PERSPECTIVE

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Perspective

Perspectives on modelling epidemics with human mobility

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Abstract – Human mobility serves as a fundamental component in shaping the contact networks through which infectious diseases propagate during pandemics. It significantly influences the spatial and temporal patterns of disease transmission among individuals. Traditional epidemic models often struggle to capture the complexity of these heterogeneous contact patterns. In contrast, models incorporating human mobility, which account for the movement of individuals across regions, offer a detailed perspective on micro-level interactions and their impact on disease spread. The discussion highlights four types of epidemic models that integrate human mobility, including compartment models, complex network models, agent-based models and machine learning models, emphasising their crucial roles in epidemic prediction and control. Additionally, it provides insights into the broader implications of human mobility on dynamic-modelling and decision-making within the context of epidemics.

Introduction. – In line with the definition from WHO, a disease outbreak occurs when the incidence of a disease exceeds what is typically expected in a defined community, geographical area, or season [1]. Outbreaks are sustained by infectious agents that spread either directly between people, through exposure to an animal reservoir or environmental source, or via insect or animal vectors [2]. Figuring out the origins of infectious diseases and monitoring their spread among individuals has historically been challenging [3,4]. Epidemic models, as useful methods to depict the process of pandemics and reveal the inherent mechanism of epidemic spread, can effectively address these issues [5,6].

Over the centuries, epidemic modelling began with the use of mathematical approaches to understand disease dynamics (*e.g.*, assessing the impact of smallpox vaccination) [7], then has since evolved significantly. A milestone occurred with the development of the SIR (Susceptible-Infected-Recovered) model [8], which simplified the complex dynamics of disease spread by categorizing populations into specific compartments. This model, along with its variations like SIS (Susceptible-Infected-Susceptible) and SEIR (Susceptible-Exposed-Infected-Recovered), has become fundamental in epidemic research, particularly in studies of outbreaks like SARS [9], EBOV [10], and coronavirus disease [2]. The widespread occurrence of epidemics has often coincided with periods of increased human mobility, such as during World Wars and large-scale migrations, as well as advances in the transportation technology. The growing speed and frequency of human movement, facilitated by modern transportation systems, have further amplified the risk of rapid, large-scale disease transmission [11]. Traditional epidemic models that do not account for human mobility struggle to capture the complex, large-scale, and heterogeneous transmission patterns observed across space and time [12].

Taking into account the roles of local healthcare systems, vaccination rates, and socio-economic variables, previous studies have shown that human mobility data is a more effective predictor of epidemic transmission than traditional indicators like search indices [13], population size [14], or city GDP [15]. For example, different human mobility patterns (*e.g.*, short ties, long ties, strong ties, and strong long ties, as shown in fig. 1 exhibit different kinds of influence on the spread of the COVID-19 pandemic [16]. Beyond COVID-19, H1N1 [17], cholera [18], dengue [19], MPOX [20], and Ebola [21] have also demonstrated the significance of mobility patterns in shaping infection dynamics. Accurately measuring and leveraging mobility data has become increasingly critical as human mobility behaviours significantly influence the rate and extent of epidemic spread. Fortunately, developments in

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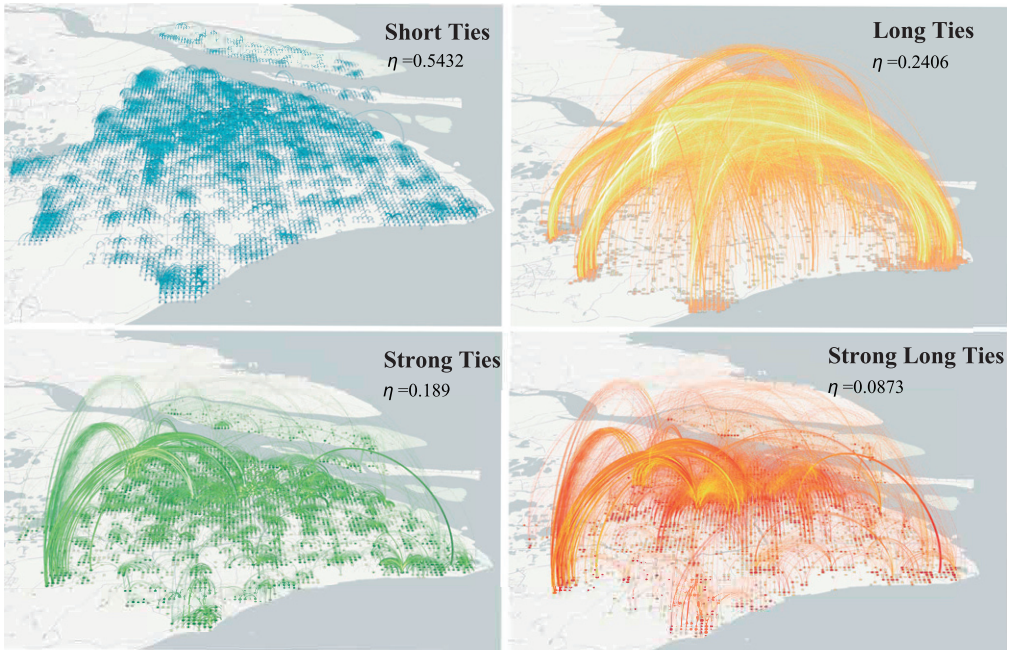

Fig. 1: The distribution of different human mobility patterns with Moran's indices η. The linewidth represents the strength of the connection [16].

modern tracking technologies have greatly enhanced the precision, comprehensiveness, and real-time observations of human movement, leading to substantial improvements in human mobility data collection [22].

This review aims to explore the effective incorporation of human mobility information into risk assessment and epidemic modelling. Specifically, we examine widely adopted epidemic models that incorporate human mobility and introduce four distinct types of models: compartment models, complex network models, agent-based models, and machine learning models. By leveraging mobility data, these models accurately and promptly capture the dynamics of epidemic spread, thereby enhancing prediction accuracy and supporting targeted control strategies.

Compartment models. – Compartment models are mathematical frameworks that divide a population into groups based on individual states to simulate and predict the spread of epidemics [2,12,23]. These models assume people mix homogeneously, *i.e.*, that each individual in the population has an equal chance of interacting with others, as exemplified by the SI, SIS, SIR, SEIR, and SEIRS (Susceptible, Exposed, Infected, and Recovered) models. Hitherto, the compartment models are among the most prevalent mathematical frameworks utilized for epidemic modelling [24]. However, the assumption of homogeneous mixing does not accurately represent real-world situations. Human mobility exhibits strong heterogeneity for both the number and types of contacts, making it impossible for individuals to engage in uniform contact across populations [25]. To account for mobility, researchers often use metapopulation models, where each population with specific attributes is treated as a separate compartment, and movement between these compartments is modelled explicitly [26,27].

Considering the exposure and contamination rate, a metapopulation SIR model that integrates human mobility can be defined as follows [28]:

$$\begin{aligned}\frac{\mathrm{d}S_i}{\mathrm{d}t} &= \mu(H_i - S_i) - \mathcal{F}_i(t)S_i + \rho R_i,\\ \frac{\mathrm{d}I_i}{\mathrm{d}t} &= \mathcal{F}_i(t)S_i - (\gamma + \mu + \alpha)l_i,\\ \frac{\mathrm{d}R_i}{\mathrm{d}t} &= \gamma I_i - (\rho + \mu)R_i,\end{aligned} \tag{1}$$

where $S_i(t)$, $I_i(t)$, and $R_i(t)$ represent the number of susceptible, infected, and recovered individuals at node i over time. The parameter H_i denotes the population size at node i, while μ represents the natural mortality rate. Infected individuals recover at rate γ or die at rates μ (natural mortality) and α (disease-induced mortality). Temporary immunity is lost at rate ρ, returning individuals to the susceptible pool.

The contact rate $\mathcal{F}_i(t)$ (eq. (2)) includes both local interactions and mobility-related transmission, influenced by the contamination rate β, the half-saturation constant K, and mobility patterns defined by a connection matrix $\mathcal{Q}_{ij}$ (eq. (3)),

$$\mathcal{F}_i(t) = \beta\left(\frac{(1-m)\,B_i}{K+B_i} + \frac{m\sum_{j=1}^{n}\mathcal{Q}_{ij}B_j}{K+B_j}\right). \tag{2}$$

The human mobility patterns are defined as an OD (Origin-Destination) matrix in which individuals leave their original node (say i) with a probability m, reach their target location (say j) with a probability $\mathcal{Q}_{ij}$, and then come back to node i.

$$\mathcal{Q}_{ij} = \frac{H_i e^{-\frac{d_{ij}}{D}}}{\sum\limits_{k\neq i}^{N} H_k e^{-\frac{d_{ik}}{D}}}, \tag{3}$$

where d_{ij} is the shortest-path distance between node i and j, and D is the deterrence cutoff distance.

Subsequently, $\mathcal{O}_i(t)$ is introduced to account for the effects of the increase in exposure and contamination rate due to the increased population density [26], which can be represented by the following:

$$\mathcal{O}_i(t) = \exp\left(\frac{\omega}{H_i}\sum_{i=1}^{N} M_{ij}(t)H_j\right). \tag{4}$$

This increase is modelled as an exponential function with the exponent involving parameter ω and the calibrated human mobility matrix $M_{ij}(t)$, derived from the base matrix $\mathcal{Q}_{ij}(t)$ [26].

While effective for basic epidemic modelling, compartment models face limitations when incorporating human mobility, as they often assume homogeneous mixing within compartments and overlook complex interaction patterns between individuals across different locations. These models may fail to capture the intricate dynamics of disease spread facilitated by varying mobility behaviours, social structures, and high-connectivity hubs, such as transport centres or densely populated urban areas.

Complex network models. – Compared with compartment models, complex network models offer a more nuanced approach by representing populations as interconnected nodes and links, capturing heterogeneous contact patterns and mobility flows. A complex network model is a mathematical framework that represents individuals or subpopulations as networks of nodes connected by edges (representing interactions or movements). Specifically, the transmission dynamics of infectious diseases are normally elucidated through two interconnected types of complex network models. The first is the mobility network model, with nodes representing subpopulations or distinct geographic locations, and edges denoting the amount or probabilities of individuals moving between them [29]. The second is the contact network model, taking individuals as nodes, and edges as the interaction behaviours (contact frequency or rates) which facilitate the spread of epidemics [30].

Mobility network models leverage OD matrices to analyse and quantify the impact of individuals' movements on spatial interactions and population dynamics. For instance, by incorporating human mobility data from airline networks, such models can predict how an epidemic propagates internationally through air travel connections [31]. These models assume that individuals within the same area are homogeneously mixed and transition between disease states based on predefined dynamics. To simplify the modelling process, the population at each location is assumed to remain stable over time, with disease transmission occurring primarily within local communities. The movement of individuals between subpopulations is conceptualised as a diffusion process and can be described as [32]

$$\frac{\mathrm{d}N_i}{\mathrm{d}t} = -\sum_{j=1}^{K} f_{i,j}N_i + \sum_{j=1}^{K} f_{j,i}N_j, \tag{5}$$

where N_i counts the number of individuals currently located at location i, and the constant $f_{i,j}$ represents the rate at which individuals located at i travel to j, where $f_{i,j} = 0$ for all i.

Contact network models focus on the design of contact patterns by integrating human mobility with epidemic spread, providing a finer level of resolution. These microscopic and granular models capture detailed interactions between individuals, thereby enhancing the accuracy of infection rate estimations and strengthening predictive capabilities through the incorporation of precise human mobility data. Regarding the human contact behaviours as discrete-time Markov processes [33,34], a contact network model in heterogeneous networks with recurrent mobility patterns can be modelled as eq. (6). It assumes that infected individuals are uniformly distributed in residence i and the number of infected neighbours for each node is k_i^{in},

$$k_i^{\mathrm{in}}(p, \mathrm{infected}) = k_i^{\mathrm{in}}(1-p)\psi_i(t). \tag{6}$$

Similarly, the number of infected neighbours across locations for individuals in residence i can be calculated as

$$k_i^{\mathrm{out}}(p, \mathrm{infected}) = \sum K_{ij}(1-p)\psi_j(t), \tag{7}$$

where K_{ij} is the average number of edges coming from individuals in location i to location j.

Both mobility and contact network models are increasingly utilized in epidemiology to understand the intricate patterns of disease spread, which provide a fine-grained and non-linear representation of disease spread by explicitly modelling the contact structure between individuals [35]. Complex network models capture the diverse and heterogeneous nature of human interactions, providing deeper insights into the roles of different individuals and groups in transmitting infectious diseases. However, these models also present challenges. Accurate modelling requires detailed data on contact patterns and interactions, which can be challenging. Additionally, complex network models can be computationally intensive, especially when dealing with large populations and multiple layers of interactions. Validating these models is also tricky due to the dynamic and multi-faceted nature of real-world contact networks.

Agent-based models. – Compartmental models simplify human behaviour by assuming uniformity within compartments or by weakening individual behaviour patterns. These approaches lead to a predominant focus on global state changes and transitions, often neglect the nuanced interactions between individuals. The human mobility behaviour is inherently random, unpredictable, and diverse. Ignoring individual autonomy, preferences, and aversions in movements limits the ability of models to simulate the spread of infectious diseases accurately. Incomplete knowledge of human interactions and mobility processes hinders the ability to describe disease spread fully [36]. Agent-based models (ABMs) emphasise the dynamics of epidemic spreading by focusing on human behaviours such as movement, staying, and interactions. This granular approach allows ABMs to capture the complexities of human behaviour and mobility more effectively than aggregate models, leading to more accurate and realistic predictions of epidemic spreading [37].

ABMs simulate the contact behaviours following specific ways in a more detailed dimension, and the mobility patterns determine the likelihood of contact between agents [38,39]. In general, artificial society models simulate interactions between agents based on predefined rules, focusing on emergent behaviours such as resource allocation, information diffusion, or crowd dynamics. They are rule-driven and ideal for studying cause-effect relationships in social systems [40]. Each agent is assigned a mobility trajectory dictating their daily routines, interactions, and movements between locations. Agents' decisions to visit public places or stay home will influence their exposure risk. Population ABMs focus on demographic structures and dynamics, analysing variables like age, birth rates, and mortality to predict population trends and evaluate policy impacts [15]. Additionally, this model also includes an individual profile for each agent, defining the main social characteristics and health conditions used during interactions [41].

From other perspectives, geographic models integrate spatial data to examine how location, distance, and movement influence human behaviour, often applied in urban planning, disaster response, and epidemiology [40]. These models rely on GIS (Geographic Information System) for spatial precision and link mobility patterns to environmental constraints [42]. Finally, grid computing serves as the backbone for large-scale simulations [43], distributing computational tasks across multiple machines to handle the high demands of complex models like artificial societies or geographic simulations, making it essential for scaling up and integrating data-intensive processes. Together, these ABMs provide a comprehensive toolkit for analysing and simulating complex human mobility systems.

Machine learning models. – With advancements in computational and statistical technologies, machine learning (ML) methods have been extensively explored and applied in epidemic modelling. These models can handle large, complex datasets to enhance the predictive and real-time response capabilities. Interpretable ML models, such as Bayesian [44] and Autoregressive Integrated Moving Average (ARIMA) [45], provide clear insights into the human mobility factors influencing disease transmission. In contrast, black-box models like Deep Neural Network (DNN) [46], Graph Neural Network (GNN) [47], and Long Short-Term Memory (LSTM) [48], perform remarkably in epidemic prediction tasks with the non-linear function mechanism. Usually, the non-interpretable models focus more on the inherent relevant patterns between human mobility data. Therefore, it usually performs better than interpretable models in most sub-stream tasks in the context of epidemics. However, how to balance the equilibrium between accuracy and interpretability is always the challenge that scholars have faced for a long time.

Interpretable ML models leverage historical mobility data embedded in inherent human mobility patterns to forecast disease outbreaks and assess the impact of interventions such as vaccination and travel restrictions. The Bayesian method is used to model the mobility change, where the prior distribution represents our initial view of the parameter in the absence of data, and the posterior distribution combines the observed data and prior information to estimate the model's parameters. By treating mobility patterns as probabilistic inputs, these models can assess the likelihood of spreading disease between regions or populations. For example, a conditional Bayesian spatial modelling framework was proposed to assess perceived infection risk [44]. Conditional on the selected covariates, the percentage in mobility is assumed to follow a Beta distribution:

$$\mathbb{E}[y|\mathbf{x}] \sim B(\mu(\mathbf{x}), \phi), \tag{8}$$

where $\mathbf{x}$ is the set of fixed covariates, given by the selected principal component constructed in PCA (Principal Component Analysis) and the epidemiological covariate; ϕ is the precision parameter of the Beta distribution and $\mu(\mathbf{x})$ is the mean. This integration enables the Bayesian method to reveal how each covariate affects mobility and, in turn, disease transmission risk. This probabilistic treatment not only improves the accuracy of identifying transmission pathways and outbreak hotspots but also allows for quantifying uncertainty, making it a valuable tool for policy-making.

ARIMA models are time-series forecasting techniques that capture human mobility data trends, cycles, and seasonality. In the Autoregressive Integrated Moving Average with Exogenous Inputs (ARIMAX) method, human mobility data serve as the exogenous input [45]. The parameters of the ARIMAX model are: p, the number of autoregressive terms; d, the number of non-seasonal differences needed for stationary; q, the number of lagged forecast errors in the prediction equation; n, the number of exogenous variables; η, a constant; and, ϕ_i, for $i = 1, \ldots, p$, θ_j, for $j = 1, \ldots, q$, and ζ_l, for $l = 1, \ldots, n$.

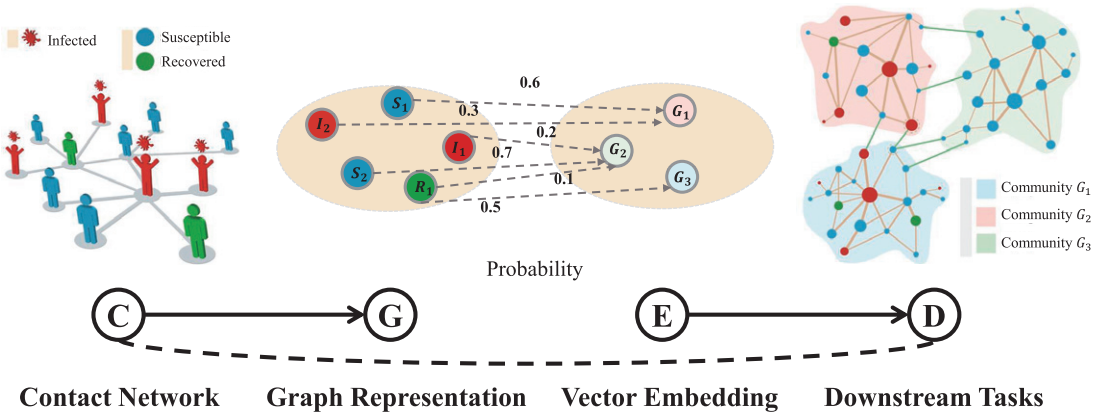

Fig. 2: The process of transforming a contact network to embedding vectors for downstream tasks [47,49].

Mathematically, this model can be formulated as

$$W_t = \eta + \sum_{i=1}^{p} \phi_i W_{t-i} - \sum_{j=1}^{q} \theta_j e_{t-j} + \sum_{l=1}^{n} \zeta_l Y_l, \qquad (9)$$

where W_t and W_{t-i} for $i = 1, \ldots, p$, are the predicted values of the time series; Y_l, for $l = 1, \ldots, n$, stands for the exogenous variables of human mobility; and e_{t-j}, for $j = 1, \ldots, q$, represents the error terms.

Non-interpretable ML models are becoming increasingly prevalent in epidemic modelling with the development modern artificial intelligence. A basic neural network model consists of layers of interconnected neurons, where each neuron processes information by applying weighted inputs and a non-linear activation function. These models are well-suited for handling complex, non-linear problems, offering powerful tools for understanding and predicting disease dynamics in ways that traditional ML methods may struggle to achieve. For example, a DNN (IPSO-DNN) model is proposed [46] to predict the effect of social distancing during epidemics, which have been constructed to evaluate people's mobility pattern changes. Human mobility data is scaled and split into training and testing sets, ensuring features are normalized to improve learning effect, and then the optimized DNN model predicts the impact of social distancing. This model explains how keeping appropriate social distancing helps flatten infectious diseases.

The GNNs can receive the graph-structured human mobility data such as the OD matrix and contact matrix, making them ideal for modelling the interconnectedness of individuals. A Deep Graph Diffusion Infomax (DGDI) was proposed to address the problems caused by limited human mobility data [47]. This model computes the relevance score between a diffusion and a location from the micro perspective of mobility modelling. Figure 2 demonstrates the comprehensive workflow for converting a contact network into embedding vectors, which are essential for various downstream tasks such as high-risk area identification, epidemic spreading prediction, and community intervention control. Another GNN model addresses the changes in spatiotemporal travel mobility and community structure detection induced by the pandemic, which is based on the premise of normalized preparedness [50]. This method integrates graph learning and optimization in an end-to-end training process, constructing dynamic travel networks that uncover shifts in user mobility patterns and explore correlations between different travel modes. These GNNs capture the spatial and relational dependences in mobility networks, learning complex patterns of disease spread through interconnected communities.

LSTMs specifically address the challenges of temporal dependences in the context of pandemics. This feature makes LSTM effective for rapidly probing and quantifying the effects of government interventions (*e.g.*, lock-down [22] and re-opening strategies [2]). During COVID-19, a LSTM model based on graph learning and optimization was proposed to uncover changes in user mobility patterns [48]. This model was trained on Google and Uncast mobility data, and the experiment results suggested that there was a significant trip volume and connectivity reduction during the pandemic, while post-pandemic recovery showed a shift towards more polycentric travel patterns. By capturing temporal patterns and trends in human movement, LSTMs can model the non-linear relationships between past mobility behaviours and current infection levels, providing critical insights even when dealing with complex, time-dependent data.

Conclusion and perspectives. – Human mobility plays a pivotal role in understanding, predicting, and controlling the spread of epidemics. By leveraging real-time mobility data, models such as metapopulation models, complex network models, and agent-based models (ABMs) provide a more detailed and granular representation of epidemic transmission dynamics across diverse geographical regions. Furthermore, advanced machine learning techniques have revolutionised predictive capabilities by efficiently processing large-scale human mobility

datasets, uncovering the underlying mechanisms that drive epidemic propagation with unprecedented depth and precision.

The integration of human mobility data into epidemic models has transitioned from theoretical exploration to practical necessity for global health security. This approach offers crucial insights into disease dynamics and human interactions, bridging the gap between theoretical models and real-world applications. To fully realize this potential, future research and development should focus on several key areas:

1) *Advancing high-resolution digital contact tracing.* Digital contact tracing has demonstrated its effectiveness in the public response to the COVID-19 pandemic by enabling rapid identification and isolation of high-risk individuals. To maximize its utility, challenges related to data accessibility and privacy must be addressed. Techniques like differential privacy, federated learning, and multi-source data harmonization can safeguard individual anonymity while ensuring data reliability, making digital contact tracing more robust and universally applicable. Besides, the authorities should consider the data ownership and informed consents to provide legal ways to enact high-resolution digital contact tracing.

2) *Enhancing large-scale computational capacity.* The scalability and predictive power of epidemic models are directly linked to computational resources. Leveraging parallel and grid computing, coupled with advanced artificial intelligence techniques, is essential for handling the complex datasets generated by human mobility tracking. Large-scale simulations, powered by these technologies, can provide critical insights into outbreak scenarios and identify high-risk areas. Investment in robust computational infrastructure is therefore paramount.

3) *Integrating real-time human mobility into epidemic models.* This integration enables models to dynamically reflect current movement patterns, enhancing their ability to identify emerging hot spots and evaluate the effectiveness of intervention strategies. However, incorporating real-time data presents challenges such as ensuring data accuracy, managing the high velocity of incoming information, and maintaining seamless data flow between mobility sources and epidemiological frameworks. Leveraging advanced machine learning techniques and robust data processing pipelines can address these challenges by enabling efficient real-time data assimilation, improving model responsiveness, and ensuring the reliability of predictions.

4) *Establishing open and accessible data repositories.* Establishing open and accessible data repositories is vital for fostering global collaboration and accelerating research. These platforms should prioritize transparency, reproducibility, and equitable access to high-quality human mobility data. Standardized data formats, clear metadata documentation, and robust data governance frameworks are essential for ensuring data interoperability and facilitating data sharing.

5) *Addressing data biases.* Human mobility data is susceptible to various biases stemming from factors such as demographic representation, data collection methodologies, and geographic coverage. Robust statistical methods and rigorous validation techniques are crucial for mitigating these biases and ensuring model reliability. Specific statistical approaches should be employed to correct for sampling bias, measurement error, and other data imperfections. Careful consideration of these biases is essential for generating accurate and reliable model outputs.

6) *Translating models into actionable policies.* The ultimate goal of epidemic modelling is to inform effective public health interventions. Translating model outputs into actionable policies requires clear communication and collaboration between researchers, policymakers, and public health officials. Models should be designed to provide concrete recommendations for interventions such as targeted lockdowns, resource allocation, and vaccination campaigns.

By focusing on these key areas, we can unlock the full potential of human mobility data in epidemic modelling, leading to more effective strategies for preventing and mitigating future outbreaks and strengthening global health security.

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REFERENCES

[1] `www.emro.who.int/health-topics/disease-outbreaks`.
[2] Zhang J., Tan S., Peng C., Xu X., Wang M., Lu W., Wu Y., Sai B., Cai M., Kummer A. G., Chen Z., Zou J., Li W., Zheng W., Liang Y., Zhao Y., Vespignani A., Ajelli M., Lu X. and Yu H., *Proc. Natl. Acad. Sci. U.S.A.*, **120** (2023) e2306710120.
[3] Lu X., Bengtsson L. and Holme P., *Proc. Natl. Acad. Sci. U.S.A.*, **109** (2012) 11576.
[4] Cohen M. L., *Nature*, **406** (2000) 762.
[5] Hess A., Hummel K. A., Gansterer W. N. and Haring G., *ACM Comput. Surv.*, **48** (2015) 38.
[6] Chen Y., Xie N., Xu H., Chen X. and Lee D.-H., *IEEE Trans. Intell. Transp. Syst.*, **25** (2024) 2139.
[7] Dietz K. and Heesterbeek J. A. P., *Math. Biosci.*, **180** (2002) 1.
[8] Poletto C., Gomes M. F., Piontti A. P. Y., Rossi L., Bioglio L., Chao D. L., Longini I. M. jr., Halloran M. E., Colizza V. and Vespignani A., *Eurosurveillance*, **19** (2014) 20936.

[9] Colizza V., Barrat A., Barthélemy M. and Vespignani A., *BMC Med.*, **5** (2007) 34.

[10] Wesolowski A., Buckee C. O., Bengtsson L., Wetter E., Lu X. and Tatem A. J., *PLoS Curr.*, **6** (2014) ecurrents.outbreaks.0177e7fcf52217b8b634376e2f3efc5e.

[11] Bouzaghrane M. A., Obeid H., González M. and Walker J., *EPJ Data Sci.*, **13** (2024) 1.

[12] Liu H., Wang J., Liu J., Ge Y., Wang X., Zhang C., Cleary E., Ruktanonchai N. W., Ruktanonchai C. W. and Yao Y., *Sustain. Cities Soc.*, **99** (2023) 104872.

[13] Hu T., Wang S., She B., Zhang M., Huang X., Cui Y., Khuri J., Hu Y., Fu X., Wang X., Wang P., Zhu X., Bao S., Guan W. and Li Z., *Int. J. Digit. Earth*, **14** (2021) 1126.

[14] Chen Z., Lemey P. and Yu H., *The Lancet Microbe*, **5** (2024) e81.

[15] Jia J. S., Lu X., Yuan Y., Xu G., Jia J. and Christakis N. A., *Nature*, **582** (2020) 389.

[16] Mou J., Tan S., Zhang J., Sai B., Wang M., Dai B., Ming B.-W., Liu S., Jin Z., Sun G., Yu H. and Lu X., *PNAS Nexus*, **3** (2024) 515.

[17] Yang Y., Sugimoto J. D., Halloran M. E., Basta N. E., Chao D. L., Matrajt L., Potter G., Kenah E. and Longini I. M., *Science*, **326** (2009) 729.

[18] Bengtsson L., Gaudart J., Lu X., Moore S., Wetter E., Sallah K., Rebaudet S. and Piarroux R., *Sci. Rep.*, **5** (2015) 8923.

[19] Bomfim R., Pei S., Shaman J., Yamana T., Makse H. A., Andrade J. S., Neto A. S. L. and Furtado V., *J. R. Soc. Interface*, **17** (2020) 20200691.

[20] Murayama H., Pearson C. A. B., Abbott S., Miura F., Jung S., Fearon E., Funk S. and Endo A., *J. Infectious Diseases*, **229** (2024) 59.

[21] Malvy D., McElroy A. K., de Clerck H., Günther S. and van Griensven J., *The Lancet*, **393** (2019) 936

[22] Tan S., Lai S., Fang F., Cao Z., Sai B., Song B., Dai B., Guo S., Liu C. and Cai Z., *Nat. Sci. Rev.*, **8** (2021) nwab148.

[23] Sallah K., Giorgi R., Bengtsson L., Lu X., Wetter E., Adrien P., Rebaudet S., Piarroux R. and Gaudart J., *Int. J. Health Geogr.*, **16** (2017) 42.

[24] Lv S., Yang H., Lu X., Zhang F. and Wang P., *ISPRS Int. J. Geo-Inf.*, **13** (2024) 267.

[25] Barabási A.-L., *Nature*, **435** (2005) 207.

[26] Finger F., Genolet T., Mari L., de Magny G. C., Manga N. M., Rinaldo A. and Bertuzzo E., *Proc. Natl. Acad. Sci. U.S.A.*, **113** (2016) 6421.

[27] Gatto M., Bertuzzo E., Mari L., Miccoli S., Carraro L., Casagrandi R. and Rinaldo A., *Proc. Natl. Acad. Sci. U.S.A.*, **117** (2020) 10484.

[28] Rinaldo A., Bertuzzo E., Mari L., Righetto L., Blokesch M., Gatto M., Casagrandi R., Murray M., Vesenbeckh S. M. and Rodriguez-Iturbe I., *Proc. Natl. Acad. Sci. U.S.A.*, **109** (2012) 6602.

[29] Hou X., Gao S., Li Q., Kang Y., Chen N., Chen K., Rao J., Ellenberg J. S. and Patz J. A., *Proc. Natl. Acad. Sci. U.S.A.*, **118** (2021) e2020524118.

[30] Zhang J., Litvinova M., Liang Y., Wang Y., Wang W., Zhao S., Wu Q., Merler S., Viboud C., Vespignani A., Ajelli M. and Yu H., *Science*, **368** (2020) 1481.

[31] Moreno Y., Nekovee M. and Pacheco A. F., *Phys. Rev. E*, **69** (2004) 066130.

[32] Citron D. T., Guerra C. A., Dolgert A. J., Wu S. L., Henry J. M., Sanchez H. M. C. and Smith D. L., *Proc. Natl. Acad. Sci. U.S.A.*, **118** (2021) e2007488118.

[33] Feng L., Zhao Q. and Zhou C., *Phys. Rev. E*, **102** (2020) 022306.

[34] Xu X.-K., Liu X.F., Wang L., Wu Y., Lu X., Wang X. and Pei S., *Fundamental Res.*, **3** (2023) 305.

[35] Pastor-Satorras R., Castellano C., Van Mieghem P. and Vespignani A., *Rev. Mod. Phys.*, **87** (2015) 925.

[36] Balcan D., Colizza V., Gonçalves B., Hu H., Ramasco J. J. and Vespignani A., *Proc. Natl. Acad. Sci. U.S.A.*, **106** (2009) 21484.

[37] Pei S., Teng X., Lewis P. and Shaman J., *Nat. Commun.*, **12** (2021) 222.

[38] Longini I. M., Nizam A., Xu S. F., Ungchusak K., Hanshaoworakul W., Cummings D. A. T. and Halloran M. E., *Science*, **309** (2005) 1083.

[39] Zhu Z., Chen B., Chen H., Qiu S., Fan C., Zhao Y., Guo R., Ai C., Liu Z., Zhao Z., Fang L. and Lu X., *The Innovation*, **3** (2022) 100274.

[40] Zhao Y., Zhu Z., Chen B., Qiu S., Huang J., Lu X., Yang W., Ai C., Huang K., He C., Jin Y., Liu Z. and Wang F.-Y., *The Innovation*, **4** (2023) 100521.

[41] Min B., Goh K.-I. and Vazquez A., *Phys. Rev. E*, **83** (2011) 036102.

[42] Xu T., Gao J., Coco G. and Wang S., *Int. J. Geogr. Inf. Sci.*, **34** (2020) 2136.

[43] Zhang W., Valencia A. and Chang N.-B., *IEEE Trans. Neural Netw. Learn. Syst.*, **34** (2023) 2170.

[44] Carella G., Trufero J. P., Álvarez M., and Mateu J., *Am. Statistician*, **76** (2022) 64.

[45] da Silva T. T., Francisquini R. and Nascimento M. C. V., *Expert Syst. Appl.*, **182** (2021) 115190.

[46] Liu D., Ding W., Dong Z. S. and Pedrycz W., *Comput. Ind. Eng.*, **166** (2022) 107970.

[47] Liu Y., Rong Y., Guo Z., Chen N., Xu T., Tsung F., and Li J., *Proc. AAAI Conference Artif. Intell.*, **37** (2023) 14347.

[48] Bhouri M. A., Costabal F. S., Wang H., Linka K., Peirlinck M., Kuhl E., and Perdikaris P., *Comput. Methods Appl. Mech. Eng.*, **382** (2021) 113891.

[49] Zhan C., Zheng Y., Lai Z., Hao T. and Li B., *Neural Comput. Appl.*, **33** (2021) 4915.

[50] Chang X., Wu J., Yu J., Liu T., Yan X. and Lee D.-H., *Transp. Res. A: Policy Practice*, **180** (2024) 103973.