A Markov Chain Model for Predicting ETI Resistance Breakdown in Tomato–Pseudomonas syringae

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Agricultural pesticide use remains substantial, increasing chemical exposure, environmental burden, and production costs. U.S. farms spent $21.7 billion on agricultural chemicals in 2024. Better-timed interventions can reduce applications by 44–50% without compromising control, leading to cost savings. A key limitation is the lack of predictive tools to determine when host resistance remains effective and when pesticide use becomes biologically justified. This study presents the first in silico model designed to predict effector-triggered immunity (ETI) failure in the tomato–Pseudomonas syringae pathosystem by estimating stacked R-gene resistance breakdown timing. The model uses Markov chains with a mean-field approximation to simulate pathogen evolution under R-gene stacking. It incorporates three R-genes (Pto/Prf, Roq1, Ptr1) and four effectors (AvrPto, AvrPtoB, AvrRpt2, HopQ1). Pathogen states are encoded as bitstrings representing effector presence or absence, with transitions representing mutation and horizontal gene transfer (HGT). Effector-specific rates are parameterized, and the model is implemented in C++ for efficiency. Validation under 26–28 °C showed the model correctly reproduced (i) the inverse relationship between R-gene–effector overlap and pathogen growth, with long-run populations stabilizing at ~10^7 to 5×10^8 CFU/cm², (ii) the multi-season durability of Roq1-mediated resistance (>3 years), and (iii) short-term bacterial growth dynamics in susceptible tomato lines, predicting sharp growth to ~10^8 CFU/cm², matching the expected ~10^7–10^8 CFU/cm² range in susceptible tomato lines. These results support the model's realism and potential to guide R-gene stacking in crop protection programs, reducing unnecessary pesticide use.

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First Award of $6,000
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