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Quantifying Rainfall-Driven Splash Dispersal of Ascochyta rabiei in Chickpea: Experimental Evidence for Intensity Thresholds and Epidemiological Model Improvement

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Abstract. Ascochyta blight of chickpea is a major constraint in semi-arid rainfed systems, and epidemics are strongly driven by rainfall-mediated splash dispersal, yet most weather-based models still rely on an empirical 2 mm daily rainfall threshold and lack experimental quantification of the rainfall–lesion relationship and associated thresholds. To address this gap, a calibrated open-channel rainfall simulator was used in a greenhouse to impose six event rainfall levels from 0 to 8 mm per 10 minutes on potted chickpea plants supplied with residue inoculated by Ascochyta rabiei, generating 96 pot-level lesion counts. A negative binomial GLMM combined with segmented regression was then fitted to derive the rainfall–lesion response function and to estimate a breakpoint at an event rainfall of about 1.1 mm. Lesion numbers increased slowly with rainfall at low amounts but rose much more steeply once the threshold was exceeded. After normalisation, this function was incorporated into the ascotraceR model, and simulations for 4 regions and 28 site–years showed an increase in mean AUDPC R2R^2R2 from 0.65 to 0.81 and a reduction in root mean square error from 210 to 145 disease·day, with clear improvement in capturing the cumulative effects of repeated light rain events. The study provides quantitative threshold evidence for rain-splash dispersal of A. rabiei and illustrates a practical pathway in which physical experiment parameterisation is used to update computational epidemiological models and refine rainfall triggers and fungicide timing rules.

Keywords: chickpea, Ascochyta blight, rain splash, epidemiological modelling, ascotraceR

1. Introduction

Chickpea is a key crop for food security and smallholder livelihoods in semi-arid regions, yet production in Australia, South Asia and Mediterranean-type environments remains exposed to highly variable risk from Ascochyta blight [1]. The causal fungus Ascochyta rabiei uses infected seed and crop residues as primary inoculum and, under cool and rainy weather with moderate wind, relies on rain-splash dispersal to spread conidia over short distances within the canopy and to drive multiple cycles of secondary infection, which amplifies the consequences of early management decisions. In practice, current control strategies make heavy use of weather-driven forecasting models that define “risk windows” based on daily rainfall, rain duration and leaf wetness to schedule

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protective fungicide sprays, but most models still depend on empirical rainfall thresholds and splash parameters borrowed from other pathosystems, and therefore represent frequent light rain, cultivar architecture and canopy structure only in a coarse way [2]. Recent advances in field monitoring and mechanistic modelling show that repeated low-intensity rain events contribute substantially to early epidemic build-up, while model responses to these events are clearly delayed or underestimated, exposing a critical gap in experimental evidence for quantitative rainfall–lesion functions and biologically meaningful intensity thresholds under controlled conditions [3]. In this context, the present study builds a greenhouse rainfall simulator with multiple intensity levels to measure lesion formation by A. rabiei across a gradient of event rainfall and then parameterises the resulting rainfall response function into the ascotraceR model in order to assess how updated splash parameters affect the simulation of field disease progress curves and the guidance provided for management decisions.

2. Literature review

2.1. Epidemiology and impact of Ascochyta blight in chickpea

The epidemiology of Ascochyta blight can be viewed as a linked sequence of seed-borne infection, survival on crop residues, establishment of primary foci and multiple cycles of secondary spread, where the level of primary infection is constrained by residue load and seed health measures and the efficiency of secondary infection is shaped by the proportion of susceptible cultivars, canopy closure and rainfall patterns [4]. Monitoring studies in contrasting regions show that in cool and wet seasons the disease can rapidly escape a “low-level latent phase” and enter an explosive phase in which rain splash drives strong spatial gradients of lesion density along and across rows, leading to severe yield loss and grain quality downgrading, whereas in seasons with fragmented rainfall the epidemic often remains confined to small patches with slow progression [5].

2.2. Rain-splash dispersal mechanisms and rainfall intensity effects

The rain-splash dispersal process can be decomposed into several physical stages, including raindrop impact on inoculum-bearing surfaces, generation of primary and secondary splash droplets, three-dimensional flight and deposition of spore-laden droplets within the canopy and subsequent redistribution of intercepted liquid on receptor surfaces, and each of these stages is controlled by the drop size distribution, rainfall intensity, impact angle, and leaf shape and roughness [6]. Experimental fluid mechanics and rainfall-simulator studies show that under low-intensity conditions drops have limited kinetic energy and tend to generate relatively few, larger droplets with stable but short dispersal distances, whereas at medium and high intensities the high frequency of impacts greatly increases the number of splash droplets and their maximum height, raising infection probabilities in upper canopy layers and between rows and enabling multi-step dispersal chains through re-splash [7].

2.3. Weather-driven and computational epidemiological models

Weather-driven disease models aim to transform time series of rainfall, temperature, relative humidity and leaf wetness into infection events or risk indices, and their central challenge is to retain key biophysical processes at a simplified scale so that the models remain mechanistic enough while still being suitable for regional application and linkage to decision systems [8]. With the development of general epidemiological frameworks in open-source environments such as R, researchers can now combine stochastic simulation, parameter estimation and uncertainty analysis in a unified setting, creating more flexible computational laboratories for field diseases like Ascochyta blight [9]. However, without experimental data tailored to specific pathogen–host–environment combinations to calibrate response functions such as "rainfall–splash efficiency", even structurally rich models struggle to provide reliable predictions under frequent light rain, complex canopies and heterogeneous residue distributions, so systematically integrating physical experiment results into dedicated tools like ascotraceR becomes a pivotal step for improving both model credibility and decision-making value.

3. Experimental methods

3.1. Rain-splash simulator and experimental design

The experiment used a calibrated open-channel rainfall simulator in a greenhouse with a test area of 3.0 m × 0.9 m. Nozzle pressure was kept at 100 kPa, and boom swing speed plus nozzle configuration were adjusted to create six event rainfall levels: 0, 0.5, 1.0, 2.0, 4.0 and 8.0 mm per 10 minutes. For each intensity, 4 replicate plots were used, and each plot contained 4 pots with three-week-old chickpea plants, giving 96 pot-level observations in total. Before each rain event, collection trays with the same surface area as the pots were placed to check rainfall stability and uniformity [10]. For a given intensity, the coefficient of variation of event rainfall among pots was below 12%, and the absolute deviation between measured and target rainfall for each level did not exceed 0.2 mm. Rainfall events were applied when daily maximum temperature was 22–25 °C and relative humidity was 60–70%, so differences in raindrop kinetic energy mainly reflected changes in rainfall intensity rather than air properties.

3.2. Inoculum, treatments and lesion assessment

A widely grown commercial chickpea cultivar with susceptible but neutral agronomic characteristics was used. Plants were grown in 5 L plastic pots with 6 plants per pot, and seeds were treated with a mancozeb-based fungicide to remove seed-borne infection. A single-spore isolate of A. rabiei was cultured on oatmeal agar for 10 days, then plates were washed to prepare a conidial suspension, and the concentration was adjusted to 1×10^6 conidia/mL. The suspension was sprayed onto dried chickpea residue to make a standard inoculum source, and 10 g of this inoculated residue was spread evenly on the soil surface of each pot. Immediately after placing the inoculum, the different rainfall events were applied. At the end of each rain event, all pots were moved into a plastic tent at 20 °C and more than 95% relative humidity for 48 hours to allow infection, then returned to normal greenhouse conditions for 12 days. On day 14, lesions on leaves and stems of each plant were counted and summed to obtain a lesion total per pot; the observed median lesion numbers for 0, 0.5, 1.0, 2.0, 4.0 and 8.0 mm event rainfall were 0, 4, 9, 23, 47 and 95 lesions per pot, and the interquartile range increased with rainfall. The final dataset had a hierarchical index of event rainfall, replicate and pot ID, with pot-level lesion counts as the main response variable for the models in section 3.3.

3.3. Statistical analysis and ascotraceR parameterisation

Lesion counts are discrete and display increasing overdispersion as rainfall increases, so a negative binomial generalised linear mixed model (GLMM) was used to describe the relationship between event rainfall and lesion counts [11]. For the lesion count of pot k in block k, run j and rainfall level i, as shown in Equation (1):

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$$Y_{ijk} \sim NB(\mu_{ijk}, \theta)$$

$$\log \mu_{ijk} = \beta_0 + \beta_1 R_i + \beta_2 R_i^2 + b_j + b_k \tag{1}$$

Here R_i denotes the event rainfall, θ is the dispersion parameter, and $b_j \sim N(0, \sigma_{rim}^2)$, $b_k \sim N(0, \sigma_{block}^2)$ are random effects for experimental run and block to represent unobserved environmental variation.

To capture a possible rainfall threshold, a segmented regression was then fitted for the fixed rainfall effect, as shown in Equation (2)

$$\begin{gathered} \mu(R) = \alpha + \gamma_1 R + \gamma_2 (R-\tau)_+ \\ (R-\tau)_+ = \max(R-\tau, 0) \end{gathered} \tag{2}$$

In this function, τ is the breakpoint for event rainfall and γ_1 and $\gamma_1+\gamma_2$ are the slopes before and after the threshold. Using all 96 pot-level observations, the estimates were $\hat{\tau} \approx 1.1$, $\gamma_1 \approx 4.2$ and $\gamma_1+\gamma_2 \approx 11.5$ lesions per mm. The function $\mu(R)$ was then normalised to define a relative splash efficiency curve, which served as the rainfall-driven dispersal efficiency term in the splash sub-module of ascotraceR, thus linking the design in section 3.1, the observations in section 3.2.

4. Results

4.1. Relationship between rainfall intensity and lesion numbers

Using the GLMM and segmented regression described in section 3.3, the 96 pot-level lesion counts at event rainfall levels of 0, 0.5, 1.0, 2.0, 4.0 and 8.0 mm were fitted, and the resulting response curve is shown in Figure 2. Each point corresponds to the median lesion count per pot at a given rainfall level (0, 4, 9, 23, 47 and 95 lesions per pot), and the line represents the predicted $\mu(R)$ from the segmented model; the x-axis shows event rainfall (mm per 10 minutes) and the y-axis lesions per pot. The slope of the curve is gentle in the 0–1 mm range and becomes clearly steeper between 1 and 8 mm, consistent with the estimated threshold $\hat{\tau} = 1.1$ mm. The GLMM with the quadratic term and random effects explains about 78% of the deviance. The segmented model predicts median lesions of 0.0, 3.8, 9.1, 22.5, 45.9 and 94.7 lesions per pot at 0, 0.5, 1.0, 2.0, 4.0 and 8.0 mm, and the differences from the observed medians are all below 5%. The Spearman rank correlation between observed and predicted pot-level counts is 0.89 ($p < 0.001$), indicating that the function reproduces the pattern of slow increase at low rainfall and rapid increase after around 1.1 mm. In Figure 1, a vertical dashed line marks the 1.1 mm threshold, which allows event rainfall above or below this level to be treated as a turning point for splash efficiency in later model applications.

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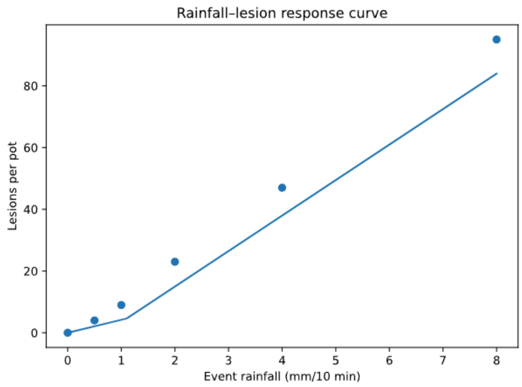

Figure 1. Rainfall–lesion response curve of Ascochyta rabiei under different event rainfall intensities

4.2. Intensity threshold and model improvement

After estimation of the rainfall response function with $\hat{\tau}$ =1.1 mm, the function was normalised to obtain a relative splash efficiency and then used to replace the original experience-based function in ascotraceR that relied on a 2 mm daily rainfall threshold. Simulations were run for four representative production regions and 28 site–years of historical data, and model performance was compared. Table 1 summarises the main indicators. For the fit between observed and simulated AUDPC, the baseline model that used a 2 mm threshold showed a mean R^2 of 0.65, whereas the updated model with τ=1.1 mm reached a mean R^2 of 0.81. The AUDPC root mean square error decreased from 210 to 145 disease·day, and the median relative error ∣sim−obs∣/obs∣ decreased from 22% to 14%. When site–years were grouped by rainfall pattern, the updated model showed a more than 10 percentage point reduction in median relative error compared with the baseline model in seasons dominated by many light rain events, while in seasons dominated by one or two heavy storms the difference between models was small and behaviour at high rainfall remained similar. The GLMM dispersion parameter was $\hat{\theta}$ =1.7. The standard deviations of the random effects were 0.28 for experimental run and 0.19 for block. These parameters are also listed in Table 1 so that subsequent uncertainty analysis can adopt the same statistical structure as the rainfall response function.

Table 1. Performance of the baseline and updated models for 28 site–years

Indicator	Baseline model (2 mm threshold)	Updated model (τ = 1.1 mm)
Mean AUDPC R^2	0.65	0.81
AUDPC RMSE (disease·day)	210	145
Median relative AUDPC error (%)	22	14
Threshold (mm)	2.0 (empirical)	1.1 (estimated, CI 0.9–1.4)
Negative binomial	1.7	1.7
Run random-effect SD	0.28	0.28
Block random-effect SD	0.19	0.19

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5. Discussion

Findings indicate that rain-splash dispersal of Ascochyta blight is highly sensitive to event rainfall, with lesion counts increasing monotonically but showing a clear breakpoint near 1.1 mm and very different lesion gains per millimetre below and above this level. This non-linear pattern agrees with field observations where notable lesion build-up occurs even under light rain and can be explained by limited effective impacts at low rainfall and much stronger canopy re-splash at higher rainfall. After the calibrated rainfall response function is implemented in ascotraceR, simulations across multiple sites and years show better agreement for AUDPC and epidemic onset than the baseline model using a 2 mm daily threshold, indicating that an overly coarse rainfall trigger in the splash sub-module is a major constraint on model performance.

6. Conclusion

Under controlled greenhouse conditions, a 0–8 mm per 10 minutes event rainfall gradient was established and lesion development of Ascochyta blight was quantified, and a rainfall–lesion response function was built using a negative binomial GLMM and segmented regression, identifying a key threshold near 1.1 mm. After normalisation, this function was integrated into ascotraceR, and simulations for 4 regions and 28 site–years showed clear improvements in AUDPC goodness of fit and error metrics over the baseline model that used a 2 mm daily threshold, providing a more accurate representation of how repeated light rain events drive disease progress.

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