THESIS

THE IMPACTS OF HIGH TEMPERATURE ON BACTERIAL BLIGHT RESISTANCE GENES IN RICE

Submitted by

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ABSTRACT

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Rice is cultivated around the world and serves as a primary source of income and calories for many people. However, rice yield is threatened by the bacteria *Xanthomonas oryzae* pv. *oryzae* (*Xoo*), and outbreaks can be devastating to global communities. *Xoo* is the causal agent of bacterial blight (BB) in rice, and it proliferates in rice-growing climates.

As climate change progresses, the trend of increasing BB severity may result in increased losses for growers. Disease severity, quantified through lesion lengths, increases at high temperature in rice. Previous studies indicated this pattern of increased disease phenotypes occurs even when a resistance (*R*) gene is present, except for one, *Xa7*. Our rationale for these experiments is to determine if the classification of an *R* gene can predict its performance against BB outbreaks. The classification of *R* genes in rice is a recent addition to the scope of our knowledge of plant pathology and has been the result of studies on nucleotide polymorphisms, genetic mapping, and fluorescent imaging of protein localization. Grouping the underlying mechanisms of action of individual *R* genes, such as the executor genes *Xa7* and *Xa10*, allow for comparative studies to further elucidate details of their assigned classes. Not all *R* genes have been classified but establishing a trend that some *R* genes maintain efficacy under higher temperatures would provide breeders with more tools to develop climate-friendly rice lines.

This study indicates that *R* genes that remain effective at high temperature may be classified into the same category of executor *R* genes. More research is needed to determine if *R* gene classification predicts durability under heat stress.

This study explores BB lesion lengths and *Xoo* colony counts at high and low temperatures. We find that at high temperature relative to low temperature, disease lesions were more severe in IR24, containing no active *R* gene, and in plants containing the *R* genes *Xa21*, *xa5*, and *Xa3*. Lesions were shorter in plants with *Xa7* and *Xa10*. Additionally, under the same treatments, bacterial numbers increased to higher levels in IR24, *Xa21*, *xa5*, and *Xa3*. Numbers in *Xa7* were reduced while numbers in *Xa10* were low early in infection, but eventually increased beyond those measured at low temperature. Degree of lesion restriction did not always correspond to degree of restricted bacterial numbers, suggesting that severity of lesions may not always be a predictor of bacterial multiplication in the plant.

Xa7 and *Xa10* are classified as executor *R* genes. The mechanism of action in these genes may play a role in their durability at high temperatures. We hypothesize that the success of executor *R* genes may be a result of protein accumulation in the nucleus. This mechanism might be analogous to instances of temperature sensitive pathogen defense related protein accumulation, as seen in *Arabidopsis*. This mechanism may be induced or enhanced by the presence of reactive oxygen species (ROS) or other heat-stress related markers. More research is needed to explore the signaling between heat-stress pathways and *R* genes.

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For D.

TABLE OF CONTENTS

Chapter One – Introduction: The Patterns of Our Planet are Shifting

The environmental conditions of our planet are in a constant state of flux, but it has become evident that average temperatures are progressing beyond previously recorded ranges (Lee, 2021; IPCC, 2023). This has impacted the planet in several ways including altering ocean temperatures (Harley et al., 2006; Brierley and Kingsford, 2009; Wernberg et al., 2011) and their associated trade winds, modifying global weather patterns (Prospero and Lamb, 2003; Luo et al., 2012; England et al., 2014) and local growing seasons (Hijmans, 2003; You et al., 2009; Uleberg et al., 2014; Van Oort and Zwart, 2018), and simultaneously increasing and decreasing precipitation levels resulting in both flooding and drought (Prospero and Lamb, 2003; Guhathakurta et al., 2011; Trenberth, 2011; Jamieson et al., 2012; Li et al., 2015; Arnell and Gosling, 2016; Haile et al., 2020) in numerous regions. We can extrapolate that these environmental changes further influence ecological diversity and soil health (Cheung et al., 2009; Bellard et al., 2012). Changing environmental conditions require global agricultural systems to keep pace while also meeting the demands of an increasing global population (Lutz and Samir, 2010) and fighting to remain free from urbanization (Fazal, 2000; Pandey and Seto, 2015; Beckers et al., 2020). Earth's population is predicted to reach at least 9.6 billion by 2050 and upwards of 10.4 billion by 2100 (Bradshaw and Brook, 2014; United Nations Department of Economic and Social Affairs, 2022), meaning that harvests will need to utilize less space while stretching further to accommodate all who rely on it for sustenance.

Climate-Based Challenges in Agriculture

Globally, agricultural crops are used in everything from shelter, to clothing, to fuel, to medicine (Mwaikambo, 2006; Yuan et al., 2008; Mamedov, 2012; Petrovska, 2012; Baines, 2015). But most importantly, they help us meet our daily nutritional and caloric needs (Prescott-Allen and PrescottAllen, 1990) and provide us with the physical energy to go about our day. However, as favorable growing conditions change these same crops begin to face steeper odds of making it all the way from the field to our plate. Tomatoes, cabbage, onion, peppers, and eggplant, just to name a

few, are susceptible to deviations in local growing conditions. In cases where shifts in temperature, drought, or altered soil conditions occur, so too must the methods of irrigation and pest or pathogen management, in turn often requiring large modifications to manual labor practices and increased use of pesticides (Pena et al., 2019; Ristaino et al., 2021) Post-harvest storage methods in grapes, apples, avocados, and pears, for example, also require modifications in the form of additional labor and processing to prevent excess spoilage and further crop loss. Care must also be taken to monitor changes in ethylene production and ripening response (Eaks, 1978; Ferguson et al., 1994; Woolf et al., 1999; Thompson et al., 2002), which can impact the quality of harvest throughout the process of transport and distribution to consumers.

Altered temperatures also change the phytobiome (Jansson and Hofmockel, 2020; Abdul Rahman et al., 2021), or the community of organisms that coexist in, on, and around crops. Interruptions of these communities have profound effects on the soil environment, the plant itself, and the surrounding agroecosystem. Phytobiomes have a range of temperatures in which they thrive and remain balanced, and in cases where temperatures extend beyond those limits, severe damage and death may occur within the community of microorganisms (Ratkowsky et al., 1982). These organisms have evolved alongside plants and often have beneficial symbiotic relationships with them

(Venkateshwaran et al., 2013; Van Der Heijden et al., 2016), such as nitrogen fixing bacteria (Franche et al., 2009) and other types of metabolite exchanges (Piel, 2009). Disruption of this delicate community can result in additional damage to plant health and can reduce local biodiversity (Berendsen et al., 2012; Wallenstein, 2017; Wei et al., 2019). Some of these organisms may also offer a line of defense against harmful pests and pathogens and removal of them may result in colonization by disease causing agents or parasites (Zarraonaindia et al., 2015; Vannier et al., 2019; De Corato, 2020; Zhang et al., 2021).

Impacts of Climate Change on Crop Physiology

Temperature fluctuations are major regulators of the internal machinery that allows plants to thrive (Li et al., 2022; Zhu et al., 2022). It is important for plants to coordinate temperature sensing capabilities to facilitate vernalization, germination, stem elongation, flowering, ripening, and senescence in time with seasonal weather conditions. These processes are designed to correlate with optimal levels of seasonal precipitation, emergence of local pollinators, and the onset of frosts that plants must work around to produce viable fruit and seeds (Searle and Coupland, 2004; Donohue, 2005). For example, temperature tells a plant when conditions are right for flowering, which involves detailed signaling for organ development (Matsui et al., 1997; Greenup et al., 2009; Fjellheim et al., 2014). Temperature also changes the water content of the plant by altering rates of transpiration (Atkin and Tjoelker, 2003), which can disrupt salt concentration and organelle functions (Sharmin et al., 2021). CO_2 concentrations often increase with temperature which also changes rates of photosynthesis (Hikosaka et al., 2006; Sage and Kubien, 2007; Mathur et al., 2014). When carefully controlled, increased photosynthesis can extend yield capacities, but when left unchecked it can cause an accumulation of reactive oxygen species which will severely damage the plant (Kimball and Idso, 1983; Dat et al., 2000; Gupta et al., 2015; Foyer, 2018).

Since plants are sessile, or rooted in one spot, they are unable to physically move to better conditions when undesirable conditions arise. Instead, they rely on physiological changes above and below ground (de Lima et al., 2021a) to cope with surrounding stimuli such as the presence of drought, saline soils, or temperature stress (Maisura et al., 2014; Xu et al., 2021; Liu et al., 2022). Plants prefer a specific window of temperatures to thrive, with an individual range of needs for each species or cultivar. In the case of any plant, extremes of hot or cold can be incredibly damaging or even deadly (Zhu et al., 2022). In the meantime, plants work overtime to adjust to their surroundings from one moment to the next to keep their internal processes running optimally

(Treshow, 1970; Chapin et al., 1987; Lamers et al., 2020). Plants are equipped to exist within a range of conditions, but continuous exposure to temperatures outside of that range will cause them to pull resources away from processes that support healthy growth and development (Heckman et al., 2019; He et al., 2022). Warmer than average temperatures that aren't quite severe enough to trigger stress responses still impact crops, like rice, by causing leaves and other plant organs to develop faster (Casal and Balasubramanian, 2019). But when those temperatures cross the threshold into the range of prolonged stress, they cause plants to lose water quickly through increased evaporation, or transpiration, rates leading to wilt and later to desiccation and death.

Heat stress is defined as exposure to temperatures outside the optimal range for physiological processing and may range from severe (irreversible) to milder (reversible). In less severe cases, plant responses to heat stress include fluctuations in a range of processes such as: photosynthesis, cell membrane thermostability, production of oxidative species, transcription, post-transcriptional modifications (noncoding RNAs), epigenetic regulation (DNA methylation, histone modification, and chromatin remodeling) and epigenetic memory (Zhao et al., 2020).

We can gather insight on temperature-related stress from many models. For example, in *Brassica napus* high temperature is associated with defects in seed development, altered oil composition, and reduced capacity for dormancy/storage (Mácová et al., 2022) which impacts its value as an oilseed crop. We also have examples in wheat in which harvests were delayed and accompanied by less than market standard quality of grain (de Lima et al., 2021b) do. If pushed far enough, temperature impacts become damaging to almost all species of flora and fauna.

Legacy of Rice Production & Modern Challenges

While all agricultural crops have a place on the global table, the bulk of the calories consumed from plants comes from various cereal crops, including maize, wheat, and rice (fao.org -statistical yearbook). Of these, rice is the most valuable crop worldwide (USDA-ERS, 2023). In fact, rice is so

connected with nourishment that in some languages the word for rice is synonymous with the word for food or meal.

The history of rice cultivation is described in (Crawford and Shen, 1998). Rice thrives in tropical and subtropical regions and farming may have begun in multiple countries at around the same time, over 6500 years ago. China, Thailand, Cambodia, Vietnam and India all developed local methods for farming rice with Japan, Korea, Myanmar, Pakistan, Sri Lanka, Philippines, and Indonesia following suit soon after. (Gnanamanickam, 2009) adds that while rice is often associated with paddy farming, or submersion in watery soils, it originally may have thrived in more traditional field grass conditions. A mutation in a gene related to aerobic gas exchange likely allowed rice to adapt to anerobic soil conditions (Miro and Ismail, 2013). There is evidence that rice can grow in a multitude of environments, but common paddy varieties perform more robustly in areas with high humidity and warm climates (Huke, 1976).

Most of the world's population is concentrated in Asia and it follows that the same region produces 90% of the world's rice, destined for local markets and export trading (Bandumula, 2018). Rice production in southeast Asia alone was approximately 418.56 million tons in 2019, which accounts for over half of the planet's rice yield (Lin et al., 2022). Rice production is currently led by India and China. Brazil and the United States are important non-Asian producers and Italy is the largest contributor in Europe. Production has been increasing in Asia due to advancement in crop varieties and agricultural technology (Gnanamanickam, 2009). For example, careful rice breeding at the International Rice Research Institute produced Miracle Rice, also known as IR8, in 1968 which required minimal fertilizer and produced yields of up to 10 times previous varieties. This variety and others, such as IR5 had significant impacts on food availability in Asia, which in turn improved qualities of life for a large part of the world's population (Cullather, 2004).

80% of rice is grown on small-scale farms and stays in neighboring markets. Small farms are closely linked with local ecosystems and are influential on the flora and fauna that reside within.

The FAO Director-General points out that one billion families in Asia, Africa, and the Americas are heavily reliant on rice farming as their primary source of income. Therefore, careful and sustainable increases in production can greatly improve the lives of growers, women, children, and the lowest income families (N.A., 2003). However, the detrimental effects of climate-induced sub-par growing conditions can endanger those gains. The timing of annual planting dates will become unsuitable, growing windows will be shortened, and locally derived varieties of crops will be less productive, leading to crop failure and market shortages. The resulting losses will in turn be severely detrimental to the quality of life of households who are relying on it as a source of income (Gitz et al., 2016). This often leads to fewer resources for purchasing other necessary items, too. If market availability of rice is reduced in quantity or if it is sold at an increased price due to shortages, residents will have to make substitutions with inferior, cheaper, or more processed goods – often accompanied by less nutritional value. Fresh produce and higher quality protein will be outside the reach of those households (Krishnamurthy et al., 2014; Nelson et al., 2018).

In cases where rice remains a viable commodity, research has shown that the increases in CO_2 levels associated with climate change have a negative impact on both total yield and nutritional value of crops. Studies conducted by the Intergovernmental Panel on Climate Change (IPCC, 2023) acknowledge that crops may increase biomass production due to elevated CO_2, which often leads to stimulation of plant growth in cereal crops, but more is not always better. These plants often have lower levels of vitamins and minerals such as iron, and vitamin A (Krishnamurthy et al., 2014; Myers et al., 2014; Smith et al., 2017). In addition, plants are more likely to be major sources of protein in regions where animal protein can be prohibitively expensive. Plant protein plays a notable role in satisfying global protein consumption goals and is found in many crops including rice

(Medek et al., 2017) Protein levels in rice, wheat, barley, and potato were reduced by up to 7.6%, 7.8%, 14.1% and 6.4% respectively when grown with increased CO_2. Reduced access to plant protein could put 148.4 million people in danger of not meeting their dietary requirements.

There have been suggestions that warmer average temperatures can be of benefit in some regions. In higher latitude areas, warmer temperatures can prolong growing seasons, offer an additional season of production, or allow new crop varieties to be included in an annual rotation (Uleberg et al., 2014; Chaloner et al., 2021; Saunders, 2021). Increased CO_2 can bring benefits as well. In South Korea, the impacts of greenhouse gas emissions were modeled (Park et al., 2018), delivering predictions that rice yields could increase using certain models (Riahi et al., 2011). (Deryng et al., 2011) reported that higher concentrations of CO_2 can improve biomass accumulation and water use efficiency, specifically by reducing transpiration, therefore limiting water loss. But this was more noticeable in wheat than rice. Unfortunately, these benefits are offset by the other interactions between CO_2, increasing temperatures, and local ecosystems. Altering these conditions often favors pests, including weeds, herbivores, and disease-causing microorganisms (Gitz et al., 2016). Taking advantage of increased CO_2 for an overall net gain will require development of specially adapted varieties, soil inputs, and careful pest management (Uleberg et al., 2014).

While we have spent centuries developing varieties of rice that are reliable and hardy (Ladha et al., 2000; Sweeney and McCouch, 2007; Maclean et al., 2013; Cordero-Lara, 2020), we must acknowledge that the challenges that come with rice farming have and will continue to change over time, and that our attention to these difficulties must follow closely alongside them.

Impacts of Climate Change on Cell and Molecular Biology of Crops/Rice

We know that plants respond to the conditions of their environment and that those responses and subsequent adjustments are necessary for coordinating physiological processes with optimal conditions. Plants sense temperature by sending environment-induced signals along a series of downstream pathways (Ruelland and Zachowski, 2010). In some cases these may share overlapping

signaling components with the *FLC* pathway for vernalization, temperature-induced flowering, and circadian regulation (Penfield, 2008). Light and temperature signaling seem to overlap to a certain degree, which may account for the fact that both factors are strongly associated with temporal/seasonal changes. Despite a number of studies investigating these signaling pathways, plant temperature sensors have not been fully characterized (Penfield, 2008).

The previously mentioned environmental conditions fall under the umbrella of abiotic stimuli. Plants differentiate between abiotic and biotic stimuli, or that caused by living organisms such as birds, bugs, and microbes with specialized cellular receptors. These receptors initiate activity along signaling pathways and they rely heavily on plant hormones to do so. Plant hormones, or phytohormones, are small molecules that travel between cells and tissues to operate the switchboard of possible physical responses. Hormones simultaneously control the timing of plant growth, maintenance of essential internal processes, and responses to the surrounding environment (Bari and Jones, 2009). This leads to overlaps and tradeoffs between signaling for events like environmental stress and signaling for processes like photosynthesis and yield production. This overlap is designed to benefit the plant – i.e., germination is timed to avoid drought. However, it can cause complications too. For example, when plants experience multiple stresses simultaneously, such as heat and pathogen stress, they often struggle to respond fully to both (Cohen et al., 2019).

The roles of hormones in plant physiology are complex. Jasmonic acid is produced in response to herbivory and can also be associated with stomatal opening. It also facilitates expression of some disease resistance genes (Ruan et al., 2019). Salicylic acid is important for systemic acquired resistance against pathogens and is also released when physical damage has been done to a plant (Koo et al., 2020). Abscisic acid was originally credited with signaling leaf abscission, but expression has since been reported as a controller of seed dormancy and as a response to heat, drought, or

other forms of abiotic stress (Chen et al., 2020). Ethylene controls fruit ripening, cytokinin controls cell division and auxins control stem morphology (Cholodny, 1928; Went, 1928; Werner et al., 2001; Iqbal et al., 2017). To further complicate their roles, hormones can induce and suppress each other. Jasmonic acid and salicylic acid can be antagonistic, the presence of one may decrease the presence of the other. This may shed some light on the difficulties plants face while trying to prioritize stresses, even though both stresses can, and often do, occur alongside each other in natural environments.

Plant Disease Response Strategies

Some of the same mechanisms that allow plants to adjust to their physical environment also signal their response to microorganisms. Microorganisms share local ecosystems with plants, above and below ground. Some of them are beneficial, even essential to plant survival. But others can be a serious threat to a plant's wellbeing. It is just as essential for plants to respond to these biotic threats as it is with abiotic ones, and similarly, they have developed precise response signaling to counteract them.

Plant-pathogen responses occur as soon as bacteria encounter the plant. The outer surfaces of leaves are covered in a layer called the waxy cuticle, designed to create an external barrier to unwanted contact. Some organisms can penetrate this layer, or seek out natural openings such as stomata, hydathodes, or openings from wounded or broken tissues. When bacteria make their way into the plant, they are met with a series of unpleasant chemicals. Receptors on cells in the plant sense the presence of bacteria, viruses, or fungi and release a series of unpleasant responses, such as reactive oxygen species or secondary metabolites. This is called PTI, or PAMP triggered immunity. Depending on the biotrophic or necrotrophic nature of the pathogen, the plant cell will induce jasmonic or salicylic acid that initiates defense signaling. If this response isn't enough to deter the pathogen, the plant switches over to a more specialized response system called ETI, or effector

triggered immunity – which will be expanded on in a later section after introducing effectors as "BB effector strategy".

The Danger of Disease: Bacterial Blight

When discussing diseases associated with rice farming, one stands out for its potential to disrupt production: bacterial blight, or BB. In rice, bacterial blight can cause yield losses of up to 70% which can be heavily detrimental to growers and the community markets that rely on them (Reddy, 1979; Ou, 1985). In India for example, yield losses ranged from as low as 6 to up to 60% in most of the rice growing states (Srivastava, 1967; Gnanamanickam, 2009) but could be as severe as 74.20%. BB was originally observed in Kyushu, Japan around 1884 (Fiyaz et al., 2022) it was formally classified in Japan in the 1920s and renamed multiple times since (Mew et al., 1993). It has subsequently been observed in continental Asia, southeast Asia, Africa, and Australia. The global motility of BB is part of what makes it so significant and damaging to growers. BB has been spotted as far south as 20◦S in Australia and as far north as 58◦N in China (Mizukami and Wakimoto, 1969; Ou, 1985; Mew, 1987; Mew et al., 1993; Gnanamanickam et al., 1999). BB spreads through waterbased methods of transmission, like shared irrigation water and splashing of raindrops between infected plants and nearby healthy plants (Huang and De Cleene, 1989), making it a serious threat in Asia during the rainy season. It also lingers in paddies after repeated seasons of use. It has been estimated that millions of hectares of rice are severely affected throughout equatorial and temperate regions around the world (Savary et al., 2012).

Bacterial blight in rice is caused by the bacterium *Xanthomonas oryzae* pv. *oryzae*, abbreviated as *Xoo*. *Xoo* impacts yield by reducing the number of panicles and associated grain weight. Discovered in Japan in 1884 (Ezuka and Kaku, 2000), *Xoo* prefers an ambient temperature of approximately 28C (82.4F) and is well-suited to many rice growing regions and thrives in warm, humid conditions. *Xoo* is a gram-negative bacterium named after its distinct yellow extracellular polysaccharide layer, resulting in mucoid colonies when plated. It enters plants through openings in the leaves such as

hydathodes and through broken or damaged tissues (Mew, Mew, & Huang, 1984). *Xoo* is a vascular, systemic pathogen that colonizes within the xylem of the plant that seeks sugars for growth and blocks vascular tissue as it multiplies (Mew, 1987).

Infected rice leaves display contrasting grey-tan lesions along the leaf, following internal veins (Mew et al., 1993). Tissue necrosis occurs from the tip of the leaf toward the base (Yang and Bogdanove, 2013). Bacterial ooze can leak from infected leaves in humid climates and contribute to the spread of the bacteria by falling into irrigation water and be transmitted further through the splashing of rain and wind. Although the presence of water might account for the bacteria's ability to spread, the impact of standing and splashing water has been challenged, citing that *Xoo* survives for only 15 days in paddy water (Mizukami and Wakimoto, 1969).. BB can impact all stages of plant growth with distinct phases of blight on tillered leaves and a seedling stage called kresek. This latter phase is the most severe, with chlorosis on the entire plant followed by wilt and plant death, but infection at late tillering stages is more common (Gnanamanickam, 2009). Yield is most impacted when infection coincides with panicle development and early flowering. Infection by *Xoo* can be confirmed with several methods such as PCR (Lang et al., 2010; Lang et al 2014) or monoclonal antibodies (Mew and Alvarez, 1994). *Xoo* also survives on alternative host species like *Leersia* and others such as *Leptocloa chinensis* and *Cyperus rotundus* (Gnanamanickam, 2009), and the role that these alternate hosts have played in evolution of *Xoo* has been discussed(Lang et al., 2019). Additionally, the bacteria can still survive in drier environments, like leftover field litter and stored seeds and can become active again in wet conditions.

The impact of BB throughout the global community demands solutions that are cost effective and have minimal impacts for local ecosystems. Treatment of BB is limited to management of irrigation sources, optimal soil nutrition, and minimized field litter (Leung et al., 2003; Mew et al., 2004), and chemical solutions have limited success (Devadath, 1989; Gnanamanickam et al., 1999; Chaudhary

et al., 2012). This leaves growers primarily reliant on rice lines carrying resistance genes, or *R* genes, for long-term protection. Generally, BB disease management is based on minimizing multiplication of *Xoo* in plants through planting of disease resistant cultivars, thereby limiting accumulation in field litter and local water sources.

BB effector strategy

When bacteria encounter a host, several things happen. PAMP-triggered immunity, or PTI, as described above, is activated in the plant, which relies on the activation of membrane receptors to initiate a series of defensive responses (Fig.1a) inside the plant cell (Fig.1a; Gómez-Gómez and Boller, 2002; Jones and Dangl, 2006). But when a plant defense mechanism is activated, bacteria often evolve a strategy to overcome it. This results in a tug of war of defense responses between host and pathogen, with each organism attempting to take control back from the other. Over time, this has led to highly specific responses from both the host and the pathogen, in which both participants have evolved molecular pathways that target one another.

To overcome the defenses associated with PTI, *Xoo* directly injects effector proteins into plant cells with a syringe-like mechanism called a type III secretion system, or T3SS.(Coburn et al., 2007) This specialized protein complex spans the inner and outer membrane of the bacterium (Coburn et al., 2007). One kind of *Xoo* effector protein, the transcription activator-like (TAL) effectors, target nuclear DNA and bind to promoters of susceptibility and resistance genes, mimicking transcription activators in the host(Hopkins et al., 1992; Yang et al., 2000). Some of the TAL effectors shift gene transcription to favor bacterial virulence, leading to progression of the disease in the host(Doyle et al., 2013; Teper et al., 2023).

Plant *R* gene responses

In response to the bacterial effector strategy, plants have developed a defense response called effector triggered immunity, or ETI. In a few cases, plants have evolved promoters in resistance genes, or *R* genes, that are difficult for the bacteria to distinguish from their original targets in

susceptibility genes (Luo et al., 2021). The "decoy" promoters have protein binding affinities that are similar to or greater than the corresponding *Xoo* targets. This causes TAL effectors to bind to the promoter of the decoy *R* gene instead of their original target, which in turn activates *R* gene pathways for hypersensitive response, or HR. HR produces a distinct phenotype in the leaf, with dark brown necrotic tissue at the intersection of healthy and infected cells.

HR is a disease containment strategy in plants that triggers localized cell death to minimize the bacteria's ability to spread and cause additional damage throughout the rest of the plant (Hopkins et al., 1992; Leach et al., 1993). Here, plants inadvertently use bacterial virulence strategies against them, by activating plant defense mechanisms instead of the traits that would benefit bacteria (Luo et al., 2021). In some cases, TAL effectors specifically activate a class of *R* genes in rice called executor *R* genes, by acting as gene transcription switches, turning on genes in pathways that direct plant resources toward self-preservation.

One such executor R gene *is Xa7* which has evolved to function in the presence of a corresponding effector protein from *Xoo* called AvrXa7(Fig. 1c). The *Xa7* effector binding element (EBE) in its promoter bears a strong resemblance to that of a promoter in a susceptibility gene targeted by *Xoo*, called *SWEET14*. This is exactly what makes it an effective decoy: close enough to original EBE to attract AvrXa7, but with a stronger binding affinity (Chen et al., 2021; Luo et al., 2021). *Xa10*, which is also an executor *R* gene, initiates HR in the form of ER membrane bound hexamers that disrupts cellular calcium homeostasis, resulting in cell death and the slow of BB progression (Tian et al., 2014a)

However, *R* genes come in many flavors. Some *R* genes function as receptor-like kinases, or RLKs, such as *Xa3* and *Xa21*. RLKs are a type of pattern recognition receptors (PRRs) that perceive unique protein ligands in the cell membrane or cytoplasm (Fig.1 b), and in turn initiate defense signaling pathways (Lee et al., 2009). PRRs such as these are an important component of PTI. (Song

et al., 1995; Sun et al., 2004a; Xiang et al., 2006a; Wang et al., 2015). Other *R* genes are the result of nucleotide polymorphisms that reduce or interrupt the function of a gene or its promoter. *xa5* is one such modified gene (Fig 1d), in which the shape of a subunit of a transcription factor for a susceptibility gene is modified, thus reducing the susceptibility that would otherwise result from the binding of TAL effectors with their corresponding effector binding elements (Iyer-Pascuzzi et al., 2008; Huang et al., 2016a; Yuan et al., 2016). *R* genes provide diverse strategies for plant defense against many races of Xoo and have remained a significant strategy to minimize damage of BB.

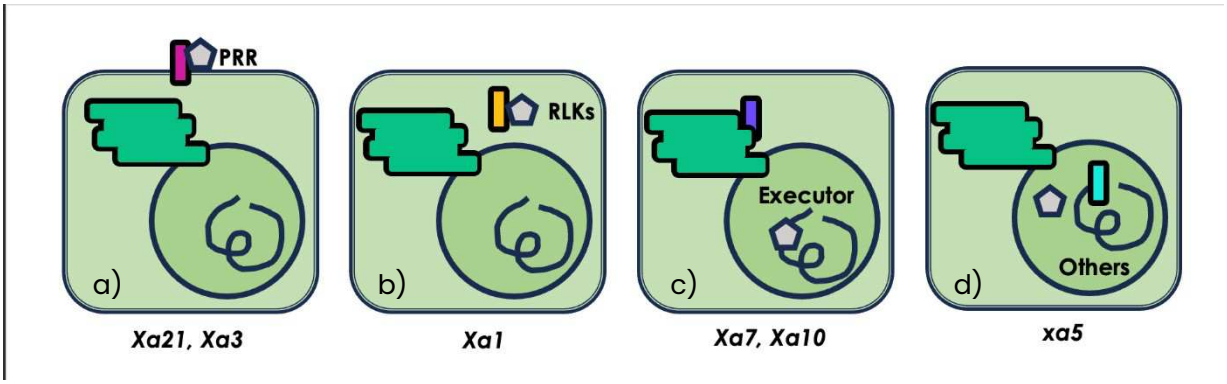

Figure 1.1: Locations of rice resistance proteins within the cellular environment. a) PRRs: first line of defense, exterior of cell, b) RLKs: response to injected effectors, inside cell cytoplasm, c) Executors: response to injected effectors, within the nucleus, d) Others: polymorphisms in susceptibility, in the nucleus. These responses occur in different parts of the cell, and thus impact host cell physiology in different ways. Some, including executor genes, initiate HR to minimize the spread of *Xoo*.

Resistance genes are often introgressed into elite varieties from wild cultivars. For example, *Xa21* from the wild rice *O. longistaminata* was introduced into IR24, to construct the near-isogenic line IRBB21 (Khush et al., 1990). About forty-six major BB resistance genes have been identified, and around 15 have been cloned (Mew et al., 1992; Zhang et al., 1998; Fiyaz et al., 2022; Yang et al., 2022). Single-gene resistance has been effective in the fight against BB, but it breaks down with the emergence of new *Xoo* strains (Mew et al., 1992). For example, *Xa4* was a strong resistance gene against *Xoo* before *Xoo* eventually became virulent to it Marker-assisted breeding has facilitated introduction of *xa5*, *Xa13, Xa21*, and others into more than 70 rice varieties and continues to make

R genes, particularly when pyramided together, a robust strategy for reducing the impact of BB (Fiyaz et al., 2022).

Table 1: The impacts of temperature on *R* genes for bacterial blight and other types of *R* genes

***R* gene**	**Type (if known)**	**Function**	**Crop**	**Disease**	**Temperature response**	**Source**
Xa1	NBS-LRR	Induced by trauma, infection		Bactieral Blight		(Yoshimura et al., 1998)
Xa2/Xa31	NBS-LRR	Allele of *Xa1*		Bactieral Blight		(Ji et al., 2020)
Xa3/Xa26	LRR-STK	Age specific/dose dependent; LRR receptor kinase-like protein;	Rice	Bacterial Blight	Increased susceptibility (high temp)	(Sun et al., 2004b; Cao et al., 2007a; Li et al., 2012; Xiang et al., 2006b; Webb et al., 2010; this study)
Xa4	WAK-STK	Cell wall associated kinase; cell wall reinforcement	Rice	Bacterial Blight	Increased susceptibility (high temp)	(Horino et al., 1982; Sun et al., 2003; Hu et al., 2017; Webb et al., 2010; Zhao et al., 2022)
xa5	recessive	Small subunit of transcription factor IIA; dampens TALE function generally	Rice	Bacterial Blight	Increased susceptibility (high temp)	(Blair et al., 2003; Iyer-Pascuzzi et al., 2008; Huang et al., 2016b; Yuan et al., 2016; Webb et al., 2010; this study)

***R* gene**	**Type (if known)**	**Function**	**Crop**	**Disease**	**Temperature response**	**Source**
Xa7	executor genes from TALs,	*SWEET* decoy, binding target to protect *S* *Unique, 113 AA membrane protein; Executor gene*	Rice	Bacterial Blight	Increased resistance (high temp)	(Chen et al., 2021; Luo et al., 2021; Webb et al., 2010; Wang et al., 2021; Liu et al., 2020; this study.)
xa8	recessive			Bacterial Blight		(Vikal et al., 2014)
Xa10	executor	ER protein, disrupts Ca+ ions, Hexamers localized in ER; associated with CA++ depletion; executor function	Rice	Bacterial Blight	Sustained resistance (high temp)	(Webb et al., 2010; Tian et al., 2014b; this study)
Xa11	dominant			Bacterial Blight		(Goto et al., 2009)
Xa12	dominant			Bacterial Blight		(Si-yuan et al., 2006)
xa13	recessive	-membrane receptor? SWEET protein, Cu+ transport		Bacterial Blight		(Chu et al., 2006)
Xa14	NBS-LRR	Allele of *Xa1*		Bacterial Blight		(Ji et al., 2020)
xa15	recessive			Bacterial Blight		(Nakai et al., 1988)
Xa16	dominant			Bacterial Blight		(Ogawa, 1993)

R gene	Type (if known)	Function	Crop	Disease	Temperature response	Source
Xa17	dominant			Bacterial Blight		(Ogawa, 1993)
Xa18	dominant			Bacterial Blight		(Ogawa, 1993)
xa19	recessive			Bacterial Blight		(Taura et al., 1992)
xa20	recessive			Bacterial Blight		(Taura et al., 1992)
Xa21	LRR	Binds to WRKY62	Rice	Bacterial Blight	Increased susceptibility (high temp)	(Khush et al., 1990; Song et al., 1995; this study)
Xa22	dominant			Bacterial Blight		(Lin et al., 1996)
Xa23	executor	Protein similar to Xa10		Bacterial Blight		(Wang et al., 2015)
xa24	recessive			Bacterial Blight		
xa25	recessive	Dominant at adult stage, sugar transporter		Bacterial Blight		(Liu et al., 2011)
Xa27	executor	Novel protein, local defense, possible cell wall thickening		Bacterial Blight		(Gu et al., 2005)
xa28	recessive			Bacterial Blight		(Lee et al., 2003)
Xa29	dominant			Bacterial Blight		(Tan et al., 2004)
Xa30	dominant			Bacterial Blight		(Xuwei et al., 2007)
xa31	recessive			Bacterial Blight		(Wang et al., 2009)

R gene	Type (if known)	Function	Crop	Disease	Temperature response	Source
xa32	recessive			Bacterial Blight		(Zheng et al., 2009)
Xa33				Bacterial Blight		(Kumar et al., 2012)
xa33(t)				Bacterial Blight		(Korinsak et al., 2009)
xa34	recessive			Bacterial Blight		(Ram et al., 2010)
Xa35	dominant			Bacterial Blight		(Guo et al., 2010)
Xa36	dominant			Bacterial Blight		(Miao et al., 2010)
Xa38	dominant			Bacterial Blight		(Kaur et al., 2005; Cheema et al., 2008)
Xa39	dominant			Bacterial Blight		(Zhang et al., 2015a)
Xa40	dominant			Bacterial Blight		(Kim et al., 2015)
xa41	recessive	Sugar transporter *SWEET14*		Bacterial Blight		(Hutin et al., 2015)
xa42	recessive			Bacterial Blight		(Busungu et al., 2016)
Xa43				Bacterial Blight		(Kim and Reinke, 2019)
Xa44	dominant			Bacterial Blight		(Kim, 2018)
Xa45	NBS-LRR	Allele of *Xa1*		Bacterial Blight		(Ji et al., 2020)
Yr36			Wheat	Stripe Rust	Increased resistance (high temp)	(Segovia et al., 2014)
Yr36			Wheat	Stripe Rust	Increased resistance (low temp)	(Segovia et al., 2014)

***R* gene**	**Type (if known)**	**Function**	**Crop**	**Disease**	**Temperature response**	**Source**
Sr6			Wheat	Stem Rust	Increased susceptibility (high temp)	(Moerschbacher et al., 1989)
Rlm6			Rapeseed	Phoma Stem Canker	Increased susceptibility (high temp)	(Huang et al., 2006)
N			Tobacco	TMV	Increased susceptibility (high temp)	(Wright et al., 2000)
Cf-4			Tomato	Leaf Mould	Increased resistance (high temp)	(de Jong et al., 2002)
Pi54			rice	Rice Blast	Increased resistance (low temp)	(Madhusudhan et al., 2019)

Breakdown of *R* Gene Function at High Temperature

Heat stress interferes with the responses that plants rely on to resist pathogen invasion. As a result of the complicated nature of hormone-based responses, heat and disease damage are often more severe when they are combined than the instances in which they occur separately (Cohen et al., 2019). Lesions produced from *Xoo* are longer on leaves growing at warmer temperatures (35 °C day/29 night) than at lower temperatures (29C day/ 23C night (Yamada et al., 1979; Horino et al., 1982; Ou, 1985; Ezuka and Kaku, 2000) This means that yield damage from some bacterial pathogens during warmer-than-average field conditions can be more severe. While plant hormones are rapidly activated in response to both heat and disease stress, signaling for heat stress appears to be prioritized, resulting in longer lesions at high temperatures.

Although there is research on the ways in which temperature stress and disease stress separately impact the health of rice, we know much less about the complex internal processing that plants rely

on when they are exposed to multiple forms of stress simultaneously. Many plant pathogens have evolved in their respective ecosystems, side by side with their respective hosts, and have ideal temperature ranges and humidity preferences that may overlap with their host. *Xoo* bacteria thrives in a 25 °C–30 °C (Ou, 1985) climate with high humidity. These are common conditions in rice growing regions, thus allowing *Xoo* and rice to exist side by side. However, as temperatures creep upward, the reduced fitness of plants under heat stress can be enough of an opportunity for pests to overcome their host.

One reason for the increased severity of disease at high temperature is the breakdown of plant disease resistance genes (Cohen and Leach, 2020; Sahu et al., 2020). *R* genes are of the most effective, and therefore important, strategies against bacterial blight. Other approaches to BB management are more costly, unsustainable, or marginally effective. Most rice *R* genes like *xa5* and *Xa3,* have reduced capacity for pathogen defense when combined with heat stress (Webb et al., 2010). At higher temperatures, lesions in rice with these genes were lengthier than those measured at low temperatures, indicating reduced or broken *R* gene functionality However, in a survey of disease phenotypes however, one *R* gene, *Xa7* was shown to function more effectively at high temperatures (Webb et al., 2010).

(Cohen et al., 2017) found that gene expression in *Xa7* plants under heat and disease stress was different from that of plants without the *R* gene of interest under the same stress treatments. ABA signaling, which is usually upregulated in response to both stresses, was downregulated in *Xa7* plants activated for resistance. It is possible that the internal hormonal crosstalk is altered in plants with downregulated ABA, such that prioritization of multiple stresses is handled in a unique manner. ABA and SA interactions could be differentially regulated in *Xa7* (Cohen et al., 2017). We know that combined heat and disease stress signaling creates unique synergies that intensify the symptoms of each stressor to be more severe than if they occurred separately. We do not know if this synergy

results in greater susceptibility signaling or if a buildup of cellular products enhances combined stress phenotypes. One could speculate that by downregulating ABA associated signaling, *Xa7* plants would reduce the phenotypes associated with inputs of heat and disease stress. There are studies that drought also makes rice more susceptible to BB (Dossa 2016). *Xa7* and an unknown gene from African rice, continue to provide resistance during abiotic stresses (Dossa et al. 2016a; Dossa et al. 2017; Webb et al. 2010). Responses to "general" stress were upregulated in plants without *Xa7* and downregulated in plants with *Xa7* (Cohen et al., 2019).

Gaps in Knowledge

It has been observed that when heat and disease stress are combined, symptoms associated with the combination of both stresses are more severe than if they had been observed singularly (Cohen et al., 2019; Balfagon et al., 2020; Desaint et al., 2021; Kim et al., 2022). This suggests that the importance of understanding combined heat and disease stress is of greater urgency than independent models predict. We do not yet know the degree to which the type of *R* gene determines its endurance under increasing temperatures. The reduced lesion lengths observed in *Xa7* have only been observed in rice with that *R* gene (Webb et al 2010). In contrast, other *R* genes, including *R* genes with functions other than executors, were less efficacious at high relative to low temperatures as measured by lesion lengths. A comparison of the *R* gene functions relative to the impacts of high temperatures was not discussed because there was little knowledge of what the *R* genes were at the time of the Webb publication. Further, in prior studies, the impact of higher temperatures on the multiplication of *Xoo* in the plant was not evaluated. Although the higher temperatures associated with increased performance of *Xa7* are not ideal for *Xoo* proliferation, preliminary information from our group shows that the expression TALE is sufficient to activate *Xa7* expression.

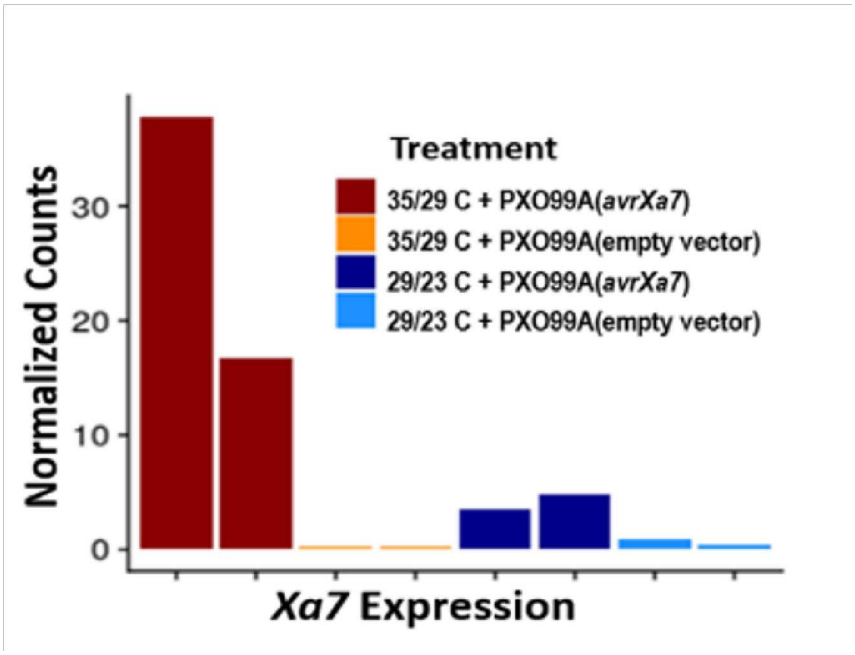

Figure 1.2. Expression of *Xa7* after inoculation with *Xoo* strain PXO99 with and without the effector gene *avrXa7*, at high and low temperatures. The presence of effector AvrXa7 is required for expression of *Xa7* as seen in the columns corresponding to PXO99A(*avrXa7).* In addition, expression of *Xa7* is enhanced at high temperature, as seen in the columns in red. (Alvaro Quintero-Perez, Rene Corral, J.E. Leach, unpublished).

We hypothesize that rice lines with *R* genes other than *Xa7* are more susceptible to bacterial blight at high temperature because of a mechanism involved in *Xa7* expression. We know that *Xa7* is upregulated at high temperatures, even when fewer bacteria are present, which may indicate that heat stress complements or enhances the rate of expression that would be caused by bacterial numbers alone. Heat stress may induce a positive feedback loop that enhances transcription efficiency or binding affinities of effectors from *Xoo.*

Another possibility is that protein accumulation in the nucleus of the host cell improves disease resistance mechanisms that share a signaling pathway with *Xa7*. There is evidence in some plants that nuclear localization of proteins can be induced by changes in temperature. In Arabidopsis, accumulation of the immune receptor protein, SNC1 in the nucleus has been shown to enhance disease resistance phenotypes at high temperature. (Zhu et al., 2010). In tomato, a heat shock transcription factor, HSFA1, accumulates in the nucleus after exposure to heat treatments (Scharf et

al., 1998). Cold temperatures can also influence this. HOS1 in Arabidopsis also relocates from cytoplasm to nucleus to induce inhibition of cold responses (Ishitani et al., 1998).

Alternatively, *Xa7* plants might be more responsive to accumulation of stress-induced ROS. (Cheng et al., 2013) introduce the observation that changes in temperature can induce oscillations between ETI and PTI at 10-23C and 23-32C, respectively. If the optimal temperature range predicts favorable induction of PTI, perhaps stressful temperatures, and associated ROS accumulation, on both the low and high ends of that range might induce ETI, though at the time of writing, the study did not investigate higher temperatures. We know that ROS are important signaling molecules in plants exposed to both heat and pathogen stress (Suzuki et al., 2012; Rejeb et al., 2014; Pandey et al., 2015). We also know that accumulation of ROS is linked to induction of programmed cell death, a broader classification of HR (Laloi and Havaux, 2015). We could speculate that the rapid and effective onset of HR in *Xa7* plants is due to a higher sensitivity in ROS-related response signaling, likely in the ER, where Xa7 localizes (Wang et al., 2021).

This study will determine the impact of high temperatures on the resistance phenotypes that are controlled by other rice BB *R* genes, such as *Xa3, xa5, Xa10,* and *Xa21*. This set of *R* genes was chosen because they can all be induced by the same strain of *Xoo* and because they span multiple types of *R* gene classifications. In addition, this set of *R* genes allows us to build upon previous work that has observed increased disease at high temperatures, and negative impacts of high temperature on *R* gene function. These genes are also in many near-isogenic lines (IRBB lines, approximately 20 at the time of writing (IRRI, 2006a). The use of isogenic materials reduces background noise in our experiments. Further, these genes are deployed in numerous rice lines internationally because they confer resistance to many common strains of *Xoo*.

This study reports the measures of bacterial numbers and lesion lengths in rice containing the previously listed *R* genes at high and low temperatures to assess *R* gene performance and bacterial multiplication in the leaf.

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Chapter Two – The impacts of high temperature on *R* gene efficacy

Introduction:

Rice has long been bred for desirable traits, such as yield and disease resistance, but the challenges and threats that accompany rice farming can change, and our attention to these difficulties must follow accordingly. One well-established threat to rice is bacterial blight, abbreviated as BB, a disease with devastating impact on rice. BB can reduce rice yields by up to 50%, and in rare cases as much as 70%. Recent estimates are more conservative, estimating damage to be 8.9% (Ou, 1985; Cohen et al., 2019; Savary et al., 2019). Symptoms of BB include vertical greyyellow discolorations and tissue necrosis moving from the tip of the leaf toward base (Yang and Bogdanove, 2013). BB in rice is caused by the bacterium *Xanthomonas oryzae* pv. *oryzae*, subsequently abbreviated as *Xoo*. *Xoo* is well-suited to many rice growing regions, and thrives in warm, humid conditions at approximately 28C (82.4F). *Xoo* can be transmitted in irrigation water and through seed stocks. The bacteria enter the plant through peripheral hydathodes or wounds on leaves (Gnanamanickam et al., 1999). Reduction of *Xoo* in rice is limited to management of irrigation sources, optimal soil nutrition, and minimized field litter (Leung et al., 2003; Mew et al., 2004), while chemical applications have limited success (Devadath, 1989; Gnanamanickam et al., 1999;

Chaudhary et al., 2012).

A more recent threat to rice is temperature stress, which can decrease rice yields further (Xu et al., 2020; Su et al., 2023). Each 1 °C of increased nighttime temperature reduces rice grain yield by 10% (Peng et al., 2004). In addition, when both disease stress and temperature stress are combined, development of BB disease symptoms are more severe (Yamada et al., 1979; Horino et al., 1982; Ou, 1985; Ezuka and Kaku, 2000). In trials of select rice varieties, symptomatic lesions were longer at warm season temperatures (35 °C/29 °C) than at cooler season temperatures (29°C /25 °C). Rice plants relying on BB resistance genes (*R* genes) as a defense strategy show reduced resistance at high temperatures in lab experiments (Horino et al., 1982; Webb et al., 2010). At field scale, a reduction in

resistance in one or a few plants can become an opportunity for *Xoo* to multiply and spread between neighboring rice, contributing to further crop loss.

Interestingly, (Webb et al., 2010) found that compared to near-isogenic rice lines containing *R* genes (*Xa3, Xa4, xa5, Xa7,* and *Xa10*), one *R* gene, *Xa7*, functioned more effectively at high temperatures compared to lower temperatures. *Xa7* is characterized as an executor *R* gene (Chen et al., 2021; Luo et al., 2021) (Table 1) which activates an ETI response, typically culminating in a hypersensitive response (HR), when the corresponding bacterial TAL effector enters the nucleus and binds to the promoter of the *R* gene (Zhang et al., 2015b).

For the executor gene *Xa7*, an *Xoo* effector called AvrXa7 (Hopkins et al., 1992; Yang et al., 2000) must be present to trigger *R* gene expression. When *Xoo* injects AvrXa7 effectors into the host plant cell, the effector travels to the cell nucleus where it can bind to either the effector binding element (EBE) of the promoter of *Xa7* or the EBE in the promoter of a susceptibility target of AvrXa7, the *OsSWEET14* gene, which will activate resistance or susceptibility, respectively. Studies of binding affinities for the two promoters suggest that the EBE in *Xa7* has evolved to have a higher binding affinity than the EBE in *SWEET14*, thereby allowing *Xa7* to act as a decoy to reduce effector binding to the susceptibility target (Wang et al 2021; Luo et al 2021). When the effector AvrXa7 binds to *Xa7*'s promoter, it will act as a transcription activator for an HR (Chen et al., 2021; Luo et al., 2021). When the HR is activated, infected cells will die off, creating a barrier of inhospitable tissue to slow the spread of the pathogen in the leaf (Agrios, 2005). The HR produces a distinctive phenotype in leaves composed of reduced disease lesions and a dark brown band of discoloration. In plants with stronger resistance mechanisms, these brown lesions remain short and compact. In plants where resistance is delayed or absent, diseased tissue phenotypes will spread over a greater area, including longer, light brown lesions, chlorosis, and leaf curling following the spread of the

pathogen. *Xa10,* also an executor *R* gene, induces an HR by localizing Xa10 to the endoplasmic reticulum and disrupting the balance of calcium ions within the cell (Tian et al., 2014a).

Other BB *R* genes, such as *Xa3* and *Xa21,* are receptor kinase-like (RLK) proteins, which perceive bacterial effectors and activate defense pathways (Song et al., 1995). In receptor-like kinases (RLKs) like *Xa3* and *Xa21*, pattern recognition receptors (PRRs) in the cell membrane bind with RaxX proteins, a sulfated protein from *Xoo* (Pruitt et al., 2015; Wei et al., 2016; Ercoli et al., 2022). RaxX initiates signaling to protein domains in the cytoplasm that activate immune responses in the host, termed pathogen-associated molecular pattern triggered immunity, or PTI. (Song et al., 1995; Sun et al., 2004a; Xiang et al., 2006a; Wang et al., 2015).

Yet another type of *R* gene is typified by *xa5*. *xa5* is an allele of the *Xa5* gene, which codes for a transcription factor subunit, TFIIAy5 (Iyer-Pascuzzi et al., 2008). The protein encoded by the dominant allele *Xa5* is a contact point between TALEs and the transcriptional machinery (Huang et al 2016). The product of *xa5* harbors a single amino acid substitution which interferes with its interaction with diverse TALEs, and thereby reduces the ability of TALEs to activate their target S genes (Iyer-Pascuzzi et al., 2008; Huang et al., 2016a). The resistance provided by *xa5* is hypothesized to occur when *S* gene expression, and, by inference, sucrose leakage, falls below a threshold level (Huang et al. 2016).

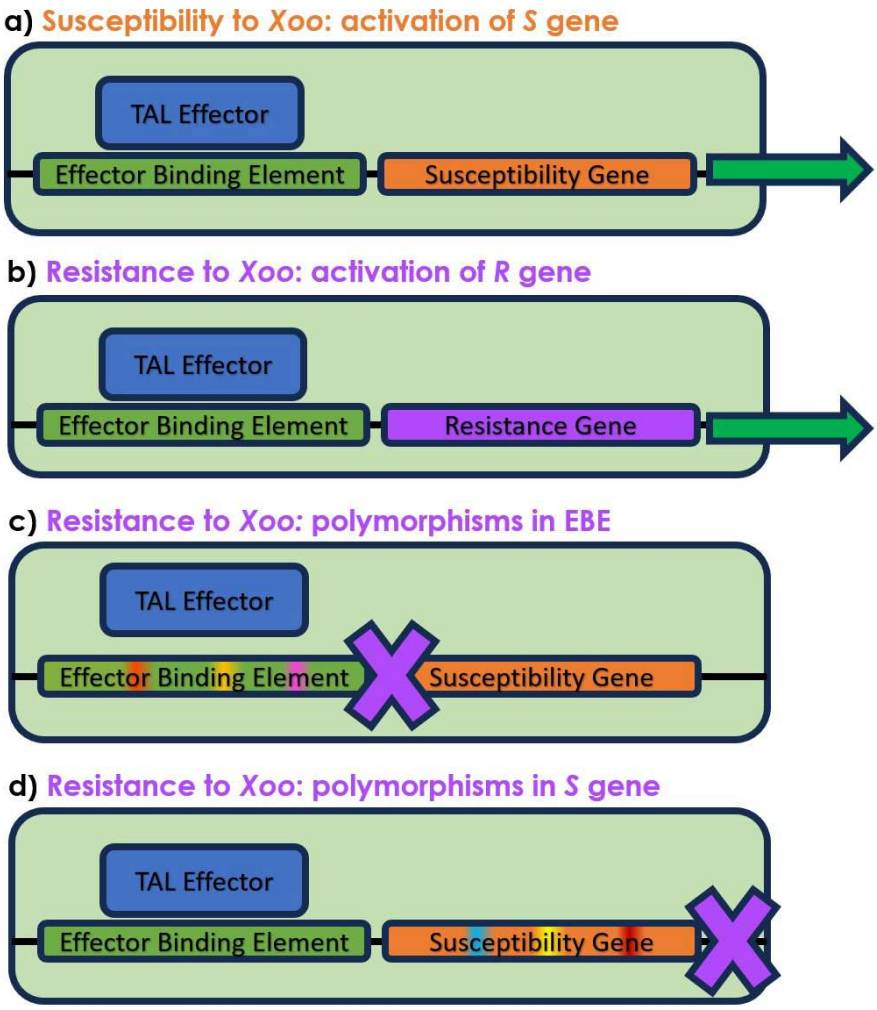

Figure 2.1: Outcomes of TAL effector binding in the nucleus of the rice host: a, b) Transcription activator-like effectors (TALs) may bind successfully to the effector binding element (EBE) in the promoter of either a susceptibility or resistance gene, resulting in the activation of the corresponding response in the plant. c) TALs may be unable to bind to an EBE with polymorphisms in the local sequence, preventing activation of the downstream susceptibility gene. d) Alternatively, TALs may bind to a promoter that activates a susceptibility gene containing polymorphisms, resulting in failed induction of susceptibility in the host.

Why BB is more severe in rice at high temperatures (above 35C) is unknown. Previous literature states that increased bacterial numbers are associated with increased disease severity (Barton-Willis et al., 1989; Verdier et al., 2012). Yet the way that high temperatures impact bacterial multiplication *in planta* is not yet known. Optimal temperatures for *Xoo* multiplication are reported to be approximately 28C, *in vitro* (Saddler and Bradbury). Thus, it would seem that higher environmental

temperatures might impair bacterial multiplication *in planta*. However, studies to determine if increased temperatures provide an advantage to the pathogen or the host are lacking. In this study, we evaluate bacterial numbers *in vitro* and *in planta* to assess the impact of temperature on bacterial numbers to characterize the *Xoo*-rice interactions at high temperature.

The Webb study (Webb et al 2010) suggested that several *R* genes might lose efficacy at high temperatures, but this observation was based only on lesion lengths, with no assessment of bacterial numbers during the interactions. Here, I expand our understanding of *R* gene functional classification and degree of efficacy under heat stress by studying different *R* gene classes, e.g., executor, RLK, and a loss-of- or reduced-susceptibility *R* gene. Disease is more severe at high temperatures (Yamada et al., 1979; Horino et al., 1982; Ou, 1985; Ezuka and Kaku, 2000) and not all *R* genes are equipped to make up for increased severity at high temperatures (Webb et al 2010). However, we do not know how loss of *R* gene function with high temperatures will impact bacterial numbers in the plant, nor do we know if all *R* genes will lose function equivalently. We also do not know the degree to which individual *R* gene performance is impacted at high and low temperatures. We hypothesized that final bacterial numbers and lesion lengths would be higher in rice lines with no *R* genes or with most *R* genes at high temperature regimes due to loss of *R* gene efficacy, but that the extent of increase in disease severity would vary depending on the *R* gene (function).

Results:

Lesion lengths in rice with no *R* gene at high and low temperature regimes

In this study thresholds for the definition of susceptibility were set at lesion lengths of 5 cm (50mm) or greater and $5x10^7$CFU/leaf or higher bacterial numbers, based on methods developed at IRRI (IRRI, 2006b) and previous studies (Barton-Willis et al., 1989), respectively. However, it is important to note that we used younger plants (21 day old; 2-3 leaf stage) that were propagated in growth chambers in these studies, while the previous work is based on older plants (30-40 days past sowing;

4-5 leaf stage; Barton-Willis et al., 1989) grown in either greenhouses or the field. Thus, in many cases, the extent of lesions in this study were more pronounced than if they would have occurred in older plants.

Lesions in *Xoo*-inoculated IR24 rice (no relevant *R* gene) confirmed patterns previously described in other studies, that is, that high temperature (35C day 29C night) growth conditions result in longer lesions as early as 8 days post inoculation (DPI) (Fig. 2.1). Lesion lengths of 5 cm or greater were evident at 8 DPI at high temperatures and expanded more quickly at high relative to low temperature regimes. Lesions exceeding 12 cm developed at high temperature by 12 DPI, as opposed to lesions averaging 7 cm in length at low temperature by 16 DPI.

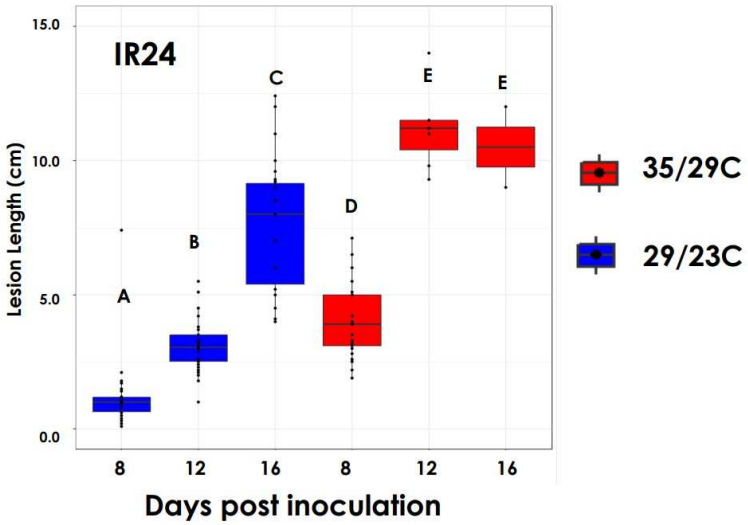

Figure 2.2: Lesion lengths caused by *Xoo* infection developed more quickly and were longer at the high temperature regime. In IR24 rice with no relevant *R* gene, lesions of *Xoo* PXO86 were measured at high (35/29C) and low (29/23C) temperature regimes at 8, 12, and 16 days post inoculation. In the high temperature treatment, lesions surpassed the threshold for resistance (5 cm) by 12 DPI, earlier than observed in low temperature treatment (16 DPI). Letters indicate significant differences at *P*-value of less than 0.05 as determined by post-hoc Tukey adjusted pairwise comparison. The experiment was performed 3 times, with 10 replications per treatment per timepoint.

Optimal temperatures for bacterial multiplication *in vitro*

The optimal temperatures for growth of *Xoo* strain PXO86 are around 27-29C (Fig 2.3). Above (31C) and below (25C) those temperatures, bacterial multiplication slowed, with the final population

size reduced relative to the population at optimal temperatures (Fig. 2.3). Extrapolating these data to temperature conditions in the rice leaf, one could predict that increased temperatures may reduce bacterial multiplication in the plant delaying onset of symptomatic phenotypes.

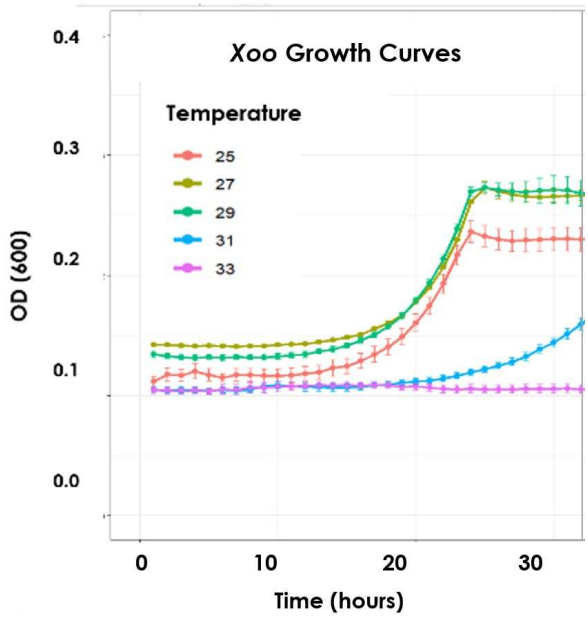

Figure 2.3. In vitro growth curves of *Xoo* strain PXO86 at 25, 27, 29, 31, and 33C. Bacterial multiplication was reduced at temperatures at ≥ 31C or ≤ 23C. Data are based on 25 replications per timepoint per temperature.

Bacterial numbers in inoculated rice leaves at high and low temperature regimes

In rice with *Xa7,* bacterial numbers increased at low temperatures from 8 DPI to 16 DPI by an order of magnitude, from 5 x 10^6 to 2 x 10^7 CFU/leaf. However, at high temperatures, bacterial numbers initially increased to higher numbers at 8 DPI, but subsequently, multiplication declined, with steadily decreasing numbers at 12 and 16 DPI (Fig. 2.4). *Xa7* was more effective at restricting bacterial numbers at high temperature than at low temperature and out of the genes tested, performed best overall.

Xa10 was nearly as effective as *Xa7,* with bacterial numbers reaching their maximum by 8 DPI

(approximately 10^6 CFU/leaf) at low temperatures and remaining at that level through 16 DPI. At the high temperature regime, bacteria in plants with *Xa10* steadily increased through 16 DPI, approaching $5x10^7$ CFU/leaf (Fig. 2.4).

Similarly, plants with *xa5* and *Xa21* exhibited restricted bacterial numbers, between 10^7 and 10^8 CFU/leaf at low temperatures (Fig. 2.4) and *xa5* produced moderately higher bacterial numbers than *Xa21*, peaking at about 7 x 10^7 CFU/leaf. At high temperatures, bacterial numbers in *xa5* were slightly higher than *Xa2*1, with only *Xa3* displaying further reductions in resistance under both temperature treatments.

IR24 and *Xa3* plants were the most susceptible to disease with the largest increases in bacterial numbers occurring at 8 DPI and the earliest leaf senescence occurring by 12 DPI (Fig. 2.4). At a threshold of $5x10^7$ CFU/leaf (resistance), IR24 and *Xa3* were more susceptible to disease at high temperatures as seen in the rapid increase in bacterial numbers by 8 DPI, earlier than in other *R* genes. In *Xa3* plants, bacterial numbers increased rapidly and significantly at both low and high temperatures, suggesting that the *R* gene is not effective against *Xoo* under the conditions used in these experiments. *Xa3* has been rated as conferring moderate resistance and expression of resistance is dependent on the age of the plant at the time of inoculation (Cao et al., 2007b). It is possible that resistance had not developed in the young plants used in these experiments. In rice lines with effective *R* genes, bacterial multiplication was restricted under ideal rice growing conditions. However, at the higher temperature regime (35C day 29C night), bacterial numbers varied, indicating *R* gene effectiveness varies at the two temperature regimes. It is important to note that lesions expanded to the entire leaf length as early as 12 DPI in high temperature interactions (IR24*, Xa3, xa5,* and *Xa21*) and by 16 DPI in low temperature interactions (IR24, *Xa3*, and *Xa21*). The death of the leaves may have inhibited further bacterial multiplication (Fig. 2.4). In a repeat of the experiment, starting inoculum was measured in CFU/leaf as a log higher than that of the

experiment depicted, and lesions and CFU/leaf increased by more significant rates as a result. However, trends in lesions and CFU/leaf supported previous work throughout the duration of the experiment. Although bacterial numbers increased initially in all rice lines, final bacterial numbers were restricted by *xa5, Xa7, Xa10,* and *Xa21* relative to plants with no *R* gene (Fig 2.4), suggesting these *R* genes remained effective to varying degrees.

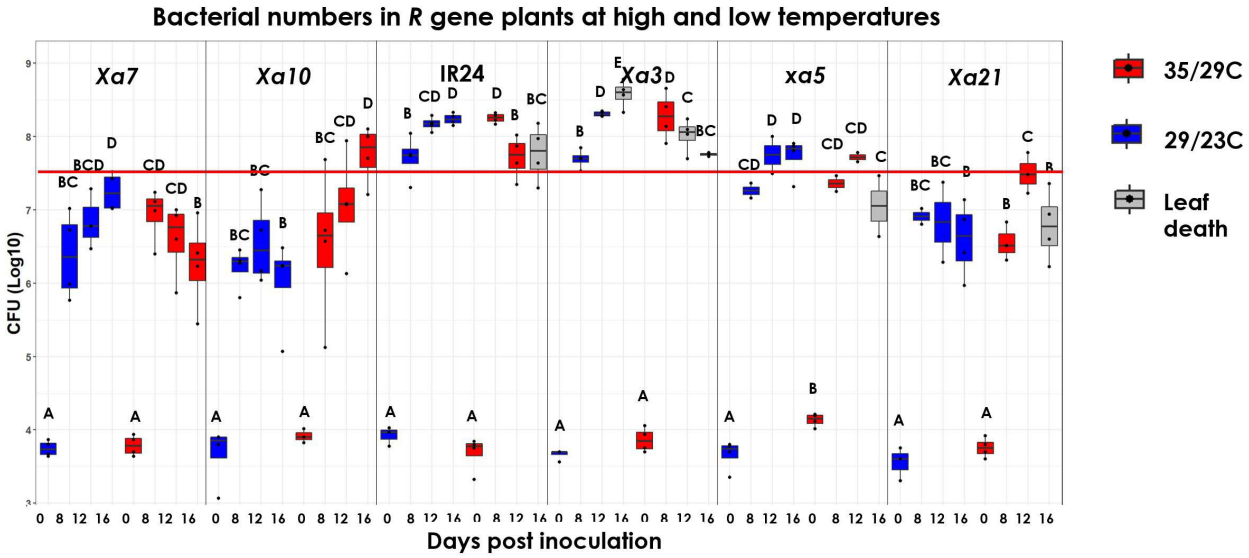

Figure 2.4. Comparison of bacterial numbers in rice lines with different *R* genes and the IR24 control after incubation at low (29 day/23 night) and high (35/29) temperature regimes. Bacterial numbers (CFU/leaf) were assessed at 0, 8, 12 and 16 DPI in leaves of rice with *Xa7*, *Xa10*, *Xa3*, *xa5*, and *Xa21*, after inoculation with *Xoo* strain PXO86. Red line indicates arbitrary threshold for resistance of $5X10^7$ CFU/Leaf. Comparisons are **across genotypes**, with letters indicating differences at P-value < 0.05. The experiment was performed two times with four replications per treatment per timepoint and showed similar trends.

Lesion lengths at high and low temperatures in rice lines with different *R* genes At high temperatures *Xa7* lesions remained compact, approximately 2.0cm, but were still longer than at low temperatures, 0.5cm (Fig. 5). *Xa10* lesions were compact at low temperatures, 0.5cm, but showed a moderate increase at high temperatures to 1.0cm (Fig. 2.5). At high temperatures, IR24 developed long lesions soon after infection, with lesions over 12.0cm at 12 and 16 DPI, while lesions remained restricted at low temperature at approximately 8.0cm by 16 DPI. *Xa3* developed long lesions quickly,

similar to IR24, and although the speed and length of lesions were more extreme at high temperatures, indicating that the *R* gene showed reduced efficacy at low temperatures and even less efficacy at high temperature regimes. *xa5* and *Xa21* lesions increased in length and speed at high temperatures, but to a lesser degree than IR24 and *Xa3*, remaining at approximately 5.0 cm in both temperature treatments. (Fig. 2.5) Overall, *Xa7* and *Xa10* remained functional at high temperatures, and *xa5* and *Xa21* retained some ability to restrict the symptoms of BB.

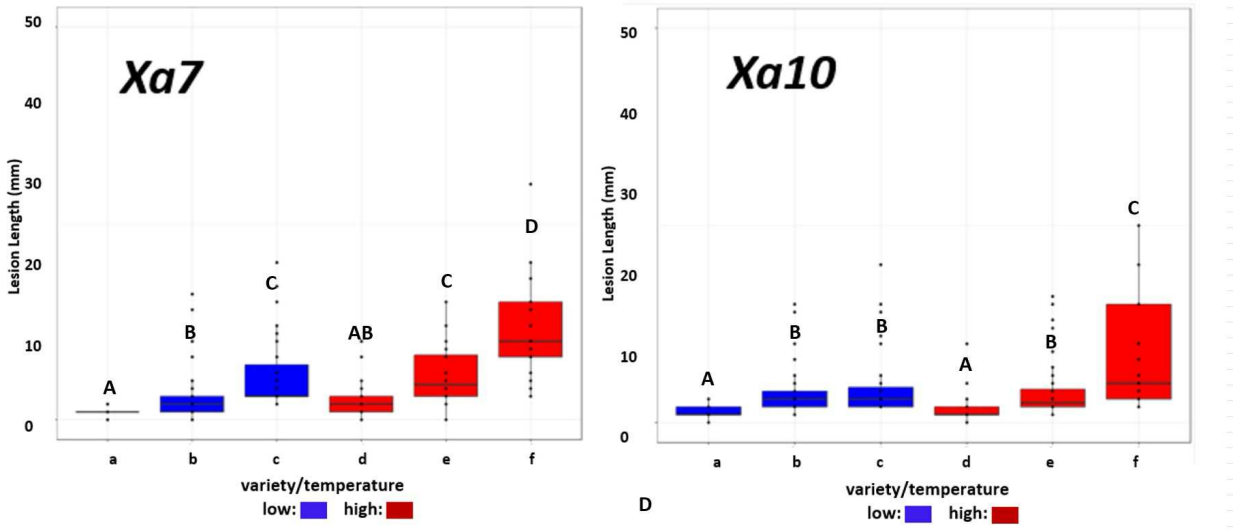

Figure 2.5. Lesion lengths caused by *Xoo* strain PXO86 on rice lines with *Xa7* and *Xa10* at low and high temperature regimes. Lesions were measured at 8, 12, and 16 DPI. Comparisons are ***within* genotype**, with letters indicating differences at *P*-value < 0.05. The experiment was performed five times with ten replications per treatment per timepoint, and results showed similar trends.

Among the six *R* genes tested, lesion lengths were longest in *Xa3* and the susceptible IR24 line at both high and low temperatures (Fig. 2.6). Lesions under the high temperature treatment were longer and developed more rapidly than those at low temperatures in all lines, but to lesser degrees in *Xa7* and *Xa10*. *Xa7* had longer lesions at high temperatures than at low temperatures and lesions on *Xa10* plants remained compact across both treatments. *xa5* and *Xa21* also were restricted in length, but exhibited moderately longer lesions at higher temperatures (Fig. 2.6).

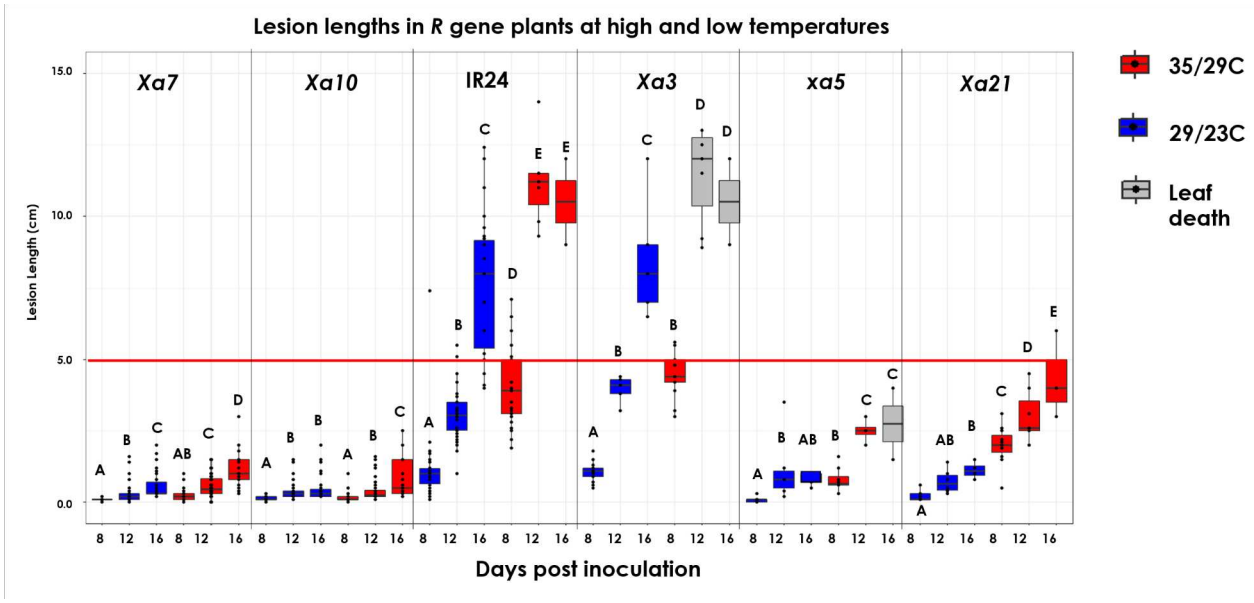

Figure 2.6. Comparison of lesion lengths in all *R* genes tested and the IR24 control. Lesions were measured at 8, 12 and 16 DPI. Comparisons are **across genotypes**, with letters indicating differences at P-value < 0.05. The experiment was performed two times with ten replications per treatment per timepoint, and results showed similar trends.

Trends in Bacterial Numbers and Lesion Lengths

Bacterial numbers and lesion phenotypes varied with rice line and temperature regime. *Xa7* plants showed a slight increase in mean lesion length at high temperature with a reduction in bacterial numbers, while *Xa10* plants showed a decrease in mean lesion length with an increase in bacterial numbers. Death of the leaves in IR24 and *Xa3* plants could have restricted bacterial numbers (Fig. 2.6), and at the 8 DPI timepoint, before death, showed the highest number of bacterial numbers at 10^8 cfu/leaf.

Discussion

High temperatures impact *R* genes in rice, and some *R* genes lose efficacy more quickly and more severely than others. This breakdown is often, but not always, accompanied by more bacteria in the leaf. When systems including bacteria and host plants are stressed, it is important to consider the

ways in which the system will gain new advantages and disadvantages when evaluating the outcomes of applied stress. In this study, we considered the implications of high temperatures on bacterial multiplication and plant health. We show that at elevated temperatures that inhibit bacterial multiplication *in vivo*, rice plants are, in general, more susceptible to BB disease, as reflected by increased lesion lengths and bacterial multiplication. BB resistance genes, such as *Xa3, xa5, Xa7, Xa10* and *Xa21*, confer diverse levels of defense against colonization by the pathogen *Xoo* at current field temperatures (Yang et al., 2000; Xiang et al., 2006a; Lee et al., 2009; Zou et al., 2010; Tian et al., 2014b). Here, we demonstrate that these genes display variation in resistance to *Xoo* at high temperature regimes, with some genes such as *Xa3* and *Xa21* showing reduced efficacy, while others maintain efficacy.

Xoo thrives at 28C (Ke et al., 2017) and is less vigorous at any more than a few degrees above or below its ideal growth conditions (Fig. 2.3). At non-optimal temperatures, i.e., temperatures exceeding 31C or lower than 25C, multiplication of *Xoo* is inhibited or delayed resulting in lower bacterial numbers overall. Thus, one could predict that in rice leaves grown under high temperatures, multiplication of *Xoo* might be inhibited. However, the opposite is true, i.e., at higher temperatures *in vivo*, disease symptoms of *Xoo* are more severe, and higher total bacterial numbers were observed (Fig. 2.4). This phenotype appears in rice plants with no active *R* gene and in several lines with *R* genes, to varying degrees. Studies measuring ambient temperatures, and the temperatures of leaf surfaces, indicate that there is variation between the two as a result of transpiration rates, light intensity, and other physiological inputs.

In our study, which started with 21-day old rice seedlings, *Xoo* multiplies rapidly in the vascular tissue of conducive (compatible) rice leaves, with numbers exceeding 5 X 10^7 and approaching 10^9 CFU/leaf. In the most susceptible interactions, the entire leaf was blighted by as early as 12 DPI, and bacteria did not increase further in this inhospitable tissue. In the presence of *R* genes, the

bacterial numbers did not exceed a threshold of 5 X 10^7 CFU/leaf, especially in strong resistant interactions, and the numbers stabilized or declined as the resistance response ensued. The exception to this is the *R* gene *Xa3*, in which the bacterial numbers and lesion lengths were more comparable to those observed in IR24, the rice without a relevant *R* gene. Our results are consistent with previous observations, although age of the plant and length of the leaf influenced ultimate lesion lengths and sizes of bacterial populations (Barton-Willis et al., 1989; Verdier et al., 2012). Overall, the data suggest that susceptibility increases at high temperatures in all *R* genes to different degrees, and bacterial populations increase more quickly in plants with longer lesions and reduced *R* gene functionality.

Disease resistance provided by *R* genes is quantified in this study by measurements of bacterial numbers in the leaf and lengths of disease lesions after inoculation (Ou, 1985; Mew, 1987). With some *R* genes, like *Xa7* and *Xa10*, short lesions with a distinct dark brown color occur, indicating the activation of an HR in the plant. At high temperatures however, even HR-inducing *R* genes display longer lesion lengths than at low temperature, indicating reduced or delayed activation of defense responses under temperature stress (Webb et al., 2010; Dossa et al., 2015; Dossa et al., 2017). In other cases, such as in the recessive *R* gene *xa5*, the resistant phenotype does not display HR but instead maintains reduced spread of BB symptoms in the leaf in the form of short chlorotic lesions, under 5cm in cases where resistance is intact. This is likely because *xa5* does not depend on the mechanism of HR for effectiveness, but instead relies on reduced transcription of a gene of *SWEET* genes that *Xoo* utilizes for nutrient availability, thereby suppressing virulence of *Xoo*. In fact, (Huang et al., 2016a) observed that induction of additional *SWEET* genes in *xa5* plants allowed *Xoo* to overcome *xa5* resistance, supporting the connection between nutrient availability and pathogen vigor.

Historical reports indicate that bacterial numbers in leaves are correlated with lesion lengths (BartonWillis et al., 1989; Verdier et al., 2012). Interestingly, in this study, we found that bacterial numbers do not always correspond to the expected length of lesions. For example, in *Xa10* plants at high temperature, HR-like lesions are shorter at high relative to low temperatures, but bacterial numbers are greater at the high temperatures. *Xa7* plants show a trend in which lesions at high temperature remain similar in length to the low temperature treatments and bacterial numbers are slightly reduced. Both *Xa10* and *Xa7* restrict bacteria below the threshold of 5 X 10^7 CFU/leaf, indicating that *R* gene efficacy, although reduced somewhat, remains intact at both temperature regimes. *Xa21* also remains effective at both temperature treatments, but to a lesser extent than *Xa10* and *Xa7*, showing increased bacterial numbers and lesion lengths at high temperatures, but notably less than the increases in *Xa3* and the susceptible line IR24. *xa5* shows restricted lesions but relatively high bacterial numbers at both temperatures.

These variations in bacterial numbers as a measure of *R* gene efficacy may reflect the fact that *R* genes employ a variety of strategies in restricting pathogen movement in the leaf. For example: *xa5* relies on a loss of transcription factor efficacy in *S* gene expression (Iyer-Pascuzzi et al., 2008), *Xa7* and *Xa10* use induction of HR (Ji et al., 2022), while *Xa3(Xa26)* and *Xa21* are designed to sense or interrupt the influence of bacterial proteins at the cell membrane. (Song et al., 1995; Sun et al., 2004a; Xiang et al., 2006b; Liu et al., 2020). In this study, both executor *R* genes performed the best in both lesion length and bacterial number comparisons. *xa5* maintained short lesion lengths but had moderately higher bacterial numbers, while *Xa21* had moderately long lesions and bacterial numbers and *Xa3* had very long lesions and high bacterial numbers. *Xa3* has been observed to be less effective in younger plants, like those used in this study which were 21 days old.

If the classification of an *R* gene predicts its stability at high temperature, this would mean that something within the mechanisms of action provides an advantage during heat stress. In the case of

executor *R* genes, which maintained resistance most effectively in this study, further studies are needed to define what physiological differences would provide them with such an advantage. (Alvaro Quintero-Perez, Rene Corral, J.E. Leach, unpublished), see chapter one, and (Chen et al., 2021) observed that under temperatures high enough to limit bacterial multiplication, *Xa7* was expressed at higher levels in rice than it was at low temperature. Considering that the bacterial effector AvrXa7 is needed to induce any *Xa7* expression, and that there are limited bacterial numbers at high temperature, we know that the increased expression of *Xa7* is not due to the presence of more *Xoo*. However, if *Xoo* effectors are a means to increase host susceptibility in favor of bacterial colonization, increased effector expression by heat-stressed bacteria could be a means of reversing the muted colonization effects of their dwindling numbers. Further studies are needed to determine the relationship of bacterial numbers, effector production, *R* gene expression, and disease resistance and susceptibility at high vs low temperature regimes.

It could also be possible that heat-sensitive mechanisms in the plant could be responsible for facilitating greater *Xa7* expression when AvrXa7 is present. (Chen et al., 2021) describes increasing rates of expression of *Xa7* when studying two strains of *Xoo* infected rice at 23C, 29C, and 35C. The study found the greatest increases of *Xa7* expression were observed at 6 and 12 hours after inoculation in the 29C and 25C treatments, which is not concurrent with the temperature-based bacterial growth curves conducted in this study (Fig.2.3) which peak at approximately 24 hours at only 27C and 29C. In addition, the Chen study found that at cooler temps (23C) expression of *Xa7* was delayed, and that increases in expression paralleled increases in temperature, suggesting that temperature-induced activation in the presence of the cognate TAL effector may be a plausible mechanism of induction, at least in this executor-type gene. (Cohen et al., 2019) described reduced expression of genes related to plant stress hormone biosynthesis in *Xa7* plants at high temperature. Specifically, he found that abscisic acid (ABA) related genes were downregulated in heat stressed

Xa7 plants challenged to respond with resistance relative to susceptibility. Expression of ABA responsive genes is normally induced in response to environmental stresses like heat. (Mang et al., 2012) credits a deficiency of ABA with improved disease resistance at high temperature in Arabidopsis. The study also observes that reduced ABA promotes accumulation of SNC1 and other *R* proteins in the nucleus, which play a key role in maintaining disease resistance phenotypes in Arabidopsis.

Interestingly, *Xa7* and our other *R* gene, *Xa10* both localize to the endoplasmic reticulum (ER). (Wang et al., 2021) visualized Xa7 localization in the ER, which plays a crucial role in the activation of the HR phenotype (Zuppini et al., 2004; Chisholm et al., 2006; Jones and Dangl, 2006). The ER turns on HR when ROS are produced in response to stress. Receptors on the ER perceive ROS at varying strengths, but when those receptors are blocked, HR is more robust. Could the protein products of executor *R* genes block these receptors and increase the HR phenotype? The relative amounts of Xa7 localization during high and low temperature treatments have not been studied at the time of writing but given the role of the ER in initiating programmed cell death and its role in ROS signaling pathways, it is possible that Xa7 localization in the ER, in conjunction with available ROS from heat and pathogen stress, results in greater induction of the *Xa7* phenotype. Plant heat sensing pathways have not been fully characterized, but literature tells us that several kinds of signaling molecules, including reactive oxygen species (ROS), fluctuate when plants experience environmental stresses including but not limited to heat and pathogen stress (Huang et al., 2019). ROS are involved in many signaling pathways in plants, and can accumulate in the ER, where programmed cell death, or PTI is initiated as a result. We can extrapolate that the protein product of *Xa7* is doing something to improve the HR specifically when heat is present. But how might it change physiology in the cell to make this possible?

(Xu et al., 2012) found that silencing of ER protein receptors in *Nicotiana ERD2* led to increased stress, resulting in exaggerated programmed cell death after bacterial infection, suggesting that the increase of stress signaling is an important component of strong HR phenotypes. The other executor gene in this study, *Xa10* also localizes to the ER, disturbing the balance of Ca+ ions to trigger cell death. Perhaps here too, the additional presence of abiotic stress signaling in the ER influences the outcomes of resistance phenotypes. We need more information to determine the degree to which changes in protein accumulation would need to occur to influence disease resistance in plants containing *Xa3, xa5,* and *Xa21*to effectively characterize this hypothesis.

In addition, (Chen et al., 2019) observes that in young rice plants, lower temperatures can activate *Xa21* and that warm temperatures will suppress it, much like we see in our *R* gene temperature study. They also note that protein accumulation of XA21 is higher in young resistant plants at approximately 14 days after germination, while the plants in this study were 21 days old, but that it was sensitive to protease degradation over time. The timing of plant age with seasonal environmental cues, may be a result of lower temperatures in the early growing season, which would allow for protection of younger plants. Overexpression of *Xa21* partially restores resistance in young plants at warm temperatures which could be similar to higher *Xa7* expression levels in the presence of *Xoo* at high temperatures resulting in resistant phenotypes.

In conclusion, resistance genes in rice generally do not perform as well when deployed under high temperature treatments as compared to low temperatures, but executor-type *R* genes maintain a degree of efficacy. More studies are needed to elucidate the mechanism that prevents breakdown in *Xa7* and *Xa10* and to determine if this mechanism is unique to executor *R* genes or if it can be found in wider categories. As average temperatures continue to increase in rice growing regions, it will become important to consider the impact of temperature stress on *R* genes employed in field

strategies to combat BB. As *R* genes remain the primary effective strategy against *Xoo*, breakdown of disease resistance will place greater yield risks on rice growers and their local communities. *Xa7* and *Xa10* maintain the most resistance at high temperatures with the shortest lesions, *xa5* maintains moderate resistance, *Xa21* maintains slightly less resistance and *Xa3* performs similarly to plants with no active *R* gene present, both with significantly longer lesions than in other *R* genes tested. In addition, the number of bacteria in the plant as measured by CFU/leaf are not always predicted by lesion length. Trends in bacterial numbers in *Xa7*, *Xa10*, and *xa5* do not increase in tandem with lesion measurements.

Future directions for this study could include the interruption of ROS signaling inputs with an abiotic stress hormone inhibitor in plants experiencing combined heat and disease stress to determine the contribution of ROS in maintaining disease resistance. Further observations of protein localization to the ER at temperatures similar to those used in this project would help determine if the hypothesis regarding the role of signaling from this organelle is true. In addition, observation of bacterial movement within the leaf, especially in relation to HR would help provide insight on the discrepancies between trends of bacterial numbers and HR phenotypes. Considering the role of humidity in maintaining disease resistance would also be useful, given the similar appearance of dry leaves with those overcome by a population of bacteria. Studying the phenotypes of *Xoo* resistance genes in older plants would allow for greater distinguishing of each phenotype along a larger leaf surface and provide more translatable evidence to the field. And perhaps most importantly, studying the combined stress phenotypes in the *R* genes not tested here, specifically additional executor genes, would shed light on whether the connection between the type of resistance gene and efficacy at high temperature is a trend beyond those used in this work.

Methods:

Bacterial strains and plant varieties

Seeds of rice near isogenic lines (NIL) IRBB61 containing the *R* gene *Xa7* (Vera Cruz CM et al. unpublished), IRBB10 containing *Xa10*, IRBB3 containing *Xa3*, IRBB5 containing *xa5*, IRBB21 containing *Xa21*, and the recurrent susceptible parent IR24 (Ogawa et al., 1991)(Khush et al 1990) were first germinated on filter paper with 5mL Maxim fungicide in the dark for 5 days at room temperature (Cohen et al., 2019). Germlings were planted in 200-unit seed trays containing a mixed medium (ProMix and Greens Grade medium, 2:1), alternating one empty pot with each germinating seed to allow access to plants. Plants were grown in Conviron growth chambers at 29 C day/23 C night, with 12,000 lumens and 80% humidity for 17 days. Humidity, temperature, and light were tracked with HoboLog dataloggers throughout the experiment. Plants were chelated at 10 days and fertilized twice a week. Water levels were designed to mimic rice paddy conditions, with some standing water always present over root systems.

Xoo strain PXO86, which induces resistance in rice containing *Xa3, xa5, Xa7, Xa10*, and *Xa21,* was grown on peptone sucrose agar (PSA, Karganilla et al., 1973) plates for 48 h at 28C and then transferred to nutrient agar (NA) plates for 48h at 28C. Bacteria were stored long term in 30% glycerol at -80C.

Plant assays

At 18 days after sowing, half of the plants were moved from 29 C/23 C to a higher temperature growth chamber (35 C day/29 C night) three days before inoculations and to allow plants to acclimate. Growth chambers were maintained at 35C/29C for high temperature treatments and 29 C/23 C for low temperature treatments, both with 12 h day/night light regimes at 12,000 lumens and 50% to 80% RH (Cohen et al., 2019).

Clip Inoculations for Lesion Lengths and Bacterial Numbers

Scissor clip inoculations for lesion length measurements and bacterial colony counts were performed on the youngest fully emerged leaf across all six rice lines as described in (Kauffman et al., 1973). Inoculum was prepared with cells of *Xoo* PXO86 suspended in sterile water at 0.2 OD_{600} (10^8 CFU/ml). Inoculum was streaked for isolated colonies on peptone sucrose agar (PSA) to confirm purity of cultures. Sterile water inoculations were used as a negative control.

Bacterial-induced lesion lengths were measured at 8, 12 and 16 DPI. Bacterial numbers were assessed in entire inoculated leaves at 8, 12 and 16 DPI. Sampled leaves were placed in a 2mL tube with 1mL sterile water. Leaves were ground with sterile metal beads in a Qiagen TissueLyser II at 30/s for 3 min. The ground tissue emulsion was then diluted with sterile water into seven total serial 10-fold dilutions (20 ul into 180 ul sterile dH_20), and 10uL of each dilution was plated on NA media, with three technical replicates for each series of four leaves. Colonies were counted after 48 h of incubation at 28 C. Data were analyzed by fitting a one way ANOVA model using the base R (version 4.3.0) function lm(). Post-hoc pairwise comparisons were then conducted using the emmeans() function from the R package emmeans (version 1.8.8).

Bacterial growth curves

Xoo PXO86 was cultured on PSA for 48 h at 28C and then transferred to PS broth for 24 h at 28C with shaking. Liquid media cultures were washed with sterile dH_20 and diluted with additional PS broth to 0.0002 OD_{600} (approximately 10^5 CFU/ml). The bacteria (200uL) were added to wells of a 96 well plate with uninoculated PS broth as a negative control. Edge rows were not used in these assays. After sealing with a breathable membrane, the plates were placed in a BioTek PowerWave HT plate reader for up to 72 h at 35C, 33C, 29C, 27C, 25C, and 23C with shaking at 225 RPM, with hourly optical readings. Data was extracted from Gen5 software to Microsoft Excel and analyzed in R Studio.

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