КОМПЬЮТЕРНОЕ МОДЕЛИРОВАНИЕ
COMPUTER SIMULATION

UDC 517.958 **DOI: 10.18413/2518-1092-2025-10-3-0-6**

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SEIRD EPIDEMIOLOGICAL MODELS FOR PLANT DISEASE

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Abstract
This paper explores the potential application of the SEIRD epidemiological model – commonly used in human disease modeling – to the context of plant disease outbreaks. Plant pathogens pose a significant threat to agricultural productivity, necessitating robust quantitative tools for understanding their dynamics and guiding control strategies. The SEIRD model was adapted to plant pathology by incorporating key agricultural variables, including environmental factors (e.g., humidity and temperature), plant growth stages, and the impact of interventions such as chemical treatments and removal of infected plants. Spatial dynamics were also modeled using traveling wave formulations. Results indicate that the modified model effectively captures the temporal and spatial progression of plant epidemics, enabling prediction of outbreak peaks and evaluation of control measures. This study presents a flexible mathematical framework that can be extended to various plant diseases, providing a valuable tool for data-driven decision-making in smart agriculture and epidemic risk management.
Keywords: SEIRD model; plant disease epidemiology; mathematical modeling; environmental factors; agricultural disease management
For citation: Konstantinov I.S., Taha A.T.T., Goldobina D.M. SEIRD Epidemiological Models for Plant Disease // Research result. Information technologies. – T.10, №3, 2025. – P. 64-71. DOI: 10.18413/2518-1092-2025-10-3-0-6

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ЭПИДЕМИОЛОГИЧЕСКИЕ МОДЕЛИ SEIRD ДЛЯ БОЛЕЗНЕЙ РАСТЕНИЙ

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Аннотация
В данной статье рассматривается потенциальное применение эпидемиологической модели SEIRD, широко используемой в моделировании заболеваний человека, к контексту вспышек болезней растений. Фитопатогены представляют серьёзную угрозу для сельскохозяйственного производства, что требует использования надёжных количественных инструментов для понимания динамики их распространения и разработки эффективных стратегий контроля. Модель SEIRD была адаптирована для фитопатологии путём включения ключевых агрономических переменных, таких как экологические факторы

(например, влажность и температура), фазы роста растений и влияние агротехнических мероприятий, включая химическую обработку и удаление инфицированных растений. Пространственная динамика была смоделирована с использованием уравнений бегущей волны. Результаты показывают, что модифицированная модель эффективно отражает временное и пространственное развитие эпидемий среди растений, позволяя прогнозировать пики заболеваемости и оценивать эффективность мер контроля. Исследование представляет собой гибкую математическую основу, которую можно адаптировать для различных фитопатологий, что делает её ценным инструментом для принятия решений на основе данных в условиях «умного» сельского хозяйства и управления эпидемиологическими рисками.

Ключевые слова: модель SEIRD; эпидемиология болезней растений; математическое моделирование; экологические факторы; управление болезнями в сельском хозяйстве

Для цитирования: Константинов И.С., Таха А.Т.Т., Голдобина Д.М. Эпидемиологические модели SEIRD для болезней растений // Научный результат. Информационные технологии. – Т.10, №3, 2025. – С. 64-71. DOI: 10.18413/2518-1092-2025-10-3-0-6

INTRODUCTION

Mathematical modeling has emerged as a fundamental tool in the study of complex biological systems, particularly in understanding the mechanisms underlying the spread of infectious diseases. By providing a quantitative framework, mathematical models enable researchers to simulate disease dynamics, predict future outbreaks, and evaluate the potential outcomes of various intervention strategies. Among the multitude of models developed in the field of epidemiology, the SIR model stands out as one of the most classical and widely adopted approaches. Originally designed for human and animal populations, this model captures the core dynamics of infectious disease transmission through the interaction of three primary compartments: susceptible (S), infected (I), and removed (R). In this study, we explore the potential of adapting the SIR model, and its extended forms such as SEIRD, to the domain of plant epidemiology.

The basic structure of the SIR model is built upon a system of ordinary differential equations that describe the rate of change of each compartment over time. [1-2] The model assumes a closed population with no births or natural deaths unrelated to the disease. The susceptible compartment, denoted as S(t), represents individuals – or in our context, plants – that are vulnerable to infection. The infected compartment, I(t), includes those currently harboring the pathogen and capable of transmitting it. The removed compartment, R(t), consists of plants that have either recovered, died, or been culled to prevent further spread. The interactions among these compartments are governed by the following equations:

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$$\begin{aligned} \frac{dS}{dt} &= -\beta\, S\, I, \\ \frac{dI}{dt} &= \beta\, S\, I - \alpha\, I, \\ \frac{dR}{dt} &= \alpha\, I. \end{aligned} \tag{1}$$

Here, β is the transmission rate, reflecting the probability of disease spread from an infected plant to a susceptible one. The parameter α denotes the removal rate, which can include recovery, death, or agricultural interventions such as the removal of infected plants. These equations form the backbone of many epidemiological analyses, offering valuable insight into the trajectory of an outbreak under various conditions.

When applying this model to plant disease epidemics, however, several critical modifications are necessary to capture the unique characteristics of plant-pathogen interactions and the spatial and environmental heterogeneity inherent in agricultural systems. One of the key limitations of the classical SIR model is its assumption of a homogeneously mixed population, which rarely holds true in agricultural fields where plants are organized in spatial patterns. To address this, spatial dynamics can be incorporated into the model by transforming the system into a partial differential equation (PDE) framework [3-5]. For example, the infected compartment equation may be rewritten to include a diffusion term:

$$\partial I/\partial t = \beta S I - \alpha I + D\,\partial^2 I/\partial x^2 . \quad (2)$$

In this extended form, D represents the diffusion coefficient, which quantifies the spatial spread of the pathogen, potentially influenced by vectors such as wind, water, or insects. This modification allows the model to simulate the movement of the disease front across a field, capturing the spatial propagation of the epidemic.

Another important refinement involves the variability of the transmission rate β\beta. Unlike human populations, where interaction patterns can be relatively stable, plant environments are subject to fluctuating external conditions such as temperature, humidity, and the presence of disease vectors. As such, β can be modeled as a function of both space and time: $\beta = \beta(x, t)$. This dynamic approach enables a more realistic representation of disease transmission, accounting for daily or seasonal changes in environmental conditions.

Depending on the nature of the plant disease being studied, it may be necessary to extend the SIR model to include additional compartments. For instance, many pathogens exhibit a latent period during which an infected plant is not yet infectious. This necessitates the incorporation of an exposed compartment (E), leading to the SEIR model. In some cases, the inclusion of a deceased compartment (D) results in the SEIRD model, providing a more nuanced understanding of disease outcomes. These extensions help to capture the biological complexity of plant-pathogen interactions more effectively. In addition to these structural enhancements, the concept of traveling waves offers a powerful tool for analyzing the spatio-temporal spread of plant diseases. A traveling wave solution assumes that the disease front moves through the field at a constant speed cc, and introduces a new variable $\xi = x - ct$. This transformation simplifies the PDEs and facilitates analytical and numerical exploration of how rapidly and in what manner the disease propagates spatially.

While these modifications significantly enhance the model's applicability to plant epidemiology, they also introduce new challenges. Chief among them is the requirement for accurate and high-resolution field data to estimate model parameters such as β\beta, α\alpha, and D. Moreover, solving PDE-based models analytically is often intractable, necessitating the use of numerical simulations and computational tools. Environmental variability and heterogeneity in field conditions further complicate parameter estimation and model calibration [6].

Despite these challenges, the integration of spatial dynamics, time-varying transmission rates, and additional epidemiological compartments renders the SEIRD framework a powerful and adaptable tool for modeling plant disease outbreaks. By capturing the essential biological and environmental factors that drive disease dynamics in agricultural systems, the SEIRD model not only aids in understanding the underlying processes but also provides a quantitative foundation for designing and evaluating intervention strategies. This study aims to demonstrate the potential of this model in informing data-driven decision-making in plant disease management and contributing to the advancement of precision agriculture.

MODIFICATION OF SEIRD MODEL TO BE IMPLEMENTED TO PLANT DISEASE

The authors in [7-11] developed a mathematical model to simulate epidemiological phenomena, particularly focusing on the dynamics of infectious diseases. A well-established framework was constructed and thoroughly analyzed to capture the essential components of disease transmission and control. The

SEIRD model is a well-established mathematical framework used to simulate and analyze the spread of infectious diseases in human populations. Given the biological similarities in the mechanisms of disease propagation, the SEIRD model can be adapted to understand the dynamics of plant disease epidemics. This report outlines how the SEIRD model can be applied to plant pathology with appropriate modifications.

In the context of plant diseases, the SEIRD model compartments can be reinterpreted as follows:

- S (Susceptible): Healthy plants that are vulnerable to infection.
- E (Exposed): Plants that have been infected but are not yet infectious (latent period).
- I (Infectious): Infected plants capable of transmitting the disease.
- R (Recovered): Plants that have recovered or are resistant to reinfection.
- D (Dead): Plants that have died or been removed due to the disease.

The differential equations governing the SEIRD model in plant pathology are similar to the human model but must consider plant-specific parameters:

$$\frac{dS}{dt} = -\beta * S * I,$$

$$\frac{dE}{dt} = \beta * S * I - \sigma * E,$$

$$\frac{dI}{dt} = \sigma * E - \gamma * I - \mu * I, \quad (3)$$

$$\frac{dR}{dt} = \gamma * I,$$

$$\frac{dD}{dt} = \mu * I.$$

Where:

β: Transmission rate influenced by planting density, vector presence, and environmental conditions.

σ: Rate of progression from exposed to infectious.

γ: Recovery rate (e.g., due to resistance or treatment).

μ: Mortality or removal rate due to disease.

In adapting the SEIRD model to accurately represent plant disease epidemics, one of the most essential modifications involves the incorporation of environmental factors – chiefly temperature and humidity – into the core dynamics of disease transmission. Unlike human epidemiology, where contact patterns and behavior largely govern transmission, the progression and intensity of plant epidemics are heavily influenced by abiotic environmental conditions. Temperature and relative humidity, in particular, play a critical role in determining the rate at which plant pathogens infect hosts, survive in the environment, and reproduce. These variables fluctuate daily and seasonally, thereby making it necessary to define the transmission rate $beta(t)$ as a dynamic function that responds to environmental inputs.

To integrate this variability, the transmission rate can be modeled as:

$$\beta(t) = \beta_0 \times f\big(T(t), H(t)\big). \quad (4)$$

Here, β_0 denotes the baseline transmission rate under optimal conditions, while $T(t)$and $H(t)$ represent the temperature and relative humidity at time t, respectively. The function $f(T(t), H(t)$ acts as an environmental response function, modifying the effective transmission rate based on how closely current environmental conditions align with the optimal thresholds for pathogen activity. A commonly used formulation for $f(T(t), H(t)$involves a product of two sigmoid functions:

$$f(T,H) = \frac{1}{1+e^{-k1(T-Topt)}} \times \frac{1}{1+e^{-k2(H-Hopt)}}. \quad (5)$$

In this expression, $Topt$ and $Hopt$ represent the optimal temperature and humidity conditions for disease transmission, while $k1$ and $k2$ determine the steepness of the response – how sensitive the pathogen is to deviations from its optimal climate. When temperature and humidity are near their respective optima, *f*(*T*,*H*) approaches 1, maximizing transmission. Conversely, under unfavorable conditions, the function declines, reducing β(t) and thus lowering the likelihood of infection. Beyond environmental responsiveness, the SEIRD model can also be improved by accounting for seasonal trends and plant growth stages. Susceptibility to infection often varies across the plant's development cycle – seedlings may be more vulnerable than mature plants, for instance. A time-dependent growth stage multiplier $G(t)$ can be introduced to scale β(t) in accordance with the current phase of plant development. For example, G(t) might take values like 0.3 during the seedling stage, 0.6 during vegetative growth, and 1.0 during flowering, when susceptibility peaks. Another vital consideration is the effect of agricultural interventions – notably fungicide application, crop rotation, and physical removal of infected plants. These interventions can be incorporated into the SEIRD model by further modifying β(t) through a treatment decay function M(t). Immediately after a fungicide is applied, M(t) = 1, indicating full effect; over time, its impact decays, Accordingly, the effective transmission rate becomes:

$$\beta(t) = \beta_0 \times f(T(t).H(t)) \times G(t) \times e^{-\lambda(t-t_0)} \quad (6)$$

Where λ is the rate of decay and t_0 is the time of treatment. In some scenarios, interventions also enhance the recovery rate $\gamma(t)$, for instance, by accelerating the healing process or removing infected plants from the population. Altogether, these adaptations – environmental modulation, growth-stage sensitivity, and intervention effects – equip the SEIRD model with the structural flexibility needed to accurately simulate real-world plant epidemics. They enable the model to not only mirror the biological realities of plant-pathogen-environment interactions but also to evaluate the effectiveness of management strategies under changing conditions. These enhancements make the model an effective decision-support tool for agricultural disease control, particularly in the context of climate variability and precision agriculture

Plant susceptibility to disease is not static; it fluctuates in response to both seasonal environmental cycles and distinct physiological growth stages. For instance, plants are generally more vulnerable during early developmental phases such as the seedling stage, while resistance tends to increase as they mature. To mathematically capture the effect of seasonal patterns on disease transmission, a periodic adjustment function g(t) can be introduced:

$$g(t) = 1 + \alpha \cdot sin\left(\frac{2\pi t}{365} + \phi\right) \quad (7)$$

In this equation, α\alpha represents the amplitude of seasonal variation, indicating the intensity of fluctuation, while ϕ denotes the phase shift used to align the seasonal peak with the critical infection period (e.g., spring or harvest time).

To further refine the model, a growth-stage sensitivity coefficient $G(t)$ is applied to account for the plant's developmental phase. This coefficient can take discrete values based on observed susceptibility: for example, $G(t) = 0.3$ for seedlings, *G*(*t*)=0.6 during vegetative growth, and *G*(*t*)=1.0 during flowering or fruiting stages.

Combining both seasonal and phenological influences, the time-dependent transmission rate becomes:

$$\beta(t) = \beta 0 \cdot g(t) \cdot G(t). \quad (8)$$

This composite function allows the model to reflect real-world variations in disease transmission potential driven by environmental seasonality and plant development.

Agricultural actions, such as chemical treatments or removal of infected plants, significantly affect epidemic dynamics and should be modeled explicitly.

The transmission rate can be reduced after treatment:

$$\beta(t) = \beta 0 \cdot e^{-\delta \cdot M(t)}. \quad (9)$$

Where:

δ: Reduction factor due to treatment

M(t): Treatment effectiveness function, such as:

- M(t) = 1 (immediately after application);
- $M(t) = e^{\left(-lambda\,(t - t^0)\right)}$ (decays over time since treatment at t_0).

Similarly, the recovery rate γ(t) can be increased due to intervention:

$$\gamma(t) = \gamma 0 + \eta \cdot M(t) \quad (10)$$

FINAL MODIFIED SEIRD EQUATIONS

$$\begin{aligned} dS/dt &= -\beta(t) \cdot S \cdot I/N \\ dE/dt &= \beta(t) \cdot S \cdot I/N - \sigma E \\ dI/dt &= \sigma E - \gamma(t) I - \mu I \\ dR/dt &= \gamma(t) I \\ dD/dt &= \mu I \end{aligned} \quad (11)$$

APPLICATIONS IN PLANT DISEASE MANAGEMENT

The modified SEIRD model offers a powerful and versatile framework for managing plant disease outbreaks in agricultural systems. One of its key applications lies in its ability to simulate both the spatial and temporal dynamics of pathogen spread. By incorporating environmental conditions, plant development stages, and intervention effects, the model can realistically reflect how diseases propagate through a crop field over time and space. This capability allows researchers and agricultural decision-makers to visualize potential epidemic trajectories and understand how fast and in what direction an outbreak may unfold. Additionally, the model provides a structured approach for assessing the impact of various control strategies, such as fungicide applications, crop rotation, or the removal of infected plants. By comparing different intervention scenarios, the SEIRD framework enables users to identify the most effective and cost-efficient disease management practices.

Furthermore, the model can forecast the timing and intensity of epidemic peaks, which is crucial for optimizing the timing of interventions and minimizing crop loss. Finally, the model contributes to broader integrated pest and disease management (IPDM) efforts by offering a data-driven foundation for coordinating multiple control measures in a holistic and sustainable manner. Through its predictive capacity and flexibility, the SEIRD model serves as an essential tool in modern, precision-guided plant disease control.

CONCLUSION

The SEIRD model, when appropriately modified, offers a robust and flexible framework for modeling the dynamics of plant disease epidemics. By incorporating critical agricultural factors – such as environmental conditions (e.g., temperature and humidity), seasonal variation, plant growth stages, and the decaying effects of control interventions – the model becomes highly relevant for real-world agricultural scenarios. These modifications enhance the model's ability to simulate spatial and temporal disease progression, estimate outbreak peaks, and assess the effectiveness of various management strategies. Furthermore, the inclusion of traveling wave analysis and time-varying transmission parameters allows for more accurate prediction of disease spread in heterogeneous crop environments. As a result, the adapted SEIRD model serves as a valuable decision-support tool in integrated pest and disease management

programs. It not only facilitates data-driven planning and timely intervention but also contributes to the development of precision agriculture by enabling more sustainable, efficient, and proactive responses to plant epidemics.

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