in infectious diseases in suburban and bordering areas. In this paper, the focus of the analysis is on the effects of urbanization indicators and related variables on infectious and emerging infectious diseases. When an animal first transmits an infectious agent to a new human host, the incidence of infectious disease increases rapidly [10]. In particular, social ecology is accompanied by social and spatial changes, which leads to the emergence of new forms of disease transmission, further contributing to the increase of emerging infectious diseases. In examining the relationship between urbanization and infectious diseases, it is suggested that the relationship can be found in landscape political ecology analysis.

The twentieth century was an important milestone in the control and eradication of infectious diseases in history. After World War II, the public health programs that were needed for the questioning and use of new drugs, vaccines and treatments, and prevention provided the effective tools needed for disease control. By the late 1960s, infectious disease scientists and surgeons in the United States declared victory in the war against infectious diseases [11]. However, after the 1960s, two world trends emerged. First, public resources that were once used were redirected in the war against cancer [12]. Second, the 60 years

of population growth that followed World War II led to the orderless urbanization, and changes in agriculture, land use, and livestock, as well as accelerated globalization, became the driving force behind the re-emergence of infectious disease outbreaks [13]. The first evidence of the resurgence of infectious diseases was in the 1970s, but the spread of this epidemic greatly accelerated in the second two decades of the twentieth century. Past diseases that were once effectively controlled began to re-epidemic, such as dengue fever, Japanese encephalitis, West Nile virus, epidemic polyarthritis, yellow fever, measles, plague, cholera, tuberculosis, Leishmaniasis, malaria, etc. In addition, many newly discovered diseases are beginning to cause epidemics, such as acquired immunodeficiency syndrome (AIDS), hemorrhagic fever (Marburg, Ebola, Lhasa, Hantavirus, Crimean–Congo, Sartremic virus, Dengue and yellow fever), avian influenza, Hendra and Nipah encephalitis, severe acute respiratory syndrome (SARS), Lyme disease, Escherichiasis, and coronavirus disease 2019 (COVID-19) in 2019. In addition to these factors mentioned above, bacterial pathogens resistance to antibiotics, malaria-resistant parasites, mosquito resistance to insecticides, new medical technologies such as organ transplantation, and ecological encroachment of humans and animals have all played a role in this situation, and infectious diseases have once again become a global public health problem [12–14]. An estimated 26 per cent of deaths worldwide in 2002 were attributable to infectious and parasitic diseases [15]; disability-adjusted life years (DALYs) are caused by infectious diseases [16].

The book “Plagues and Peoples” describes the development of major urban centers and the regional and even global trade chains through new trade routes, such as the Silk Road connecting the Middle East and Asia, explaining the historical patterns of plague emergence and the results of many key events in history [17]. Therefore, the relationship between urbanization and infectious diseases is an ancient existential relationship. The current global epidemic of infectious diseases is, to some extent, a continuation of this model. Many of the variables that affect the risk of transmission of infectious diseases in urban areas are known. Urban infectious disease outbreaks are at the greatest risk, not only where population densities are highest, but also where public infrastructure and public services are poor and where access to health care and basic public health plans keep pace. Additionally, since all areas and neighborhoods in the metropolis are connected using modern transportation systems, pathogens can spread easily. Even more ironically, the construction of a modern transport system aimed at supporting modernization and economic development has instead increased the mixing of infected and susceptible populations at an unprecedented rate [18].

Infectious disease pathogens spread diseases to other hosts through the transmission route of the host, and a great amount of the infrastructure, buildings in the city, residents, and management methods of the clusters have a profound impact on the transmission of infectious diseases and the epidemic of the disease [2].

Public transport is another important factor in the spread of infectious disease outbreaks. Researchers conducted analysis of the relationship between London underground network public transport and the spread of infectious diseases [19]. They used actual travel data to infer connections between each station at any time of the day and the number of passengers and compared them to influenza-like illness (ILI) incidences in London boroughs. The results showed a correlation between underground use in London and the number of ILI cases and, in particular, they also demonstrated a higher number of ILI cases in boroughs in the USA that spent more time underground and/or incited more travel time in contact areas. On the other hand, in areas with a small number of ILI cases, the use of subways is also relatively limited. The use of public transport and other environmental and demographic factors, such as population, density, employment, and income, can influence the spatial spread of infectious diseases [20–34]. These results are beneficial for both scientists and policy makers. Other indicators influencing the spread of infectious diseases include excessive population exposure due to overcrowding [35–42] and interpersonal links for social networks [43–48].

Emerging infectious diseases (EID) in groups that are considered pathogens in their parasitic groups has increased over the past two decades or is likely to increase in the near future [12]. In addition to describing the spread of newly evolved or previously undiscovered pathogens (pathogens that are expanding their geographical distribution to increase their impact, alter their clinical presentation, or migrate to human hosts for the first time), the term “Emerging” can also be used to describe the re-emergence of known infections after a decline in incidence [12]. It is estimated that 60 to 80 percent of emerging disease infections are zoonotic, so these pathogens depend on the animal pool for survival [49,50]. At least 70% of these emerging zoonotic diseases come from wild flora and fauna infections, cross-species transmission and forward transmission, representing a natural response to the ecological evolutionary pressures of pathogens [49,51]. While both wildlife and domestic animal banks are considered important sources of EIDs, anthropogenic impacts on ecosystems determine the level of risk of human–animal transmission in zoonotic diseases [52].

In the process of urban development, interpersonal social activities are frequent and close interaction is conducive to the spread of the virus. After the outbreak of COVID-19 in Wuhan, China in 2019, it spread rapidly to Europe, North America, and Asia. As the virus spread from one city to another, the number of COVID-19-infected people increased rapidly, reaching 5,016,172,529 infected worldwide by 6 May 2022. (Please refer to the reported data of the Coronavirus Resource Center from Johns Hopkins University and Medicine. Please visit the website of <https://coronavirus.jhu.edu/map.html>). The purposes of the study are as follows:

- (1) Geographical distribution and thermal zone analysis of the number of confirmed COVID-19 cases in Taiwan.
- (2) Spatial regression model estimation of COVID-19 epidemic spread in Taiwan.
- (3) The direct and indirect effects of COVID-19 epidemic spread in Taiwan.

The limitations of this study are as follows:

- (1) The data on the number of confirmed cases of COVID-19 starts on 1 January 2022 and the data is updated daily. Therefore, the distribution period is not a full year.
- (2) The data source is the data published by the government on the website and some data have missing values.

## 2. Materials and Methods

This research used the reported data on the number of confirmed cases of COVID-19 in Taiwan released by the Ministry of Health and Welfare and collected county and city indicators to divide urban development indicators into three categories. The study contains information on density, urbanization, and transportation system and transport implements, and the researcher constructed a spatial regression model for COVID-19 dispersion in Taiwan.

Spatial regression analysis is applied in multiple fields [53–57]. Subsequently, spatial models have been applied in many fields, such as crime, population, economics, epidemiology, politics, and public health [58–68].

In this work, the spatial regression model was used to analyze the influence on the spread of COVID-19 from urban characteristics and the spatial effects of the epidemic in Taiwan. The information comes from the cumulative number of COVID-19 confirmed cases from 1 January 2022 to 4 May 2022 published on the official website of the Ministry of Health and Welfare. (The COVID-19 confirmed data during this period is selected because the official release date started on 1 January 2022 and the information is updated daily. The epidemic during this period already contained preliminary virus variants).

The spatial effect of urban development on the spread of COVID-19 is as follows:

$$COVID19 = \delta WCOVID19 + \alpha i_N + X\beta + WX\theta + u \quad (1)$$

where *COVID19* is the number of confirmed cases of COVID-19 in Taiwan in 2022; *W* is the spatial weight of adjacent cities; *N* is the number of observation, that is, the number of counties and cities; and *i<sub>N</sub>* is the *N* × 1 unit vector. *X* is the variable vector related to urban development; *δ*, *α*, *β*, *θ* are the parameters; and *u* ~ *N*(**0**, *σ*<sup>2</sup>, *I*). Rearranging the variables, Formula (1) can be changed into Formula (2):

$$(\mathbf{i}_N - \delta \mathbf{W}) \mathbf{COVID19} = \alpha \mathbf{i}_N + \mathbf{X}\beta + \mathbf{W}\mathbf{X}\theta + \mathbf{u} \tag{2}$$

The partial derivatives of *COVID19* with respect to the explanatory variables *x<sub>k</sub>* can be expressed in Equation (3) as follows:

$$\begin{aligned} \frac{\partial \mathbf{COVID19}}{\partial \mathbf{x}_k} &= \begin{pmatrix} \frac{\partial \mathbf{COVID19}}{\partial x_{1k}} & \frac{\partial \mathbf{COVID19}}{\partial x_{2k}} & \dots & \frac{\partial \mathbf{COVID19}}{\partial x_{Nk}} \end{pmatrix} \\ &= \begin{pmatrix} \frac{\partial \mathbf{COVID19}_1}{\partial x_{1k}} & \dots & \frac{\partial \mathbf{COVID19}_1}{\partial x_{Nk}} \\ \vdots & \ddots & \vdots \\ \frac{\partial \mathbf{COVID19}_N}{\partial x_{1k}} & \dots & \frac{\partial \mathbf{COVID19}_1}{\partial x_{Nk}} \end{pmatrix} \\ &= (\mathbf{i}_N - \delta \mathbf{W})^{-1} \begin{pmatrix} \beta_k & \dots & w_{1N}\theta_k \\ \vdots & \ddots & \vdots \\ w_{N1}\theta_k & \dots & \beta_k \end{pmatrix} \end{aligned} \tag{3}$$

where *COVID19<sub>N</sub>* is the number of COVID-19 confirmed cases of city *N* and *x<sub>Nk</sub>* is the explanatory variable *k* of city *N*.  $\frac{\partial \mathbf{COVID19}}{\partial \mathbf{x}_k}$  is a *N* × *N* marginal effect matrix. The direct effect is the average of the diagonal elements of the matrix  $\frac{\partial \mathbf{COVID19}}{\partial \mathbf{x}_k}$ , while the indirect effect is the average of the non-diagonal elements of the matrix  $\frac{\partial \mathbf{COVID19}}{\partial \mathbf{x}_k}$ .

### 3. Descriptive Statistics

In this paper, data of the number of confirmed COVID-19 cases in 2022 in each county and city in Taiwan are collected as dependent variables for the spatial regression model (Table 1 and Appendix A). Table 1 lists relative urban development variables with descriptions and provides the units of calculation, variable scales, and numerical types. Urban development variables can be divided into three types including density, urbanization, and transportation systems and transport tools. Table 2 shows the descriptive statistics of the relevant variables.

**Table 1.** Variables used in the spatial regression model.

Variable	Variable Type	Description	Unit	Variable Scale/ Numeric Type
COVID19	–	Number of COVID-19-infected cases per 100,000 people	Number of COVID-19-infected cases/100,000 people	Ratio/Continuous
urban planning	urbanization	Area of the urban planning area	Square kilometers	Ratio/Continuous
forest	urbanization	Forest area	Hectare	Ratio/Continuous
car	transportation systems and transport tools	Number of the car registered	Vehicle	Ratio/Discrete
road	density	Road density	km/km <sup>2</sup>	Ratio/Continuous
road area	transportation systems and transport tools	Road area each person assigned	Square meters/person	Ratio/Continuous

**Table 1.** *Cont.*

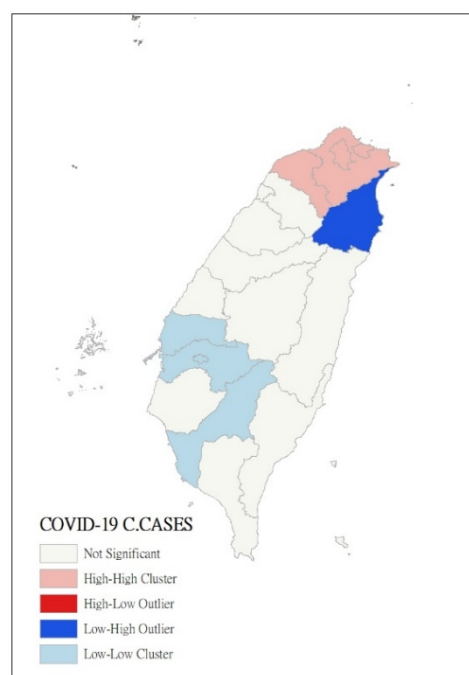
Variable	Variable Type	Description	Unit	Variable Scale/ Numeric Type
density	density	Population density of the current situation in the urban planning area	People/square kilometers	Ratio/Continuous
urbanization	urbanization	Population of the urban area/total population of the municipality	%	Ratio/Continuous

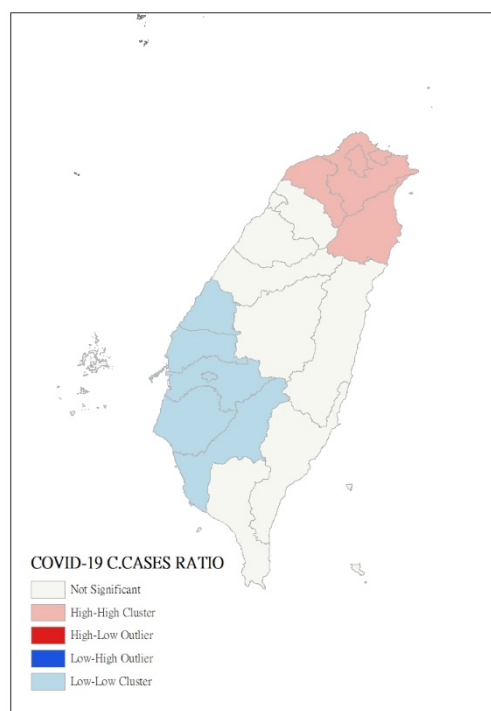
**Table 2.** Descriptive statistics of the variables in the spatial regression model.

Variable	Average	Standard Deviation	Minimum	Maximum
COVID19	514.3806	586.4232	92.77316	2251.217
urban planning	230.4198	282.3826	10.7864	1228.457
forest	109,462.3	110,347.8	773.42	372,780.6
car	343,929.4	307,666.3	27,623	953,063
road	2.305	2.11833	0.35	8.7
road area	31.3065	15.17759	6.91	60.82
urbanization	25.7941	31.14242	2.50452	101.216
density	4197.65	2152.103	1262	9818

#### 4. Discussion

Using the spatial autocorrelation index, the interpretation of the geographical location of COVID-19 in the urban area can be analyzed (Figures 1 and 2). Figure 1 shows that hotspot zones of the number of COVID-19 confirmed cases in 2022 are distributed in Taipei City, New Taipei City, Keelung City, and Taoyuan City in the northern region. The cold zones are distributed in Yunlin County, Chiayi County, Chiayi City, and Kaohsiung City in the southern region, and the threatened zone is distributed in Yilan County. Figure 2 shows that the hotspot zones of the number of COVID-19 confirmed cases per 100,000 people in 2022 are distributed in Taipei City, New Taipei City, Keelung City, Taoyuan City, and Yilan County in the northern region. The cold zones are concentrated in Changhua County, Yunlin County, Chiayi County, Chiayi City, Tainan City, and Kaohsiung City.

**Figure 1.** Hotspot zones of COVID-19 confirmed cases in Taiwan, 2022.



**Figure 2.** Hotspot zones of COVID-19 confirmed cases per 100,000 people in Taiwan, 2022.

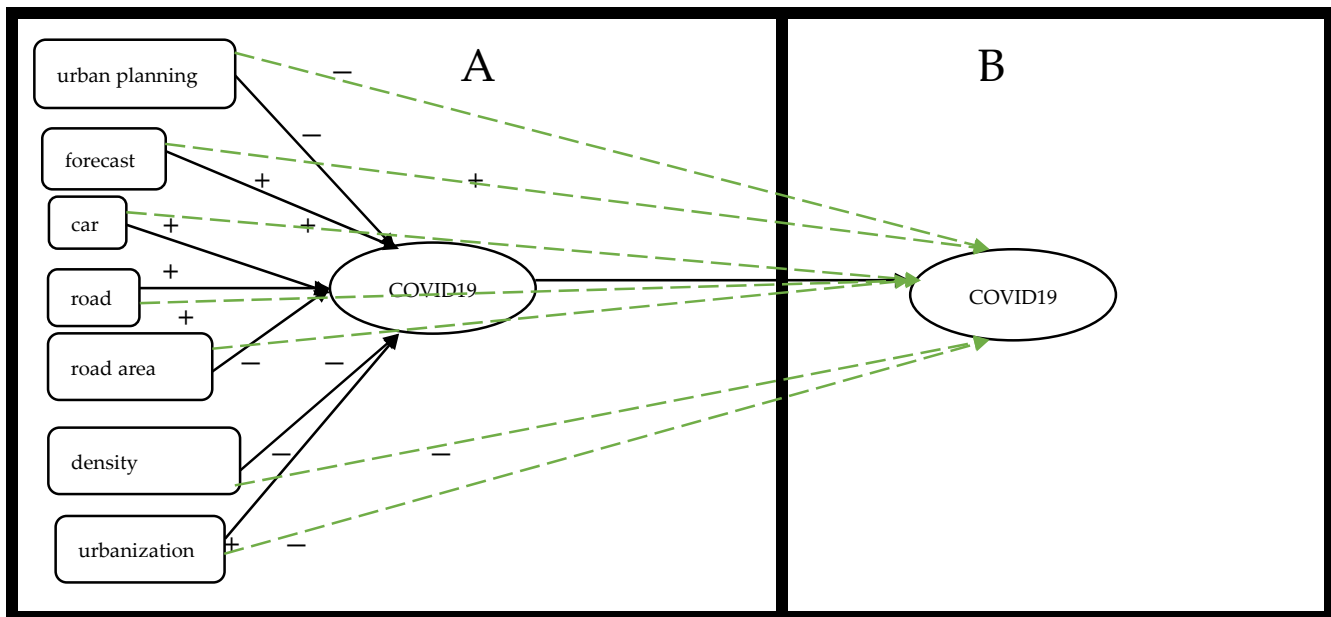
The spatial regression model can analyze the spatial effects of urban development indicators on the spatial dispersal of COVID-19 (Table 3). Table 3 shows that all urban development indicators affect the spatial spread of COVID-19 (this study uses a linear spatial regression model to estimate the spatial effects impacted from urban development, even if rapid increase in the number of infected people in the result finds that urban development indicators significantly affect the number of confirmed COVID-19 cases). The chi-square statistics is supported to reject  $H_0: i.i.d.$ , indicating that the spread of COVID-19 is spatially autocorrelated. The overall adjustment of Pseudo  $R^2$  is moderate. The coefficient of spatial lag variable “COVID19” is significantly greater than zero, indicating that the spatial regression model has a significant explanation and is better than the ordinary least square estimation. The coefficient of the error term is significantly not equal to zero, showing the presence of spatial autocorrelation. The coefficients of “urban planning”, “road area”, and “density” are negative, indicating that there is a negative correlation between “urban planning”, “road area”, and “density” and “COVID19” (Figure 3). Figure 3 shows that the higher the development indicators, such as “urban planning” and “road area”, the greater the social distance between people, and the lower the risk of infection. The higher the index of “density”, the smaller the social distance. However, the risk of infection among the people depends on the overlap of activities and epidemic prevention measures. There is a positive correlation between “forest”, “car”, “road”, and “urbanization” and “COVID19”. “Forest” represents greenness and leisure, and during the spread of the epidemic, it has become the alternative for indoor leisure shopping places and the risk of public infection is relatively high. “Car” is an alternative to public transportation during the spread of the epidemic. People’s use of cars increases mobility and social activities and the risk of infection is relatively high. “Road” can be paths that provide social activities. People’s use of roads increases social opportunities and reduce social distance and the risk of infection is relatively high. “Urbanization” is an indicator of population concentration. The higher the urbanization, the more social activities between people, and the higher the risk of infection.



**Table 3.** COVID-19 spatial regression model in Taiwan, 2022.

COVID19	Coefficient	Standard Error	p-Value
urban planning	−0.1263 ***	0.0150	0.0000
forest	0.0002 ***	0.00006	0.0008
car	0.0021 **	0.0007	0.0030
road	295.9195 *	111.8993	0.0080
road area	−42.9077 **	13.3566	0.0010
urbanization	0.5104 ***	0.0635	0.0000
density	−0.2909 ***	0.0661	0.0000
constant	2067.834 **	662.8678	0.0020
W			
urban planning	−1.4507 *	0.5356	0.0067
forest	0.0400 ***	0.0050	0.0000
car	0.0054 ***	0.0011	0.0000
road	3583.105 ***	650.8499	0.0000
road area	−224.1063 ***	27.3681	0.0000
urbanization	−186.9376 ***	48.3417	0.0000
density	−0.9433 ***	0.1083	0.0000
COVID19	1.1497 ***	0.1153	0.0000
e. COVID19	−14.4189 ***	1.8038	0.0000
Vair (e. COVID19)	165,414.3	58,576.38	
Log likelihood	−128.3633		
Prob > chi2	0.0000		
Pseudo R <sup>2</sup>	0.3629		

note: \*  $p < 0.01$ , \*\*  $p < 0.005$ , \*\*\*  $p < 0.001$ .



**Figure 3.** Causal relationship between COVID-19 and urban development.

The urban development indicators of adjacent districts have external effects, which affect the “COVID19” of a certain city. Figure 3 shows that there is a negative correlation between “urban planning”, “road area”, “urbanization”, and “density” of neighboring cities and the “COVID19” of a city, indicating that when the “urban planning” of neighboring cities has increased, “road area” increases, “urbanization” increases, and “density” increases, and “COVID19” in one city spills over to neighboring cities to increase. The “forest”, “car”, and “road” of neighboring cities are positively correlated with the “COVID19” of a certain city, which means that when the “forest” of neighboring cities increases, the “car” and “road” increase, and a certain city absorbs “COVID19” and increases.

Urban development indicators can be divided into two spatial effects on the spread of COVID-19: direct effect and indirect effect. indirect effect is equal to  $\frac{dCOVID19_A}{dx_B} + \frac{dCOVID19_A}{dx_B} + \dots$  (in both equations,  $COVID19_A$  is the number of confirmed cases of COVID-19 per hundred thousand people of the city A and, in the former,  $x_A$  is the urban development indicator of the city A). The marginal impact of COVID-19 per 100,000 confirmed cases decreases gradually to the marginal impact on neighboring cities (Table 4). Table 4 shows that urban development indicators significantly affect the number of confirmed cases of COVID-19 per 100,000 people, and COVID-19 has the effect of cross-city spread. Overall, “urban planning”, “road area”, “urbanization”, and “density” positively affect the spread of COVID-19, while “forest”, “car”, and “road” negatively affect the spread of COVID-19.

**Table 4.** Direct and indirect effect of space spread of COVID-19 in Taiwan, 2022.

COVID19	Direct Effect	Indirect Effect	Total Effect
urban planning	−0.0458084	7.323299	7.27749
forest	0.0017822	−0.1882867	−0.1865045
car	0.0016994	−0.0359325	−0.0342331
road	98.12325	−18,003.52	−17,905.4
road area	−29.12226	1254.76	1225.637
urbanization	9.905361	855.1364	865.0417
density	−0.2264669	5.863354	5.636887

## 5. Conclusions

Urban development indicators that are divided into density, urbanization, and transportation system and modes of transport significantly affect the spread of COVID-19. Density shows that the social distance between people is short and the risk of infection from interpersonal contact is high. Urbanization shows that interpersonal frequent activities and close contact make the risk of infection high. Transportation systems and transport mode show the feasibility and possibility of mobility, which affect the risk of infection. The path of urban development and the spread of COVID-19 change to an either positive or negative marginal effect due to the spillover and the adsorption effects.

In the process of urban development, interpersonal social activities have increased, and interaction between them is frequent, providing a way to facilitate the spread of the virus. Urban development is closely linked to the spread of COVID-19, and when the outbreak occurs, it spreads across cities, so no city can stay out of the way. The spreading effect of COVID-19 is nothing more than the spillover or adsorption effect. Therefore, in addition to personal epidemic prevention, the goal of disaster reduction must be achieved through the adjustment of urban development. The policy adjustment of urban development indicators can also be achieved through the spatial effect of spillover and adsorption.

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**Institutional Review Board Statement:** This study does not require ethical approval.

**Informed Consent Statement:** This study does not involve humans.

**Data Availability Statement:** The data released from the websites of Taiwan Centers for Disease Control is used in this study. Please visit the websites address <https://www.cdc.gov.tw/>.

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**Conflicts of Interest:** The author declares no conflict of interest.



## Appendix A

Table A1. Original data used in this study.

County	Variable						
	Urban Planning	Forest	Car	Road	Road Area	Urbanization	Density
Yilan County	76.5865	168,384	136,253	0.62	37.01	3.57291	3656
Changhua County	133.797	10,104.1	412,063	2.23	22.17	12.4265	4486
Nantou County	125.415	303,186	172,372	0.51	43.59	3.05423	2253
Yunlin County	97.8476	12,608.9	219,554	1.94	44.4	7.58039	2749
Pingtung County	165.123	156,194	233,371	0.9	37.98	5.94754	2624
Taitung County	88.0492	286,984	62,236	0.37	60.82	2.50452	1488
Hualien County	123.362	372,781	102,370	0.35	58.65	2.66238	1854
Penghu County	10.7864	3242.1	27,623	2.25	27.42	8.50544	4126
Keelung City	74.0575	9395.37	88,345	4.75	18.37	58.3007	4998
Hsinchu City	46.256	2804.21	142,537	5.71	13.23	44.4167	7918
Taipei City	271.8	11,490.8	729,043	4.53	6.91	100	9818
New Taipei City	1228.46	155,483	904,621	1.79	11.36	59.8937	3050
Taichung City	539.177	113,963	953,063	1.96	23.26	24.3189	4208
Tainan City	522.041	54,148.5	588,919	2.09	33.43	23.8834	3055
Taoyuan City	322.431	47,134.1	697,807	2.7	18.4	26.4081	5146
Miaoli County	75.9467	125,946	190,549	1.04	36.73	4.17237	4316
Hsinchu County	54.4983	104,211	200,014	0.79	25.57	3.81776	6453
Chiayi City	60.7557	773.42	82,733	8.7	37.7	101.216	4570
Chiayi County	169.458	79,888.3	160,985	1.21	48.93	8.88561	1262
Kaohsiung City	422.552	170,523	774,130	1.66	20.2	14.3148	5923

## References

- Brenner, N. *Implosions/Explosions: Towards a Study of Planetary Urbanization*; Jovis: Berlin, Germany, 2014.
- Connolly, C.; Keil, R.; Ali, H.S. Extended urbanisation and the spatialities of infectious disease: Demographic change, infrastructure and governance. *Urban Stud.* **2021**, *58*, 245–263. [[CrossRef](#)]
- Ren, X.; Keil, R. *The Globalizing Cities Reader*; Routledge: London, UK, 2017.
- Ali, H.S.; Keil, R. *Networked Disease: Emerging Infections in the Global City*; Wiley-Blackwell: Oxford, UK, 2008.
- Else, H.; Agyepong, I.; Huque, R.; Quayyum, Z. Rethinking health systems in the context of urbanisation: Challenges from four rapidly urbanising low- and middle- income countries. *Br. Med. J. Glob. Health* **2019**, *4*, e001501. [[CrossRef](#)] [[PubMed](#)]
- Moore, R.E.; Kim, Y.; Philpott, C.C. The mechanism of ferrichrome transport through Arn1p and its metabolism in *Saccharomyces cerevisiae*. *Proc. Natl. Acad. Sci. USA* **2003**, *100*, 5664–5669. [[CrossRef](#)] [[PubMed](#)]
- Wu, L.; He, Y.; Jiang, B.; Zhang, D.; Tian, H.; Zuo, F.; Lam, T.H. Very brief physician advice and supplemental proactive telephone calls to promote smoking reduction and cessation in Chinese male smokers with no intention to quit: A randomized trial. *Addiction* **2017**, *112*, 2032–2040. [[CrossRef](#)]
- Bollyky, T.J. *Plagues and the Paradox of Progress: Why the World is Getting Healthier in Worrisome Ways*; The MIT Press: Cambridge, MA, USA, 2019.
- Wolf, M. Rethinking urban epidemiology: Natures, networks and materialities. *Int. J. Urban Reg. Res.* **2016**, *40*, 958–982. [[CrossRef](#)] [[PubMed](#)]
- Quammen, D. *Spillover: Animal Infections and the Next Human Pandemic*; W. W. Norton & Company Inc.: New York, NY, USA, 2012.
- Patlak, M. Book reopened on infectious diseases. *Food Drug Adm. Consum. Mag.* **1996**, *30*, 19–23.
- Smolinsky, M.S.; Hamburg, M.A.; Lederberg, J. *Microbial Threats to Health: Emergence, Detection, and Response*; National Academies Press: Washington, DC, USA, 2003.
- Gubler, D.J. Dengue and dengue hemorrhagic fever. *Clin. Microbiol. Rev.* **1998**, *11*, 480–496. [[CrossRef](#)]
- Gubler, D.J. Epidemic dengue/dengue hemorrhagic fever as a public health, social and economic problem in the 21st century. *Trends Microbiol.* **2002**, *10*, 100–102. [[CrossRef](#)]
- Fauci, A.S.; Touchette, N.A.; Folkers, G.K. Emerging infectious diseases: A 10-year perspective from the National Institute of Allergy and Infectious Diseases. *Emerg. Infect. Dis.* **2005**, *11*, 519–525. [[CrossRef](#)]
- World Health Organization (WHO). *Global Strategic Framework for Integrated Vector Management*; WHO/CDS/CPE/PVC/2004.10; WHO: Geneva, Switzerland, 2004.
- McNeill, W.H. *Plagues and Peoples*, 1st ed.; Anchor Press: Garden City, NY, USA, 1976.
- Wilcox, B.A.; Gubler, D.J.; Pizer, H.F. *The Social Ecology of Infectious Diseases*; Mayer, K.H., Pizer, H.F., Eds.; Academic Press: Cambridge, MA, USA, 2008.

19. Gosce, L.; Johansson, A. Analysing the link between public transport use and airborne transmission: Mobility and contagion in the London underground. *Environ. Health* **2018**, *17*, 84. [[CrossRef](#)]
20. Acevedo, M.A.; Prosper, O.; Lopiano, K.; Ruktanonchai, N.; Caughlin, T.T.; Martcheva, M.; Osenberg, C.W.; Smith, D.L. Spatial heterogeneity, host movement and mosquito-borne disease transmission. *PLoS ONE* **2015**, *10*, 0127552. [[CrossRef](#)]
21. Arino, J. Spatio-temporal spread of infectious pathogens of humans. *Infect. Dis. Model.* **2017**, *2*, 218–228. [[CrossRef](#)] [[PubMed](#)]
22. Balcan, D.; Colizza, V.; Gonçalves, B.; Hu, H.; Ramasco, J.J.; Vespignani, A. Multiscale mobility networks and the spatial spreading of infectious diseases. *Proc. Natl. Acad. Sci. USA* **2009**, *106*, 21484–21489. [[CrossRef](#)]
23. Charu, V.; Zeger, S.; Gog, J.; Bjornstad, O.N.; Kissler, S.; Simonsen, L.; Grenfell, B.T.; Viboud, C. Human mobility and the spatial transmission of influenza in the United States. *PLoS Comput. Biol.* **2017**, *13*, 1005382. [[CrossRef](#)]
24. Chowell, G.; Bettencourt, L.M.A.; Johnson, N.; Alonso, W.J.; Viboud, C. The 1918–1919 influenza pandemic in England and Wales: Spatial patterns in transmissibility and mortality impact. *Proc. R. Soc. B Biol. Sci.* **2008**, *275*, 501–509. [[CrossRef](#)] [[PubMed](#)]
25. Eggo, R.M.; Cauchemez, S.; Ferguson, N.M. Spatial dynamics of the 1918 influenza pandemic in England, Wales and the United States. *J. R. Soc. Interface* **2011**, *8*, 233–243. [[CrossRef](#)]
26. Gog, J.R.; Ballesteros, S.; Viboud, C.; Simonsen, L.; Bjornstad, O.N.; Shaman, J.; Chao, D.L.; Khan, F.; Grenfell, B.T. Spatial transmission of 2009 pandemic influenza in the US. *PLoS Comput. Biol.* **2014**, *10*, 1003635. [[CrossRef](#)]
27. Karl, S.; Halder, N.; Kelso, J.K.; Ritchie, S.A.; Milne, G.J. A spatial simulation model for dengue virus infection in urban areas. *BMC Infect. Dis.* **2014**, *14*, 447. [[CrossRef](#)]
28. Merler, S.; Ajelli, M.; Fumanelli, L.; Gomes, M.F.C.; Piontti, A.P.Y.; Rossi, L.; Chao, D.L.; Longini, I.M.; Halloran, E.M.; Vespignani, A. Spatiotemporal spread of the 2014 outbreak of Ebola virus disease in Liberia and the effectiveness of non-pharmaceutical interventions: A computational modelling analysis. *Lancet Infect Dis.* **2015**, *15*, 204–211. [[CrossRef](#)]
29. Moss, R.; Naghizade, E.; Tomko, M.; Geard, N. What can urban mobility data reveal about the spatial distribution of infection in a single city? *BMC Public Health* **2019**, *19*, 656. [[CrossRef](#)]
30. Riley, S. Large-scale spatial-transmission models of infectious disease. *Science* **2007**, *316*, 1298–1301. [[CrossRef](#)]
31. Riley, S.; Eames, K.; Isham, V.; Mollison, D.; Trapman, P. Five challenges for spatial epidemic models. *Epidemics* **2015**, *10*, 68–71. [[CrossRef](#)]
32. Sattenspiel, L.; Dietz, K. A structured epidemic model incorporating geographic mobility among regions. *Math. Biosci.* **1995**, *128*, 71–91. [[CrossRef](#)]
33. Viboud, C.; Bjornstad, O.N.; Smith, D.L.; Simonsen, L.; Miller, M.A.; Grenfell, B.T. Synchrony, waves, and spatial hierarchies in the spread of influenza. *Science* **2006**, *312*, 447–451. [[CrossRef](#)]
34. Xu, B.; Tian, H.; Sabel, C.E.; Xu, B. Impacts of road traffic network and socioeconomic factors on the diffusion of 2009 pandemic influenza A (H1N1) in Mainland China. *Int. J. Environ. Res. Public Health* **2019**, *16*, 1223. [[CrossRef](#)]
35. Charaudeau, S.; Pakdaman, K.; Boelle, P.-Y. Commuter mobility and the spread of infectious diseases: Application to influenza in France. *PLoS ONE* **2014**, *9*, 83002. [[CrossRef](#)] [[PubMed](#)]
36. Church, R.L.; Cova, T.J. Mapping evacuation risk on transportation networks using a spatial optimization model. *Transp. Res. Part C Emerg. Technol.* **2000**, *8*, 321–336. [[CrossRef](#)]
37. Ebihara, M.; Ohtsuki, A.; Iwaki, H. A model for simulating human behavior during emergency evacuation based on classificatory reasoning and certainty value handling. *Comput. Aided Civ. Infrastruct. Eng.* **1992**, *7*, 63–71. [[CrossRef](#)]
38. Helbing, D.; Buzna, L.; Johansson, A.; Werner, T. Self-organized pedestrian crowd dynamics: Experiments, simulations, and design solutions. *Transp. Sci.* **2005**, *39*, 1–24. [[CrossRef](#)]
39. Helbing, D.; Johansson, A.; Al-Abideen, H.Z. Dynamics of crowd disasters: An empirical study. *Phys. Rev. E* **2007**, *75*, 046109. [[CrossRef](#)]
40. Johansson, A. Constant-net-time headway as a key mechanism behind pedestrian flow dynamics. *Phys. Rev. E* **2009**, *80*, 026120. [[CrossRef](#)]
41. Shi, J.; Ren, A.; Chen, C. Agent-based evacuation model of large public buildings under fire conditions. *Autom. Constr.* **2009**, *18*, 338–347. [[CrossRef](#)]
42. Yu, W.; Johansson, A. Modeling crowd turbulence by many-particle simulations. *Phys. Rev. E* **2007**, *76*, 046105. [[CrossRef](#)]
43. Brauer, F. Compartmental models in epidemiology. In *Mathematical Epidemiology*; Springer: Berlin/Heidelberg, Germany, 2008; pp. 19–79.
44. Colizza, V.; Barthélemy, M.; Barrat, A.; Vespignani, A. Epidemic modeling in complex realities. *Comptes Rendus Biol.* **2007**, *330*, 364–374. [[CrossRef](#)]
45. Kermack, W.; McKendrick, A. A contribution to the mathematical theory of epidemics. *Proc. R. Soc. A* **1927**, *115*, 700–721.
46. Newman, M.E. Spread of epidemic disease on networks. *Phys. Rev. E* **2002**, *66*, 016128. [[CrossRef](#)]
47. Pastor-Satorras, R.; Vespignani, A. Epidemic dynamics and endemic states in complex networks. *Phys. Rev. E* **2001**, *63*, 066117. [[CrossRef](#)]
48. Zhao, Z.; Shaw, S.-L.; Xu, Y.; Lu, F.; Chen, J.; Yin, L. Understanding the bias of call detail records in human mobility research. *Int. J. Geogr. Inf. Sci.* **2016**, *30*, 1738–1762. [[CrossRef](#)]
49. Jones, K.E.; Patel, N.G.; Levy, M.A.; Storeygard, A.; Balk, D.; Gittleman, J.L.; Daszak, P. Global trends in emerging infectious diseases. *Nature* **2008**, *451*, 990–993. [[CrossRef](#)]

50. Woolhouse, M.E.J.; Gowtage-Sequeria, S. Host range and emerging and reemerging pathogens. *Emerg. Infect. Dis.* **2005**, *11*, 1842–1847. [[CrossRef](#)] [[PubMed](#)]
51. Karesh, W.B.; DPhil, A.D.; Lloyd-Smith, J.O.; Lubroth, J.; Dixon, M.A.; Bennett, M.; Aldrich, S.; Harrington, T.; Formenty, P.; Loh, E.H.; et al. Ecology of zoonoses: Natural and unnatural histories. *Lancet* **2012**, *380*, 1936–1945. [[CrossRef](#)]
52. Hassell, J.M.; Begon, M.; Ward, M.J.; Fevre, E.M. Urbanization and disease emergence: Dynamics at the wildlife–livestock–human interface. *Trends Ecol. Evol.* **2017**, *32*, 55–67. [[CrossRef](#)]
53. Cliff, A.D.; Ord, J.K. *Spatial Autocorrelation*; Pion: London, UK, 1973.
54. Cliff, A.D.; Ord, J.K. *Spatial Processes: Models and Applications*; Pion: London, UK, 1981.
55. LeSage, J.P.; Pace, R.K. Models for spatially dependent missing data. *J. Real Estate Financ. Econ.* **2004**, *29*, 233–254. [[CrossRef](#)]
56. LeSage, J.P.; Pace, R.K. *Introduction to Spatial Econometrics*; Taylor and Francis Group, CRC Press: Boca Raton, FL, USA, 2009.
57. Whittle, P. On stationary processes in the plane. *Biometrika* **1954**, *41*, 434–449. [[CrossRef](#)]
58. Ballard, K.; Bone, C. Exploring spatially varying relationships between Lyme disease and land cover with geographically weighted regression. *Appl. Geogr.* **2021**, *109*, 102383. [[CrossRef](#)]
59. Darmofal, D. *Spatial Analysis for the Social Sciences*; Cambridge University Press: New York, NY, USA, 2015.
60. Drukker, D.M.; Egger, P.; Prucha, I. On two-step estimation of a spatial autoregressive model with autoregressive disturbances and endogenous regressors. *Econom. Rev.* **2013**, *32*, 686–733. [[CrossRef](#)]
61. Kelejian, H.H.; Prucha, I. Specification and estimation of spatial autoregressive models with autoregressive and heteroskedastic disturbances. *J. Econom.* **2010**, *157*, 53–67. [[CrossRef](#)]
62. Ku, C.-A. Incorporating spatial regression model into cellular automata for simulating land use change. *Appl. Geogr.* **2016**, *69*, 1–9. [[CrossRef](#)]
63. Lee, L.-F.; Liu, X.; Lin, X. Specification and estimation of social interaction models with network structures. *Econom. J.* **2010**, *13*, 145–176. [[CrossRef](#)]
64. Nkeki, F.N.; Asikhia, M.O. Geographically weighted logistic regression approach to explore the spatial variability in travel behaviour and built environment interactions: Accounting simultaneously for demographic and socioeconomic characteristics. *Appl. Geogr.* **2019**, *108*, 47–63. [[CrossRef](#)]
65. Sathler, D.; Adamo, S.; Lima, E.E.; Macedo, D.R.; Sherbinin, A.D.; Kim-Blanco, P. Assessing the regional context of migration in the Brazilian Amazon through spatial regression modeling. *Appl. Geogr.* **2019**, *109*, 102042. [[CrossRef](#)]
66. Tu, W.; Ha, H.; Wang, W.; Liu, L. Investigating the association between household firearm ownership and suicide rates in the United States using spatial regression models. *Appl. Geogr.* **2020**, *124*, 102297. [[CrossRef](#)]
67. Waller, L.A.; Gotway, C.A. *Applied Spatial Statistics for Public Health Data*; Wiley-Interscience: Hoboken, NJ, USA, 2004.
68. Yuan, M.; Huang, Y.; Shen, H.; Li, T. Effects of urban form on haze pollution in China: Spatial regression analysis based on PM2.5 remote sensing data. *Appl. Geogr.* **2018**, *98*, 215–223.