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Memory management principle for dynamic isolation in agent-based epidemic modeling

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ABSTRACT

This paper presents a new epidemiological modeling approach that adapts the working set (WS) concept from computer memory management to the dynamics of infectious diseases. Traditional compartmental models provide valuable insights but are limited in their ability to capture dynamic isolation and heterogeneous contact patterns. In contrast, the WS model conceptualizes a time-varying subset of agents actively participating in social interactions, allowing for dynamic adjustments to the rate of infection and the explicit identification of superspreaders. By incorporating isolation states for both susceptible and infected individuals, the model more realistically captures quarantine and targeted interventions. Including an incubation period reduces epidemic peaks by nearly 40% and delays them by more than three weeks, providing critical time for public health response. Within the WS model, moderate isolation reduces peak infection rates by more than three times compared to uncontrolled scenarios, while high isolation almost completely prevents large-scale spread. These results highlight the model's ability to estimate the intensity and timing of interventions with greater accuracy than traditional models. By integrating the time window parameter and computer resource management principles, the adapted WS model represents a robust and adaptable tool for analyzing epidemic dynamics. The results highlight its potential for advancing epidemic modeling and supporting real-time public health decision-making.

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1. INTRODUCTION

Epidemic modeling has become one of the important tools for understanding, predicting and controlling the spread of infectious diseases. By transforming biological and social processes into mathematical and computational models, researchers can simulate the course of an outbreak, estimate key parameters such as the basic reproduction number, and evaluate the effectiveness of public health measures [1], [2]. The importance of such frameworks has been highlighted by global health crises, including the COVID-19 pandemic, where timely forecasts have guided critical decisions on quarantines, vaccinations, mobility restrictions, and social distancing policies [3], [4].

Mathematical approaches, from simple deterministic compartmental susceptible–infectious–recovered/removed (SIR) and exposed–(SEIR) models to advanced stochastic and network formulations, have provided valuable insights into transmission dynamics [5], [6]. Computational methods, including agent-based modeling and machine learning approaches, have further facilitated the integration of

heterogeneous population structures, contact networks, and real-time data flows. Collectively, these approaches highlight the interdisciplinary nature of epidemic modeling, where epidemiology, mathematics, computer science, and public health come together to support evidence-based decision-making [7]-[9].

Classic compartmental models such as SIR and its extensions SEIR and SEIR-V remain widely used in epidemiology [10]-[12]. They divide populations into compartments based on disease status and apply systems of differential equations to describe transitions. The SIR model by Kermack and McKendrick [13] laid the foundation for modern epidemic theory by segmenting populations into susceptible, infected, and recovered groups. Although mathematically elegant and analytically explicable, it makes simplifying assumptions such as homogeneous mixing and instantaneous infectivity that limit its ability to capture real epidemic behavior [14].

The SEIR model enhances realism by adding an exposed compartment to represent incubation [15], while SEIR-V takes vaccination into account [16], [17]. These extensions are more representative of biological processes, but they also increase complexity and remain limited by the assumption of a well-mixed population, making it difficult to model time-varying interventions such as isolation or quarantine.

Such interventions are dynamic: people are tested, isolated, released as new data arrives, and superspreaders heavily shape epidemic curves. Traditional compartmental models struggle to capture this adaptive isolation, often requiring additional compartments that complicate calibration and interpretation [18]-[21]. This highlights the urgent need for models that can naturally capture dynamic isolation, heterogeneous contact structures, and targeted interventions within epidemic dynamics.

The working set (WS) model, originally developed in computer science, provides a useful analogy for epidemic dynamics. In computing, the idea is simple: a program does not use all of its memory at once - it actively relies on a subset of memory pages at any given time, called the "Working Set." Pages not recently used are temporarily set aside to keep the system efficient [22], [23]. In epidemiology, a similar principle applies: at any moment, only a subset of individuals is actively engaged in transmitting infection, while others may be isolated or not participating in contact networks.

Just as operating systems dynamically adjust resources by adding or removing memory pages, epidemic control measures such as quarantine and isolation dynamically remove or reintegrate people from the pool of active contacts. This makes the WS concept especially suitable for modeling real epidemics, where the size and composition of the active contact set constantly change. To address this gap, we adapt the WS model to epidemiology.

In our formulation, the "Working Set" represents the subset of individuals involved in potential transmission. Infectious agents can be dynamically removed to represent isolation, while recovered or susceptible individuals may rejoin after a defined period. This allows the model to reflect heterogeneity in contact structures, capture the influence of superspreaders [24], and adjust transmission rates in real time. Unlike classical compartmental models the adaptive WS model makes dynamic isolation a central mechanism, offering a more flexible and realistic framework for analyzing epidemic control strategies under real-world conditions.

2. METHOD

We now examine some key assumptions of the adapted WS model in epidemiology. Key elements of the original WS model are redefined as follows: population is the complete set of agents, analogous to the set of all memory pages in a computer model; *WS* is a subset of the population that includes agents that are not currently isolated and may be involved in transmission (susceptible and infected); Isolation is the process of excluding agents from the WS, and equivalent to unloading pages from RAM. Isolated agents are temporarily not involved in the spread of infection; superspreader is an infected agent (in state I) that transmits infection to an unusually large number of susceptible agents (state S). Unlike the average infected agent, a superspreader causes significantly more infections due to high contact frequency or other factors.

The adapted model introduces the following states that reflect the epidemiologic status of the agents: *Susceptible* (S) is agents that are not in isolation and can become infected through contact with infected agents; *Infected* (I) is agents, not in isolation, capable of transmitting infection to others; *Recovered* (R) is agents who have developed immunity and are no longer involved in transmission, and *Quarantined* (Q) is agents who may be both susceptible and infected, but are temporarily excluded from transmission because of isolation. The adaptive WS model is described by a system of ordinary differential equations (ODEs):

$$\begin{cases} \frac{dS}{dt} = -\beta(t)\frac{SI}{N_{WS}} - \delta_S S + \eta_S Q_S \\ \frac{dI}{dt} = \beta(t)\frac{SI}{N_{WS}} - \gamma I - \delta_I I \\ \frac{dR}{dt} = \gamma I + \eta_I Q_R \\ \frac{dQ_S}{dt} = \delta_S S - \eta_S Q_S \\ \frac{dQ_I}{dt} = \delta_I I - \eta_S Q_I - \gamma Q_I \\ \frac{dQ_R}{dt} = \gamma Q_I - \eta_I Q_R \end{cases} \quad (1)$$

where, Q_S is isolated susceptible; Q_I is isolated infected; Q_R is isolated recovered (transferred from Q_I after recovery); $\beta(t)$ is dynamic infection rate; N_{WS} is current WS size (sum of agents in states S and I) at time t; δ_S is isolation for S; δ_I is isolation for I; η_S is isolation escape velocity for S; η_I is isolation escape velocity for I; and γ is rate of recovery.

This system accounts for all key processes: infection, recovery, isolation and release. The total population in the model is defined as follows: $N = S(t) + I(t) + R(t) + Q_S(t) + Q_I(t) + Q_R(t)$. The size of the WS is determined by the formula: $N_{WS}(t) = S(t) + I(t)$.

The dynamics of infection spread in the model is determined by the following processes:

a. Infection: transition of agents from state S to I by contact with infected agents. The speed of this process depends on the frequency of contact and the probability of transmission: $\beta(t) = \frac{S(t)I(t)}{N_{WS}(t)}$.
b. Recovery: transition from I to R as infected individuals recover. Rate of transition from I to R: $\gamma I(t)$.
c. Isolation*:* the transfer of agents from S or I to Q as a result of control measures such as contact tracing or quarantine. Then the coefficient from S to Q_S will be: $\delta_S S(t)$ and from I to Q_I will be: $\delta_I I(t)$.
d. Release from isolation*:* return of agents from Q_S to S (if they remain susceptible) or to R (if recovered) after completion of the isolation period or confirmation of status by testing: $\eta_S Q_S(t)$ and from Q_I to Q_R (recovery in isolation): $\gamma Q_I(t)$. From Q_R to R will be*:* $\eta_I Q_R(t)$.

In contrast to traditional models such as SIR, where the infection rate β is assumed to be constant and the population is assumed to be homogeneously mixed, in the adapted WS model the value of β becomes a dynamic variable depending on the size of the WS:

$$\beta(t) = \beta_0 \times \frac{N_{WS}(t)}{N} \quad (2)$$

where, β_0 is basic transmission rate under full population conditions; $N_{WS}(t)$ is the current size of the WS (the sum of agents in states S and I) at time t; and N is the total population size. As the number of isolated agents (translated into Q) increases, the size of the WS decreases, which reduces $\beta(t)$ and slows the spread of infection. This approach allows us to model the effect of quarantine and other control measures on epidemic dynamics.

To summarize the effects of uneven transmission likelihoods, vulnerability distributions, and interaction patterns, we use a simple class of models in which the population is partitioned into multiple groups of agents. These adaptations aim to demonstrate that population diversity can significantly alter both the progression and total reach of an epidemic, and, critically, broaden the range of viable intervention strategies.

Let consider a multi-agent system with n agents distributed over p groups and exposed to the risk of infection through contact with each other. Let us specify that the agents' distribution into groups, and each agent group number can be easily determined by the matrix $x = (x_{ri})_{p\times n}$ as shown in Figure 1, where the element is $x_{ri} = 1$, if the agent with the number i is located in the group with the number r and $x_{ri} = 0$, otherwise.

The matrix x must satisfy constraints (a), (b), and (c). Whatever the distribution of agents over groups, we assume that each agent of the system belongs to only one of the groups (condition, (a)): $\sum_{r=1}^{p} x_{ri} = 1, i = 1,2, \dots, n$. Each agent of the system is assigned a "weight", the linear size of its living space, within which the agent can perform its set of operations assigned to it. In this case, the agents interacting with each other are exposed to infection risk through contact. Each group is also assigned a "weight" - living space within which the group's agents are located. The total "weight" of agents in any group should not exceed the weight of the group (condition (b)): $\sum_{i=1}^{n} l_i \cdot x_{ri} \le v_r, r = 1,2, \dots, p$. Here l_i is the weight of the agent i, $i = 1,2, \dots, n$, and v_r is the weight of the group with number r, r=1, 2, ..., p. Let us determine the number of a group that contains an agent, for example i, with a given matrix $x \in$ X, denoting this number by $r_i(x)$ and taking into account the constraints $(a), (b)$, we write (condition (c)): $r_i(x) = \sum_{r=1}^{p} x_{ri} \cdot r, i = 1,2, \dots, n$.

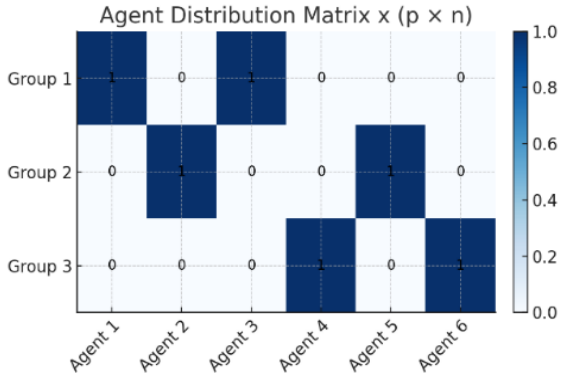

Figure 1. Matrix representation of agent distribution across groups

A WS in an epidemiological context is a dynamic group of agents that participate in social interactions and are not subject to isolation. Its size and composition depend on the following factors: Isolation policy: when an infected agent from I is identified, its contacts from S in the last τ days are relegated to the state Q. This shortens the WS and reduces the likelihood of new infections. At the end of the isolation period, agents from Q are tested: susceptible agents return to S, recovered agents to R. An alternative scenario is high-coverage isolation, in which a large fraction of the population is isolated. Time window (τ): similar to the original WS model, a parameter τ is introduced to define the period of "relevance" of contacts. Agents who have been in contact with infected individuals in the last τ time units are considered candidates for isolation. There may also be superspreaders among these agents. Their identification is important for epidemic control because isolation of such agents can significantly slow the spread of the disease. In the WS model, the τ parameter specifies the time window during which contacts are considered relevant.

To assess the dynamics of infection spread and evaluate the impact of isolation measures. For this purpose, we introduce experimental scenarios: 1. Basic scenario: no isolation ($\delta_S = 0$, $\delta_I = 0$); 2. Moderate isolation: low isolation parameters ($\delta_S = 0.08$, $\delta_I = 0.9$); 3. High-coverage isolation: high isolation parameters ($\delta_S = 0.3$, $\delta_I = 0.4$). The SIR and SEIR models do not take insulation into account, so only the basic scenario is considered. Table 1 summarizes the parameter values and their descriptions used in the numerical simulations. The parameters were derived from an extensive literature review [25]-[27] on COVID-19 and epidemic modeling. As in agent-based models [28], our model is formulated for a generalized small-city population. This abstraction allows flexible adaptation while avoiding the need for detailed prior knowledge of region-specific parameters

Table 1. Model parameters and descriptions

Variable	Default value	Explanation
N	100,000	Total number of individuals in the population
S_0	98 000	Initial number of susceptible individuals
I_0	2 000	Initial number of infected individuals
R_0	0	Initial number of recovered individuals
E_0	0	Initial number of exposed individuals (for SEIR model)
Q_{S_0}	0	Initial number of quarantined susceptible individuals
Q_{I_0}	0	Initial number of quarantined infected individuals
Q_{R_0}	0	Initial number of quarantined recovered individuals
β	0.4	Infection rate; probability of disease transmission per contact between susceptible and infected individuals
β_0	0.4	Base infection rate for the WS
σ	0.3	Incubation rate; rate at which exposed individuals become infectious (for SEIR model)
γ	0.2	Recovery rate; proportion of infected individuals recovering per unit time
η_S	0.2	Quarantine release rate for susceptible individuals; proportion released per unit time
η_I	0.2	Quarantine release rate for infected individuals; proportion released per unit time

3. RESULTS AND DISCUSSION

Figure 2 illustrates the epidemic dynamics and the impact of isolation across classical SIR, SEIR, and adaptive WS models. Figure 2(a) shows reflect the comparative epidemic dynamics in the SIR, SEIR,

and WS models. In both the SIR and WS scenarios, the infection curve peaks around day 25, reaching approximately 31,490 individuals, or 31.5% of the total population, after which infections decline sharply. The SEIR model, by contrast, produces a substantially lower peak of about 19,222 individuals, or 19.2%, which occurs later on day 52. These results demonstrate that incorporating an incubation period reduces the epidemic peak by nearly 40% and delays it by more than three weeks, offering critical additional time for healthcare response.

Figure 2(b) demonstrates the influence of isolation measures on epidemic outcomes within the WS framework. In the absence of isolation, the infection curve peaks at 35,564 cases, or 35.6% of the population, on day 24. Under moderate isolation ($\delta_S = 0.025$ and $\delta_I = 0.06$), the peak is reduced to 10,843 cases, or 10.8% of the population, and is shifted to day 27, representing a reduction of more than threefold compared to the uncontrolled scenario. With high isolation ($\delta_S = 0.2$ and $\delta_I = 0.3$), the curve is nearly suppressed, with infections never exceeding 2% of the population before rapidly declining. These findings confirm that even moderate interventions markedly reduce epidemic intensity, while strict measures can almost completely prevent large-scale spread.

Figure 2(c) highlights the differences in epidemic curves when measuring daily incidence. The SIR model reaches a peak of approximately 3,950 new infections per day, or 3.95% of the population, on day 22. The WS (no isolation) trajectory is nearly identical, with the maximum slightly higher at around 4,450 daily cases, or 4.45% of the population, on days 22-23, confirming the equivalence of the two models in the absence of interventions. The SEIR model, in contrast, produces a much lower peak of about 1,950 new infections per day, or 1.95%, and this occurs considerably later, on days 43-44. The inclusion of an incubation period, therefore, reduces the intensity of daily spread by almost half and delays the peak by approximately three weeks, creating a vital buffer for organizational and medical response. The main characteristics of the traditional SIR/SEIR models and the proposed WS framework are comparatively summarized in Table 2 to emphasize the advantages of the WS approach.

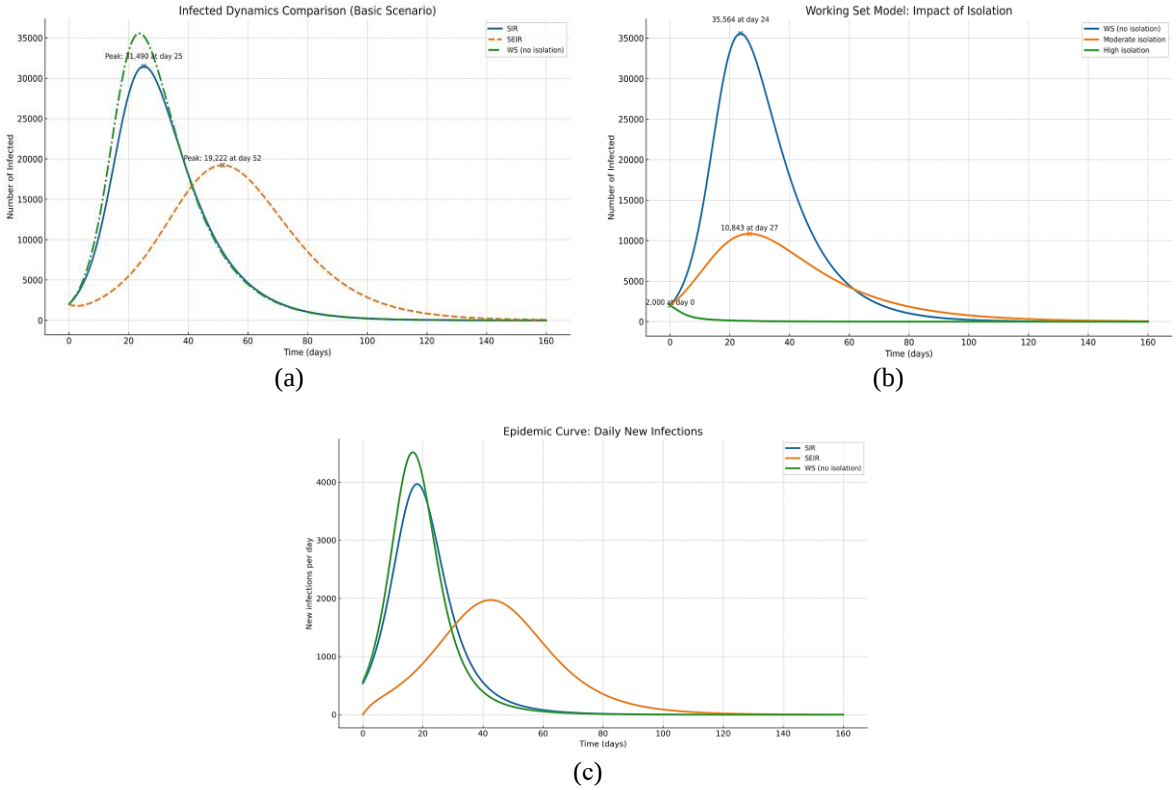

Figure 2. Epidemic dynamics and the impact of isolation in SIR, SEIR, and WS models; (a) comparative epidemic dynamics, (b) effect of isolation measures in WS model, and (c) daily incidence curves across models

Comparing the SIR, SEIR, and WS models, we may say that the WS model offers flexibility due to its isolation, making it more realistic for modeling control measures. The proposed WS model offers several

advantages, such as accounting for contact heterogeneity and the ability to quantify the impact of quarantine, contact tracing, and other strategies. In addition, like memory management in computer science, the model allows to explore the effectiveness of epidemic control. These analyses demonstrate how the adapted WS model can be useful for studying epidemic dynamics, providing valuable insights for infectious disease management. Further study of the model could be useful for public health planning and the evaluation of measures such as quarantine and social distancing.

Table 2. Comparisons of the models

Aspect	SIR/SEIR	WS
Isolation and quarantine	Not directly accounted for, expansion required	Included as centerpiece, dynamic adjustment
Transmission speed	Fixed or dependent on *S* and *I*	Dynamically adjusted based on active set
Contact heterogeneity	Requires extensions (e.g., network)	Accounting through groups and subsets
Behavioral solutions	Not modelled	May be enabled via agent rules
Applicability for interventions	Limited without modifications	Easy to model quarantine

4. CONCLUSION

The proposed WS model, adapted to the epidemiological context, represents a new and flexible approach to modeling the spread of infectious diseases. Unlike classical SIR and SEIR models, it uses dynamic containment as a central mechanism, enabling the identification of active subgroups of individuals involved in infection transmission and highlighting the role of superspreaders in shaping epidemic curves. By adjusting the effective transmission rate based on the size and composition of the group of active contacts, the model provides a more realistic representation of how interventions influence epidemic dynamics over time. In addition to its theoretical contributions, the WS model has clear practical applications. Public health officials and policymakers can use it as a decision support tool to evaluate quarantine measures, testing strategies, or phased release policies in real time. The model's ability to simulate dynamic containment scenarios makes it particularly relevant in rapidly changing epidemic situations, where timely adjustments to interventions are critical to mitigating peak incidence and reducing the burden on healthcare systems. Although the WS model requires detailed data and careful parameter calibration, its integration of principles from computer science into epidemiology opens new opportunities for designing optimal control strategies. Future research may be focused on extending the WS model through integration with agent-based simulations, which can capture individual-level heterogeneity and social network structures, which can provide real-time information on interaction patterns. These developments will help validate and refine the WS framework, ultimately transforming it into a practical tool for real-time epidemic response planning and strengthening preparedness for future public health crises.

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AUTHOR CONTRIBUTIONS STATEMENT

This journal uses the Contributor Roles Taxonomy (CRediT) to recognize individual author contributions, reduce authorship disputes, and facilitate collaboration.

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C : **C**onceptualization
M : **M**ethodology
So : **So**ftware
Va : **Va**lidation
Fo : **Fo**rmal analysis
I : **I**nvestigation
R : **R**esources
D : **D**ata Curation
O : Writing - **O**riginal Draft
E : Writing - Review & **E**diting
Vi : **Vi**sualization
Su : **Su**pervision
P : **P**roject administration
Fu : **Fu**nding acquisition

CONFLICT OF INTEREST STATEMENT

Authors state no conflict of interest.

DATA AVAILABILITY

The data that support the findings of this study are available from the corresponding author, [AM], upon reasonable request.

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