Effect of Plant Age and Leaf Position on Susceptibility to Wheat Stripe Rust

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ABSTRACT

In addition to pathogen propagule dispersal, disease spread requires successful infection of host tissue. In plant disease epidemiology, susceptibility of host tissue is often assumed to be constant. This assumption ignores changes in host phenology due to developmental stage. To examine this assumption, 3-, 4-, and 5-week-old wheat plants were inoculated with equal quantities of urediniospores of *Puccinia striiformis* f. sp. *tritici*, the causal agent of wheat stripe rust (WSR). Disease severity on each leaf was assessed and fit by mixed-effect linear model as a function of leaf position and plant age. Younger plants had significantly greater disease severity than older plants, with mean severities

of 50.4, 30.1, and 12.9% on plants that were 3, 4, and 5 weeks old, respectively, at time of inoculation. This effect was greater on leaves higher on the plant. Within same-aged plants, younger leaves had significantly greater disease severity than older leaves, with mean severities of 40.2, 34.8, and 17.7% on the uppermost, second, and third leaf, respectively. These results suggest that the vertical distribution of WSR lesions in agricultural fields could be driven by differences in host susceptibility more so than propagule dispersal.

Additional keywords: infection efficiency.

Epidemics, defined as spatiotemporal changes in disease severity (Madden et al. 2007), require virulent pathogens, susceptible hosts, and conducive environments to coincide in time and space. Therefore, to better understand epidemic spread, it is important to understand not just propagule movement but also differences in availability of susceptible host tissue over both time and space because these differences can result in changes in pathogen growth, reproduction, and infection efficiency. Several models have been developed to account for the epidemiological effects of changing host architecture and phytochemistry as a plant grows (Costes et al. 2013; Ferrandino 2008; Kushalappa and Ludwig 1982; Tivoli et al. 2012). A very common assumption in plant disease epidemiology is that the degree of susceptibility of host tissue is constant both temporally and spatially. Violation of this assumption could significantly alter expected patterns of disease spread. However, relatively little attention has been paid to the interaction of disease progress and developmental stage of the host. Plant defenses have largely been viewed singly, but it has been argued that host–pathogen relationships occur along ecological continua, including factors affecting the fitness of the host (Agrawal and Fishbein 2006; Barrett et al. 2009), but quantification of the magnitudes of these effects is sparse.

There have been many reviews written on the genetic and biochemical basis for host susceptibility (van Schie and Takken 2014) and host resistance (Chisholm et al. 2006; Michelmore et al. 2013), as well as the differences in types of resistance between race-specific major gene resistance and race-nonspecific minor gene resistance (Flor 1971; Johnson 1984; St. Clair 2010). Changes in host phenology throughout a growing season can have immense impacts on host susceptibility to infection by pathogens. Host phenology, defined as the timing of changes of developmental phase of a species over time in response to biotic and abiotic factors (Lieth 2013), has been shown to affect the ecological interactions of plants, including changes in susceptibility of plants to herbivory (Boege

and Marquis 2005; Karban 1987; van Asch and Visser 2007) and to disease (see below) with developmental stage of host plants.

Changes in host phenology occur both over time, as a plant ages, and spatially, from older plant tissues to younger ones. In domesticated crops such as wheat, these phenological changes can vary depending on a complex interaction of climatic and agronomic factors (Tao et al. 2012). The rates of these changes are dependent on many interacting ecological factors. The resource availability theory predicts that there is a negative relationship between rate of plant growth and amount of defense (Kikuzawa and Lechowicz 2011). Therefore, annual plants grown in nutrient-rich environments such as commercial wheat fields may exhibit greater intraplant variation in leaf tissue phenology than longer-lived plants in nutrient-poor environments due to reduced defenses and increased rate of tissue changes due to growth.

In addition to environmental effects, the life strategies of the microbiota of the phyllosphere can also affect host susceptibility. Population sizes of epiphytic microbes can vary with leaf position within a plant, and as a function of plant age. Epiphyte population sizes typically increase with plant age but display an inverse relationship between populations of introduced microbes and plant age (Jacques 1996). Disease severity has been found to be correlated with host phenology in varying ways, which might be accounted for, in part, by the life strategy of the pathogen. Necrotrophic pathogens, which preferentially derive their nutrition from plant tissue which they have killed, often exhibit greater infection efficiency as plant tissues age. In the case of the facultative saprophyte *Ascochyta rabiei*, younger chickpea leaflets were less susceptible to infection than older leaflets (Dolar 1997). The relationship is not always as simple as this, because some necrotrophic pathogens have displayed the greatest infection efficiency during specific developmental stages based on the presence of certain plant structures. For example, strawberry becomes much more susceptible to infection by *Botrytis cinerea* once it flowers (Mertely et al. 2002). For obligate pathogens such as rusts and mildews, the requirement of healthy host tissue for completion of their life cycles (Schulze-Lefert and Panstruga 2003) suggests that host susceptibility may decrease in later phenological stages, as plants redirect resources away from aging tissues to younger tissues. Previously, older soybean plants were found to have reduced susceptibility to infection by *Phakopsora pachyrhizi* but susceptibility did not significantly vary by leaf position (Bonde et al. 2012). Likewise, the rust *Puccinia jaceae*, a biocontrol agent on yellow star

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thistle, had reduced infection efficiency on plants older than 4 to 6 weeks but did not have a clear relationship with leaf position (Bennett et al. 1991).

Because biotrophic pathogens such as *Puccinia striiformis* f. sp. *tritici* require healthy host tissue to complete their life cycles, it follows that senescence could have a significant effect on their ability to cause disease. Age-related resistance (ARR), in which, as plant tissues age, they become less susceptible to pathogen infection, has been found in many systems and can take many forms, including reducing susceptibility of plants to infection by viruses, bacteria, and fungi by race-specific or nonspecific mechanisms (Panter and Jones 2002). It has been previously shown that wheat plants with high-temperature adult plant resistance (HTAP), a form of genetic race-nonspecific resistance in which minor resistance genes are influenced by plant growth stage and temperature (Chen 2013; Mallard et al. 2005), were found to be present in wheat, reducing the severity of stripe rust infections at later growth stages and at higher temperatures (Qayoum and Line 1985). More general physiologic changes in receptivity, the change in infection frequency with plant growth stage in susceptible hosts (Richard et al. 2012), has been observed in slow rusting cereals (Herrera-Foessel et al. 2007; Steffenson and Wilcoxson 1987), and may play a significant role in shaping the speed and severity of epidemics. In this study, we tested the effect of plant age and leaf position on the susceptibility of wheat to *P. striiformis* f. sp. *tritici* when grown in a common environment.

MATERIALS AND METHODS

Study site and materials. Three runs of the experiment were performed in the Oregon State University West Greenhouse facility, which was controlled to maintain nominal temperatures of 18 and 16°C temperatures (day and night, respectively). Actual daily high and low temperatures for the greenhouse were recorded, with a mean high of 23.1°C and a mean low of 17.3°C. For each run of the experiment, seeds were planted on three dates at 1-week intervals. For each planting date of each run, 10 seeds of soft white winter wheat (*Triticum aestivum*) ‘Jacmar’, which has not been found to exhibit HTAP or hypersensitivity reaction to the race of *P. striiformis* f. sp. *tritici* used in this study (Akanda and Mundt 1997; Jacquot 1977), were planted in each of 10 cylindrical, 3.785-liter plastic plant pots containing potting Sungro Sunshine LA4 P soil. All plants were grown directly below grow lamps in the greenhouse in a randomized complete block design, where each of the 10 blocks contained one pot of each of the three plant ages. This blocking structure was maintained during the inoculation procedure and during post-inoculation growth in the greenhouse (see below). The plants were watered every other day to keep soil at or near field capacity. Plants were fertilized every other week with 0.39 ml of dry Miracle Gro fertilizer (Scotts MiracleGro, Marysville, OH) per pot, dissolved into 100 ml of water. Lights were set to a schedule of 16 h on and 8 h off, turning on at 06:00 and turning off at 22:00. During hours when the lights were scheduled to be on, if ambient light levels exceeded 90 $\mu\text{mol photons m}^{-2} \text{s}^{-1}$, the lights shut off and turned back on when light levels dropped below 75 $\mu\text{mol photons m}^{-2} \text{s}^{-1}$.

Inoculation. Inoculations were performed with *P. striiformis* f. sp. *tritici* CDL 29 (Chen 2005), which was stored in liquid nitrogen. Fresh inoculum was produced by inoculating plants grown at 15°C with frozen inoculum, and harvesting the progeny urediniospores. Each block of three pots received a mixture of 15.2 mg of urediniospores and 228 mg of talc. This mass of inoculum contained approximately 858,800 urediniospores, as calculated by the density of similar-sized urediniospores of *P. hordei* (Teng and Close 1978).

In all, 9 randomized complete blocks of plants were inoculated, and the 10th block served as a noninoculated control. Plants were 3, 4, or 5 weeks old at the time of inoculation. All plants were moved from the greenhouse to the outdoors for 12 h prior to inoculation, because exposing wheat plants to cooler temperatures has been shown to increase infection efficiency (unpublished data). Prior to

inoculation, each block of plants was placed in an open cardboard box 0.56 m in length by 0.42 m in width by 0.51 m in height, and plants were misted with water from a squirt bottle. The inoculum-talc mixture was hand-pumped out of a DeVilbiss Powder Blower (model 119; DeVilbiss Healthcare, Somerset, PA) rotated around the top of the inoculation box, with the tip of the powder blower oscillating from the height of the pot (0.165 m) to the top of the inoculation box (0.51 m). The uninoculated control plants were placed in the inoculation chamber, misted with water, and placed in the dew chamber. The plants were placed in the dew chamber at 13°C overnight, then returned to the greenhouse, where the growing conditions remained as they were prior to inoculation.

Disease assessment. All plants were sampled approximately 1.75 generations after inoculation, and the exact generation stage as predicted by Shrum’s degree-day model (Shrum 1975) was recorded. This allowed for the maximum number of lesions to sporulate without encroaching on the subsequent generation. The disease severity as estimated by visual observation of percent of leaf area covered by wheat stripe rust (WSR) lesions of all leaves on the main stem of all plants was recorded. Any leaf that had senesced to the extent that it was no longer possible to accurately visually assess disease severity was excluded from the study.

Data analysis and model fitting. The effect of leaf position and plant age on disease severity over time was analyzed using R statistical computing software (R Development Core Team 2015). Due to heteroscedasticity, we used a square-root transformation of severity, which performed better than a log transformation, to normalize the errors (Ahrens et al. 1990). Leaf position was considered a quantitative variable, because it has been shown to be closely tied to growing degree-days and to attach to the culm at evenly spaced intervals (Karow et al. 1993). Leaves 1 to 3 were considered suitable for analysis because they were present at the time of inoculations, and had not senesced at the time of disease assessment. Leaf 1 was the uppermost, fully expanded leaf at the time of inoculation, and leaves 2 and 3 were sequentially lower. Disease severity was regressed against the number of degree-days elapsed between inoculation and disease assessment date to examine the disease severity at equal plant and pathogen developmental stages across runs. Mean disease severity was aggregated across all plants in each pot (the experimental unit). Differences in disease severity between runs were examined using two-way analysis of variance followed by Tukey’s honestly significant difference posthoc test to determine which runs differed from each other.

We tested the linear effects of leaf position, plant age, mean daily temperature, and all interactions thereof as fixed effects using the step function in the lmerTest package for the R Statistical Programming Language (Bates 2015). Block nested within run was considered as a random effect with potential to influence the intercept. The likelihood-ratio test using maximum-likelihood estimation was used to compare mixed-effects models with differing measures of plant age, the cumulative degree-days from planting date to inoculation date, and the cumulative degree-days from planting date to sampling date (Baayen et al. 2008; Barr et al. 2013). The fixed effects most correlated with disease severity were plant age and leaf position, each of which are examined in more detail below, as well as their interaction. Inclusion of mean daily temperature, mean daily high temperature, and mean daily low temperature, as well as each of their interactions with all other explanatory variables, resulted in linear mixed models with greater Akaike Information Criteria (AIC) values and, therefore, were eliminated from the final model. Figures were generated using the base R package and with the ggplot2 package (Wickham et al. 2013).

RESULTS

The square root of disease severity decreased linearly with plant age as measured by cumulative degree-days from planting date to inoculation date (Fig. 1), as well as from planting date to sampling date (data not shown). Although direct comparisons of degree-days

from planting date to inoculation date and degree-days from planting date to sampling date was not possible given the differing fixed effects, they were very similar in fitting the data, with AIC values of 2309.2 and 2315.3, respectively. Therefore, the final model included degree-days from planting date to inoculation date, because that resulted in a lower AIC and more closely addressed the questions this study aimed to examine regarding the effect of plant age on susceptibility to infection. The random effect of block nested within run was highly significant (P value $< 2.2 \times 10^{-16}$) as tested by the χ^2 test. The best-fit model of the square root of severity s was

$$\sqrt{s} = 23.21 - 0.016d - 2.14l \tag{1}$$

in which d is defined as degree-days from planting date to inoculation date and l is defined as the leaf position from the top of the tiller. This model was used to test significance of the effects described below.

Magnitude of fixed effects on disease severity. The overall mean disease severity across all plants in all treatments was 31.0%,

which is within a range in which susceptible leaf area is not a significantly limiting factor but disease severity differences are likely to be large enough to be significant (James 1974). Disease severity was highly correlated with plant age, calculated as cumulative degree-days from planting date to inoculation (P value $< 2 \times 10^{-16}$). Mean disease severity and variance across the uppermost three leaves in all pots was reported for each leaf and each age plant for both square-root transformed and nontransformed data (Table 1). Younger plants had greater disease severity than older plants on each individual leaf (Fig. 1). Disease severity decreased linearly as leaf position increased (Fig. 2; P value: 2.01×10^{-10}), with mean severities of 40.2, 34.8, and 17.7% on the uppermost, second, and third leaf, respectively.

Mean disease severity across the uppermost three leaves decreased with cumulative degree-days (Fig. 1). Additionally, disease severity was greater on younger plants than on older plants, with mean severities of 50.4, 30.1, and 12.9% on plants that were 3, 4, and 5 weeks old, respectively, at time of inoculation (Table 1; Fig. 2). There is a visual suggestion that disease severity decreased more rapidly as a function of cumulative degree-days on upper leaves

Fig. 1. Random-intercept linear regression of the square-root of mean wheat stripe rust severity per pot as a function of cumulative degree-days from planting date to inoculation date. Bold solid line is the mean of all replicates. **A**, Leaf 1, the uppermost leaf present at the time of inoculation (no significant effect of replicate); **B**, leaf 2, the proximal leaf below leaf 1; **C**, leaf 3, the proximal leaf below leaf 2; and **D**, mean of uppermost three leaves.

TABLE 1. Untransformed (Untrans) and square-root transformed (Trans) mean wheat stripe rust severity and variance (Var) on each of the uppermost three leaves of wheat plants by age in weeks at time of inoculation^a

Weeks	Leaf 1				Leaf 2				Leaf 3			
	Untrans		Trans		Untrans		Trans		Untrans		Trans	
	Mean	Var	Mean	Var	Mean	Var	Mean	Var	Mean	Var	Mean	Var
3	63.8	252.8	7.9	1.5	56.5	161.4	7.3	2.8	31.0	294.0	5.2	2.8
4	44.5	289.2	6.4	3.9	34.4	193.9	5.4	5.5	11.4	141.2	2.6	5.5
5	15.5	151.4	3.2	3.6	14.4	100.8	2.8	6.5	10.8	57.3	2.2	6.5

^a Leaf 1 was the youngest, fully expanded leaf at time of inoculation; leaves 2 and 3 were sequentially older. Variation produced by run was excluded.

than on lower leaves (Fig. 1; Table 2) but this interaction was not statistically significant.

DISCUSSION

Our study demonstrated a greater disease severity on younger leaf tissue, which suggests a greater proportion of urediniospores were able to successfully infect and produce sporulating progeny uredinia on younger wheat plants and younger wheat leaves within a given plant. Additionally, it has been observed that lesion expansion, which is particularly important in WSR (Berger et al. 1997; Shrum 1975), is reduced at later growth stages of wheat (Luo and Zeng 1995). These results could help inform breeders of the trade-offs conferred by selecting for delayed senescence (Wang et al. 2015).

This study quantifies the relative receptivity of a highly susceptible cultivar of wheat to infection by *P. striiformis* f. sp. *tritici* as influenced by the age of plant and leaf tissue within an individual plant. These results corroborate previous observations of greater host resistance at later growth stages in agricultural fields (Bux et al. 2012; Panter and Jones 2002; Runyon et al. 2010), as well as help elucidate how senescence-driven resistance reduces susceptibility to stripe rust to a greater extent on younger leaves relative to older leaves within a single plant. Examining cumulative degree-days from planting date to sampling date could act as a better gauge of phenological changes in host susceptibility than relying solely on temporal metrics (McMaster and Wilhelm 1997). These results suggest a general physiologic response within a susceptible cultivar influencing receptivity, in addition to differences among cultivars caused by HTAP race-nonspecific resistance in wheat (Chen 2013; Milus and Line 1986; Qayoum and Line 1985) and race-nonspecific ARR in *Arabidopsis thaliana* (Diener and Ausubel 2005; Quirino et al. 1999)

The vertical gradient of disease severity, in which severity per leaf declined from upper, younger leaves to older, lower leaves of wheat plants, displayed qualitatively the same pattern as our previously observed vertical distribution of WSR lesions in field plots (Farber et al. 2017), where a majority of progeny lesions were observed at or above the source leaf. This study also provides evidence to support a plausible alternate explanation of the mechanism resulting in the observed vertical gradient of stripe rust lesions in the field: that differences in host receptivity to infection by *P. striiformis* f. sp. *tritici* urediniospores by age and leaf position were the primary

driver of the resulting observed vertical distributions of lesions, as opposed to the hypothesis that a vertical gradient of urediniospore deposition resulted in a greater proportion of urediniospores landing on upper leaves than lower leaves.

Our results also may be of relevance to the temporally changing factors affecting the rate of spread of plant epidemics. The greater disease severity we observed on younger leaves within a plant, which are higher in the canopy, could help explain the relatively frequent long-distance dispersal events observed in WSR, because urediniospores emanating from uredia higher in the canopy could potentially have a greater probability of getting transported by higher-speed wind currents compared with urediniospores originating from lower in the canopy (Aylor 1999; Pfender et al. 2006). These long-distance dispersal events contribute to accelerating rates of disease spread (Cowger et al. 2005; Mundt et al. 2009). Therefore, incorporating a temporal and vertical gradient of susceptibility into disease-spread models could increase their ability to accurately predict the spread of epidemics.

Our study could help elucidate the complex ecological relationships between plant development, environmental conditions, and disease pressure from pathogens with greatly differing disease pressure by parsing out the effect of plant tissue age and senescence on receptivity to infection by *P. striiformis* f. sp. *tritici*. Many of these ecological relationships have been theorized (Alcázar et al. 2011; Develey-Rivière and Galiana 2007; Kikuzawa and Lechowicz 2011; Tylianakis et al. 2008). Although the conditions of an agricultural field may differ, greenhouse studies have been used to examine biological processes in a more controlled environment (Bux et al. 2012; Hou et al. 2015). Due to the great number of interacting variables affecting host receptivity in field settings (Grulke 2011), however, more studies are needed to understand how age-dependent disease risk affects the portion of a plant’s carbon budget going to disease. This research could inform future studies of the biochemical basis for nonspecific ARR to obligately biotrophic pathogens. Plant growth and development is regulated by several metabolites or phytohormones, including 3-indole-acetic acid, gibberellins, cytokinins, and many other compounds (Goodman et al. 1986). Likewise, senescence is regulated by its own set of phytohormones, principally ethylene, jasmonic acid, salicylic acid, and abscisic acid (Häffner et al. 2015), but can also be counteracted by the aforementioned plant-growth phytohormones. In the absence of pathogens, senescence has been described as a form of programmed tissue death, which redirects resources to other parts of the plant. In annual grasses such as wheat, this redirection is largely due to redirecting resources toward the uppermost leaves, which receive the most photosynthetically active radiation. Although HTAP has been well documented in many landraces of wheat (Bux et al. 2012), little attention has been paid to how susceptibility to infection by *P. striiformis* f. sp. *tritici* varies by leaf position and plant age for wheat genotypes lacking HTAP. However, it has been shown that several defense genes are upregulated during senescence of *A. thaliana* and, for many of these genes, this increase in mRNA transcripts occurs independently of pathogens or salicylic acid accumulation (Quirino et al. 1999). Similar work in wheat could aid in our understanding of the mechanisms underlying the ARR conferred by changes in phenology addressed here, which could be a benefit to controlling epidemics by breeding for resistance.

Fig. 2. Random intercept linear regression of the effect of leaf position on the square root of wheat stripe rust severity across all age treatments. Plants were 3 (○), 4 (△), or 5 (+) weeks old at the time of inoculation.

TABLE 2. Parameters and Akaike Information Criteria (AIC) for mixed linear regression of disease severity as function of age by individual leaf and replicate

Leaf	a	b	Variance of run	AIC
1	21.5	−0.16	0	309.9
2	20.95	−0.016	0.55	294.8
3	14.14	−0.011	0.93	314.7
Mean	18.97	−0.014	0.22	260

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