LEVERAGING QUANTITATIVE GENETICS TO BREED FOR DURABLE FUNGAL DISEASE RESISTANCE IN DRY BEAN (*PHASEOLUS VULGARIS L.*)

By

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ABSTRACT

Dry beans (*Phaseolus vulgaris* L.) are an important legume for human consumption worldwide, providing key dietary nutrients such as carbohydrates, protein, and fiber. Michigan is the second largest dry bean producer in the United States, producing over 400 million pounds of dry edible beans per year and contributing to a farm gate value of over $139 million. However, biotic stress caused by fungal disease infection is among the main constraints that limit yield and increase management cost of dry bean production. Two of the most devastating fungal diseases in Michigan are white mold and root rot, which can cause up to 80-100% yield and seed quality decline in susceptible cultivars under heavy disease pressure. Resistant breeding lines are the ideal solution to managing these diseases as chemical control is expensive, harmful to the environment, and doesn't ensure complete protection against infection. Unfortunately, resistance to both diseases is controlled by complex quantitative inheritance methods, with a lack of large effect resistance genes, laborious screening protocols, and historic difficulty pyramiding genes into one durably resistant phenotype. Therefore this research aimed to assist dry bean breeders in developing resistant lines by i) evaluating genomic prediction and GP + de novo GWAS as a tool for white mold (*Sclerotinia sclerotiorum*) resistance screening, ii) evaluating a diverse set of lines for resistance to root rots conferred by *Rhizoctonia solani* and *Fusarium oxysporum,* iii) investigating correlations between *Fusarium oxysporum* inoculated field and greenhouse screens and natural infection conditions and, iiii) investigating correlations between *Fusarium oxysporum* root rot resistance and major agronomic/ aboveground traits as an alternative non-destructive, high-throughput phenotyping method. The findings from these studies will ultimately assist dry bean breeders in the development of resistant lines for these important diseases.

I would like to dedicate this work to my family: My fiancé Mark, for his constant support and humor during the challenges of graduate school and life; My parents, sister, and brother for their love and encouragement in pursuing my passion; and my grandparents for always believing in me.

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GENERAL INTRODUCTION

Common bean (*Phaseolus vulgaris* L.) is one of the most important legumes for human consumption worldwide, providing an important source of key dietary nutrients such as carbohydrates, protein, fiber, iron, and zinc (Uebersax et al., 2023). There are two common bean markets, dry shelled seed (dry bean) and green pods (snap bean), often split into individual breeding programs (Singh & Schwartz, 2010). Dry beans are a dietary staple in Latin America and sub-Saharan Africa (Leterme & Carmenza Muũoz, 2002; Paparu et al., 2018) and an important agricultural commodity in the US (primarily North Dakota, Minnesota, and Michigan), Latin America, and other regions. Most dry bean breeding programs aim at improving yield, biotic resistance, nutritional quality, and abiotic stresses (Kelly and Cichy, 2012). However, there are many diseases that greatly affect dry bean production including anthracnose, common bacterial blight, viral mosaics, root rots, and white mold (Singh & Schwartz, 2010).

There are two major gene pools within *Phaseolus vulgaris* resulting from two centers of domestication in the Andes and Central America (Gepts, P., and Debouck D., 1991; Mensack et al., 2010). Within these gene pools there are over 10 major market classes of dry bean with unique seed color and morphology, growth habit, disease reaction, and adaptation. Common dry bean market classes include black, navy, red, pink, yellow, pinto, great northern, kidney, and cranberry beans. The wide genetic diversity of dry bean across market classes offers an opportunity to incorporate many novel traits within a variety development program. However, the stringent requirements for seed traits within market classes along with other traits of economic importance have made genetic progress more challenging when compared to other crops.

Michigan is the second largest dry bean producer in the United States, producing over 400 million pounds of dry edible beans per year and contributing to a farm gate value of over $139

million (USDA Crop Production Summary 2022). It also leads the nation in the black, small red, and navy bean market classes in terms of acreage. Michigan's temperate climate, with warm humid summers, is highly conducive to fungal diseases. Among the fungal diseases present in Michigan, white mold and root rots are ranked by growers as the 1st and 2nd most important diseases limiting production respectively (MBC, 2022).

Agronomic management is often inefficient in controlling fungal diseases, primarily due to the durability of overwintering structures that persist in the soil and crop residue (Bolton et al., 2006; Katan, 2017). Breeding for resistance is often the most sustainable, durable, and cost-effective method to protect against infection. However, many fungal diseases (including white mold and root rots) have complex genetic inheritance methods and cumbersome phenotypic screening that limit breeding progress (Fuller et al., 1984; Hagerty et al., 2015; Miklas et al., 2004; Nakedde et al., 2016; Park et al., 2001; Schwartz & Singh, 2013; Singh et al., 2014; Wang et al., 2018). Given the complexity of breeding for resistance, more research is needed to improve screening methods, identify, and pyramid resistance genes, and produce robustly resistant varieties.

WHITE MOLD

White mold, conferred by the fungal pathogen *Sclerotinia sclerotiorum* (Lib.) de Bary is one of the most destructive fungal diseases of dry bean, resulting in poor seed quality and reduced yield (del Río et al., 2004; Vasconcellos et al., 2017). In temperate regions it is considered the most yield limiting disease of dry bean production, causing up to 100% yield loss and may also result in significant seed quality decline in susceptible cultivars (Schwartz, 2011; Singh & Schwartz, 2010). Consequently, economic losses from white mold in the United States have exceeded 200 million dollars yearly due to yield reduction and fungicide costs (Bolton et al., 2006).

Sclerotinia sclerotiorum, a necrotrophic fungal pathogen from the phylum *Ascomycota,* has a broad host range of over 400 species of plants, primarily dicotyledonous species (Boland & Hall, 1994). Sclerotinia sclerotiorum can be seed transmitted. However, the bulk of inoculum comes from melanized sclerotia that can overwinter in soil for five or more years (Schwartz & Singh, 2013). Around dry bean flowering time, *S. sclerotiorum* sclerotia germinates and forms a fruiting structure, a small cup shaped mushroom called an apothecia (Miklas et al., 2013). Once the apothecia has developed, asci can forcefully release ascospores into the plant canopy. These ascospores can infect the plant through wounds on the stem left by the senescent flowers and on other foliar parts, by penetrating the host cell walls (Bolton et al., 2006). Once colonized, infected plants often exhibit wilted leaves due to reduced vascular function. As disease development progresses, stems become brittle, and have a bleached appearance due to oxalic acid and other enzymes produced by the fungi, often leading to severe plant lodging (Schwartz & Singh, 2013). Yield and seed quality decline occurs indirectly due to plant stress that reduces seed development or lost pods that abort entirely and directly due to colonization of bean pods that results in moldy, discolored, or shriveled seed. In severe infections, white mycelium can be observed on affected plant parts and whole plant death can occur (Schwartz & Singh, 2013). Sclerotia form inside infected tissue in the stem pith or pods and provide inoculum for subsequent infection (Bolton et al., 2006).

CONTROL

Managing white mold can be challenging due to the durability and extended viability of sclerotia in the soil and broad host range (Bolton et al., 2006). White mold can be spread from field to field by infected seed, contaminated soil on farm equipment, and wind-blown ascospores (Steadman and Boland, 2005). Control of white mold using fungicides, biopesticides, and other

disease management strategies has been difficult. Common strategies for controlling white mold include crop rotation, fungicide sprays, limiting nitrogen fertilizer and irrigation to reduce vegetative growth, wide row spacing, low density planting, and the development of low biomass, upright, and open canopy cultivars (Ender & Kelly, 2005; Miorini et al., 2017; Schwartz & Singh, 2013). Biomass reduction and upright architecture in particular focuses on reducing sclerotial germination and ascospore development by altering the microclimate to reduce canopy humidity and temperature and therefore colonization ability (Schwartz & Singh, 2013). However, these strategies are often not sustainable or economically feasible and reduce productivity of dry bean fields. Control of white mold through biomass management, wide row spacing, and reduced fertilizer and irrigation compromises yield per acre. Additionally, fungicide sprays are expensive and have adverse effects on the environment. Furthermore, these practices do not fully eliminate white mold infection due to its virulence and ability to overwinter in the soil.

HOST RESISTANCE AND SCREENING

Host resistance to white mold in dry bean can be obtained through two means: physiological resistance and avoidance mechanisms, although low resistance has been found to date. Physiological resistance (Miklas et al., 2013; Schwartz & Singh, 2013) is associated with pathogen recognition, including reactive oxygen species (oxidative burst) as an initial defense mechanism and synthesis of pathogenesis-related protein (Mamidi et al., 2016). Conversely, disease avoidance mechanisms are related to plant architecture traits that confer a tall, upright growth habit, reduced lodging, and a porous plant canopy less ideal to the pathogen. Due to the complex infection method of the pathogen, a combination of both traits is considered ideal to ensure robust white mold resistant lines.

Multiple screening methods have been proposed, including the greenhouse straw test, cut stem oxalate test (Kolkman & Kelly, 2000), and field rating systems such as the 1-9 plot wise visual scale (Miklas et al., 2001). Previous studies have failed to show a strong correlation between field and greenhouse screening methods (Terán et al., 2009). The greenhouse trial is preferred by many researchers due to higher heritability, ease of screening, and less environmental dependance but this test does not screen for resistance conferred by avoidance mechanisms in field conditions (Chung et al., 2008; Mkwaila et al., 2011; Soule et al., 2011). Many highly resistant lines in the greenhouse exhibit low expression in field conditions. Field testing is necessary to screen for plant avoidance traits that reduce infection, but physiological resistance and architectural avoidance is confounded in field conditions (Miklas et al., 2013) False positives for physiological resistance may result due to reduced disease severity in open plant canopies (Kolkman & Kelly, 2000; Miklas et al., 2013). No completely resistant lines to white mold in dry bean have been identified to date. A multi-faceted screening and selection method that incorporates both aspects of resistance would greatly improve breeding progress for this complex trait.

BREEDING FOR WHITE MOLD RESISTANCE

Developing white mold resistant cultivars in dry bean has proven difficult primarily due to the complex inheritance method of resistance, low levels of innate resistance in breeding populations (Kolkman & Kelly, 2000; Terán & Singh, 2009; Vuong et al., 2004), low heritability (Fuller et al., 1984; Park et al., 2001), and variable screening methods (Ender & Kelly, 2005). Previous studies have identified that both physiological resistance and architectural avoidance to white mold is quantitatively inherited (Fuller et al., 1984; Miklas et al., 2004; Park et al., 2001). Hundreds of germplasm accessions, cultivars, and breeding lines of common bean and related

Phaseolus species have been screened for their reaction to white mold. Unfortunately, completely resistant varieties are unavailable and most known sources of genetic resistance to white mold are of Andean origin, usually from unadapted landraces and wild relatives, and from secondary gene pools (Terán and Singh, 2009, Singh et al., 2014; Vasconcellos et al., 2017). While low levels of resistance have been identified in small Middle-American germplasm (Ender and Kelly, 2005; Mkwaila et al., 2011; Hoyos-Villegas et al., 2015), progress to develop white mold resistant cultivars has been hindered by difficulty in pyramiding resistance genes given the quantitative inheritance of disease avoidance mechanisms and physiological resistance (Singh et al., 2014).

Over 35 quantitative trait loci (QTL) with minor effects have been identified conferring resistance to white mold in dry bean by either physiological resistance or architectural avoidance which highlights the complexity of this trait (Singh et al., 2014; Vasconcellos et al., 2017). QTL conferring physiological avoidance and architectural traits have been mapped to most dry bean linkage groups with common traits being oxalate resistance, determinate growth habit, increased internode length, plant height, days to flowering, branching pattern, lodging, seed size, and yield (Ender & Kelly, 2005; Miklas, 2007; Miklas et al., 2001, 2003, 2013; Mkwaila et al., 2011; Soule et al., 2011). Miklas et al. (2007) identified two QTL located on PV2 and PV3 conditioning physiological resistance associated with the stay green stem characteristic and disease avoidance traits including late maturity and the stay-green stem characteristic, which are undesirable agronomic traits for dry bean production (Miklas et al., 2006). These traits are important to account for when screening for resistance to white mold because while they confirm a resistant genotype and can be used as a parent for breeding, they are deleterious traits for dry bean production. Recently, Vasconcellos et al. 2017 compiled 37 individual QTL across 14 recombinant inbred bi-parental populations developed previously and condensed these QTL into 17 meta-QTL found on

chromosomes 1,2,3,5,6,7, and 8. These meta QTL in particular are potential targets for marker assisted selection (MAS) of partial resistance to white mold.

Unlike breeding for qualitative resistance, breeding for quantitative resistance is more challenging because it requires multiple cycles of breeding and screening, leading to a gradual improvement of resistance in a breeding population over time. With the sheer number of QTL identified previously conferring many environmentally dependent resistance traits, it is no surprise that there has been difficulty pyramiding genes into one robustly resistant genotype with the desired agronomic traits. An integrated screening and recurrent selection method that considers all aspects of resistance and agronomic performance would greatly assist future cultivar development. Multiple breeding tools such as recurrent selection and the use of alternative populations such as multiparent intercrosses are one way to assist the identification and pyramiding of resistance genes (Escobar et al., 2022; Osorno et al., 2018). Recently, Escobar et al. explored integrated multi-parent crosses and gamete selection using a Multiparent Advanced Generation Inter-Cross (MAGIC) population, to facilitate mapping and breeding efforts, resulting in multiple partially resistant lines. This method crosses together multiple founder lines and cycles through several additional generations of crossing, resulting in offspring with multiple recombination events, often leading to improved results that maximize diversity. This and other emerging breeding tools will assist future breeding for complex traits.

GENOMIC PREDICTION

Genomic prediction creates a unique opportunity to select and pyramid major and minor alleles conferring resistance to complex diseases (Merrick et al., 2021; Poland & Rutkoski, 2016; Tiede & Smith, 2018). This established tool in Marker Assisted Selection (MAS) utilizes genome wide markers and phenotypic data to train a linear statistical model to predict and make selections

based on genomic estimated breeding values (GEBVs) for a trait. In contrast to MAS, genomic prediction does not identify significant markers and estimate individual marker effects. Instead, all marker effects are estimated simultaneously and used to accurately predict overall breeding values for a trait (Jannink et al., 2010). When properly implemented, genomic prediction has the potential to improve breeding program efficiency and reduce phenotyping costs when screening for complex traits. Genomic prediction has assisted breeding efforts for quantitative disease and pest resistance traits in many crop species including wheat: (Arruda et al., 2016; Juliana et al., 2017, 2022; Larkin et al., 2021; Merrick et al., 2021; Odilbekov et al., 2019; Rutkoski et al., 2012; Sarinelli et al., 2019) , dry bean (Diaz et al., 2021; Shi et al., 2021a), soybean (Bao et al., 2015; de Azevedo Peixoto et al., 2017; Đorđević et al., 2019; Duhnen et al., 2017; Hemingway et al., 2021; Shi et al., 2021b; Wen et al., 2018), and other crop species. Specifically, when compared to MAS or phenomic selection alone, genomic prediction has shown to be more effective for pyramiding quantitative traits controlled by many small effect QTL (Heffner et al., 2010, 2011; Massman et al., 2013; Merrick et al., 2021; Zhang et al., 2016).

Many previous studies of the genetic control of white mold in dry bean fail to account for the many small effect QTL unable to be detected by genome wide association studies (GWAS) that are likely controlling resistance to this trait. To date, there have been no major resistance genes identified for white mold resistance, unlike some disease traits such as *Fusarium* head blight in wheat (Larkin et al., 2021). Since genomic prediction utilizes all markers spread across the entire genome to make selections, it has the power to detect small effect QTL conferring resistance that would otherwise be overlooked by GWAS (Jannink et al. 2010; Meuwissen et al. 2013). Genomic selection for white mold resistance has been studied in soybean diversity panels (de Azevedo Peixoto et al., 2017; Wen et al., 2018) with moderate to high prediction accuracies (0.4-0.7)

suggesting that this warrants further research into the validity to assist breeding for resistance in dry bean.

Another benefit of genomic prediction in breeding for complex traits controlled by many major and minor QTL is that major QTL controlling a trait of interest can be identified using a GWAS and implemented in genomic prediction as fixed effects, allowing for further accuracy in selection (Merrick et al., 2021). Fixed QTL can be selected based on previous research or identified through *de novo* GWAS of the training population. GS + *de novo* GWAS involves two main stages where in the first stage GWAS is conducted on individuals in the training set to identify fixed QTL to be implemented in genomic prediction of the testing set (Bian & Holland, 2017; Haile et al., 2021; Sarinelli et al., 2019; Spindel et al., 2016). For example, Bian and Holland 2017 and Spindel 2016 both found that GS + *de novo* GWAS outperformed all other models tested. A simulation study by (Rice & Lipka, 2019) evaluated the effect of the number of QTL implemented across multiple genetic architectures and trait heritability and observed increased, decreased, and mixed (increase then decrease or decrease then increase) effect on prediction accuracy depending on the trait and number of fixed QTL. This study emphasizes that a varying number of fixed effect QTL should be tested for each trait before implementation.

ROOT ROTS

Root rot is conferred by a soilborne disease complex of multiple fungal and oomycete pathogens including members of *Fusarium sp.*, *Rhizoctonia sp.*, *Pythium sp.*, *Macrophomina sp.*, and *Alternaria sp*. (Bilgi et al., 2011; Harveson et al., 2005; Schwartz, 2011; Sendi et al., 2020; Singh & Schwartz, 2010). Pathogen composition is variable by region and usually involves multiple species. In Michigan, the most common root rot pathogens belong to the *Fusarium* and *Rhizoctonia* species as well as multiple oomycetes (Jacobs et al., 2019). Root rots conferred by

these pathogens cause up to 84% yield loss in susceptible dry bean cultivars through damage of root biomass, reducing vigor, and sometimes whole plant death (Jacobs et al., 2019). Root rots greatly affect overall plant health because a damaged root system limits nutrient and water intake, greatly affecting yield potential.

The life cycle of root rot complex pathogens are similar. First, primary inoculum (resting spores, sclerotia, or mycelium) in the soil or crop residue germinates and infects seedling roots. The pathogen spreads from plant to plant using secondary spores (conidia or sporangia) or mycelium. Finally, it produces new primarily inoculum at the end of the season that overwinters until the next growing season (Gossen et al., 2016). Root rot disease symptoms vary depending on the pathogen(s) involved, but are primarily characterized by root lesions, root and foliar biomass loss, and reduced stand. *Fusarium* and *Rhizoctonia* root rots of dry bean are both primarily characterized by water-soaked, dark brown to rust colored lesions on the root and death of lateral roots (Hall et al. 1991, Hagedorn et al. 1994). *Fusarium* root rot develops slowly in some genotypes and tends to lead to an overall root biomass loss, vigor loss, and yield decline. Entire plant death is rarer and usually occurs later in the season in only severe infections. Aboveground symptoms are difficult to see, but plants may be stunted and yellowed, exhibit premature leaf drop and have poor pod fill (Schwartz, 2011). Conversely, *Rhizoctonia* root rot tends to affect plants early in the season primarily through reduced stand resulting from seed rot, seedling blight, and pre/post emergence damping off (Gossen et al., 2016). Rhizoctonia root rot also tends to result in the formation of deeper sunken lesions at the soil level (Conner et al., 2014). Root rot disease severity is highly variable with environmental conditions. In particular, cool wet weather, soil compaction, or flooding events can increase infection severity in both infection scenarios due to additional plant stress and increased virulence of soilborne pathogens (Kumar and Kudada, 2018, Cichy, 2007).

CONTROL

Current agronomic management practices assist with lowering root rot disease severity but are not sufficient for the complete control of root rot. Fungicidal seed and soil treatments, reduced irrigation, crop rotation, cover crops, seedbed preparation, and other agronomic practices are currently used to combat yield loss from root rots (Abawi and Pastor Corrales, 1990; Gossen et al., 2016; Harveson et al., 2005; Rubiales et al., 2015). A 3-5 year crop rotation with a non-host crop (alfalfa, barley, wheat, oats, and corn) is recommended for fields with root rot disease pressure to reduce inoculum load (Schwartz, 2011). If possible, planting in warm moist soil is ideal for quick germination and emergence. High-quality treated, certified seed is also recommended to maximize plant vigor and stand.

One major limitation of many root rot control methods is that they are often not sustainable or economically feasible and reduce productivity of dry bean fields. For example, control of root rots through reduced irrigation and fertilizer limits yield per acre. Fungicidal seed and soil treatments are expensive and have adverse effects on the environment. Furthermore, these practices are not complete guarantees against root rot infection due to its virulence and ability for thick-walled spores, hyphae, or sclerotia to overwinter in the soil for multiple years (Schwartz, 2011). The development of resistant dry bean cultivars would ensure high levels of protection against this disease. Therefore, the most sustainable, durable, and cost-effective method to ensure protection against infection would be to develop resistant dry bean cultivars.

HOST RESISTANCE AND SCREENING

Resistance to root rot in dry bean appears to be a combination of physiological mechanisms and root system avoidance due to architecture traits such as root dry weight, root length, and root mass (Snapp et al., 2003; Kamfwa et al., 2013; Wang et al., 2018; Haus et al., 2020). These root

traits in particular are identified as targets for the screening and development of root rot resistant varieties. Previous research has established susceptibility to root rot in both dry bean gene pools, but susceptibility to individual fungal and oomycete pathogens differ. Large-seeded dry bean cultivars from the Andean gene pool are generally more susceptible to *Fusarium* species and small seeded cultivars from the Middle-American gene pool are generally more susceptible to *Rhizoctonia* species, with Andean types being more susceptible to root rots overall (Conner et al., 2014; Schneider et al., 2001). Previous research has also established the importance of high root biomass cultivars with high density of lateral roots, high basal root number, and many adventitious roots (Haus et al., 2020; Román-Avilés et al., 2004; Snapp et al., 2003). Andean genotypes in particular, tend to have less robust root systems which is thought to be why they have lower levels of resistance to root rots (Cichy et al., 2007; Román-Avilés & Kelly, 2005; Schneider et al., 2001). There have been multiple phenotyping methods proposed for the greenhouse and field evaluation of *Fusarium* root rots including the liquid inoculum method (Schneider & Kelly, 2000), inoculum layer method (Chaudhary et al., 2006), and nutrient culture (Boomstra et al., 1977). Inoculated grain planted alongside the crop seed is a common method for field evaluation (Haus et al., 2020; Pandey et al., 2020; Wang et al., 2018). The presence of multiple resistance mechanisms through both physiological resistance and architectural avoidance complicates screening. Greenhouse trials are preferred by researchers because they provide a controlled environment free of natural root rot pathogens, have higher heritability, and ease of screening, but they do not screen for some avoidance mechanisms that can only be observed in the field. Conversely, field screens allow for measurement of both architectural traits and physiological resistance, but are often hampered by low heritability, high coefficient of variation (CV), high error variances, and natural pathogen presence (Guzman, 2016.; Hagerty et al., 2015; Nakedde et al., 2016; Román-Avilés et al., 2004;

Román-Avilés & Kelly, 2005). Furthermore, these destructive phenotyping methods require the whole plant to be dug up with a shovel in the field or removed from the pot in the greenhouse to be evaluated for root rot symptoms, which is laborious and prevents additional measurements of yield and other traits throughout the season. Establishment of aboveground traits related to root rot disease severity would enable high-throughput methods of phenotyping such as UAS imaging to assist breeders in screening for root rot resistant varieties (Guo et al., 2021; Lu et al., 2019; Manganiello et al., 2021; Marzougui et al., 2019).

BREEDING FOR ROOT ROT RESISTANCE

Genetic resistance to root rots in dry bean is quantitatively inherited, primarily through root architecture traits such as root weight, root length and root mass (Haus et al., 2020; Kamfwa et al., 2013; Snapp et al., 2003; Wang et al., 2018) and physiological avoidance (Kamfwa et al., 2013; Mukankusi et al., 2011; Román-Avilés et al., 2004; Snapp et al., 2003). The quantitative inheritance of these traits has been demonstrated by the low narrow-sense heritability estimates (26%-44%) reported and many small effect quantitative trait loci (QTL) found for root rot (Román-Avilés et al., 2011; Nakedde et al., 2016; Kamfwa et al., 2018; Wang et al., 2018; Zitnick-Anderson et al., 2020). Previous research has established the importance of high root biomass cultivars with high density of lateral roots, high basal root number, and many adventitious roots for root rot avoidance (Haus et al., 2020; Román-Avilés et al., 2004; Snapp et al., 2003). These traits have been identified as significant targets for improving resistance. Resistance to root rot is primarily found in the small-seeded Middle-American bean germplasm compared to the highly susceptible large-seeded beans of Andean origin and has served as the only source of resistance (Cichy et al., 2007; Mukankusi et al., 2011; Román-Avilés & Kelly, 2005; Schneider et al., 2001)

Although few studies to date have evaluated genetic resistance to *Fusarium oxysporum* or *Rhizoctonia solani*, multiple studies have established genomic regions associated with resistance to root rots conferred by other *Fusarium sp.* (Hagerty et al., 2015; Kamfwa et al., 2013; Nakedde et al., 2016; Román-Avilés & Kelly, 2005; Schneider et al., 2001; Wang et al., 2018; Zitnick-Anderson et al., 2020). These studies often identify many different QTL which highlights the highly quantitative inheritance method of resistance. QTL studies of resistance to *Fusarium sp.* in dry bean have identified genomic regions associated with root traits, plant immune/defense mechanisms, and other disease resistance genes (Hagerty et al., 2015; Nakedde et al., 2016; Wang et al., 2018; Zitnick-Anderson et al., 2020). One study to date has evaluated genetic resistance to *Rhizoctonia solani*- Oladzad et al. 2019. In this study, researchers evaluated the Andean and Middle American diversity panels and observed multiple QTL associated with gene models encoding proteins like known disease resistance genes. Other *Rhizoctonia solani* resistance studies have focused on screening and ranking breeding lines and establishing trait correlations (Adesemoye et al., 2018; Conner et al., 2014; Peña et al., 2013).

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CHAPTER 1: POTENTIAL OF GENOMIC PREDICTION TO SCREEN FOR WHITE MOLD RESISTANCE IN DRY BEAN

ABSTRACT

Dry bean (*Phaseolus vulgaris L.*) production in the U.S. suffers severely from white mold (*Sclerotinia sclerotiorum* (Lib.) de Bary) infection. Dry bean cultivars lack high levels of resistance, and progress to breed new cultivars with durable levels of resistance has been slow due to the quantitative inheritance of this trait, difficulty pyramiding resistance, and screening dependence on the presence of the pathogen under suitable environmental conditions. Genomic prediction provides an alternative method to pyramid resistance genes by utilizing genome-wide marker coverage to predict genotypic values for quantitative traits. This study evaluated the efficiency of different genomic prediction models given the complex population structure of multiple market classes present in dry bean breeding programs. A panel of 303 Middle-American breeding lines were genotyped with 3,026 markers and evaluated for white mold in the field. Prediction accuracy across models and subsets was moderate (0.3 - 0.36) given the population size. Furthermore, when fixed effect QTL were identified and implemented through GP + GWAS, 1-3 QTL increased prediction accuracy (0.36 - 0.4). These results indicate that genomic prediction is a promising screening tool in dry bean breeding for white mold resistance.

INTRODUCTION

Dry bean (*Phaseolus vulgaris* L.) is one of the most important grain legumes for human consumption worldwide, providing an important source of key dietary nutrients such as protein and fiber (Uebersax et al., 2023). However, biotic stress caused by fungal disease infection is among the main constraints that limit yield and increase management cost of dry bean production. White mold, conferred by the fungal pathogen *Sclerotinia sclerotiorum* (Lib.) de Bary is one of

the most destructive fungal diseases of dry bean, resulting in poor seed quality and reduced yield (del Río et al., 2004; Vasconcellos et al., 2017). In temperate regions it is considered the most yield limiting disease of dry bean production, causing up to 100% yield loss and may also result in significant seed quality decline in susceptible cultivars (Schwartz et al., 1987; Singh & Schwartz, 2010). Consequently, economic losses from white mold in the United States have exceeded $200 million yearly due to yield reduction and fungicide costs (Bolton et al., 2006).

Sclerotinia sclerotiorum is a necrotrophic fungus with a broad host range of over 400 species of plants (Boland & Hall, 1994). *Sclerotinia sclerotiorum* can be seed transmitted. However, the bulk of inoculum comes from melanized sclerotia that can overwinter in soil for five or more years (Schwartz & Singh, 2013). Around dry bean flowering time, *S. sclerotiorum* sclerotia germinates and forms a fruiting structure, a small cup shaped mushroom called an apothecia (Miklas et al., 2013). Once the apothecia has developed, asci can forcefully release ascospores into the plant canopy. These ascospores can infect the plant through wounds on the stem left by the senescent flowers and on other foliar parts, by penetrating the host cell walls (Bolton et al., 2006). Once colonized, infected plants often exhibit wilted leaves due to reduced vascular function. As disease development progresses, stems become brittle, and have a bleached appearance, often leading to severe plant lodging (Schwartz & Singh, 2013). Yield and seed quality decline occurs indirectly due to plant stress that reduces seed development or lost pods that abort entirely and directly due to colonization of bean pods that results in moldy, discolored, or shriveled seed. In severe infections, white mycelium can be observed on affected plant parts and whole plant death can occur (Schwartz & Singh, 2013).

Michigan is the second largest dry bean producer in the United States, producing over 400 million pounds of dry edible beans per year (USDA Crop Production Summary 2022). It also leads

the nation in the black, small red, and navy bean market classes in terms of acreage (USDA Crop Production Summary 2022). Michigan's temperate climate, with warm humid summers, is highly conducive to fungal diseases. Among the fungal diseases present in Michigan, growers consider white mold the most serious disease impacting dry bean production. In the 2022 annual production practices survey, Michigan farmers and crop advisors regarded white mold as the number one disease of dry bean production that needs further research to increase yield and reduce fungicide costs (MBC, 2022).

Managing white mold can be challenging due to the durability and extended viability of sclerotia in the soil and broad host range of the pathogen (Bolton et al., 2006). Common strategies include crop rotation, fungicide sprays, limiting nitrogen fertilizer and irrigation to reduce vegetative growth, wide row spacing, low density planting, and the development of low biomass, upright, and open canopy cultivars (Ender & Kelly, 2005; Miorini et al., 2017; Schwartz & Singh, 2013). These strategies focus on reducing sclerotial germination and ascospore development by altering the microclimate to reduce canopy humidity and temperature and therefore colonization ability (Schwartz & Singh, 2013). However, they are often not sustainable or economically feasible and reduce productivity of dry bean fields. For example, control of white mold through biomass management, wide row spacing, and reduced fertilizer and irrigation compromises yield per acre. Additionally, fungicide sprays are expensive and have adverse effects on the environment. Furthermore, these practices do not fully eliminate white mold infection due to the fungal virulence and overwintering ability. Therefore, the development of resistant cultivars is the most cost-effective, sustainable, and durable approach to combat white mold (Kolkman & Kelly, 2000; Miklas et al., 2001) along with appropriate cultural management practices as needed.

Breeding for white mold resistance in dry bean is challenging due to the quantitative inheritance method of resistance, which can be obtained through both physiological defense mechanisms and architectural avoidance. Disease avoidance mechanisms (Hoyos-Villegas et al., 2015; Miklas et al., 2001, 2013) are associated with plant architecture traits that impart a tall, upright growth habit, and porous plant canopy. Physiological resistance (Miklas et al., 2001; Terán & Singh, 2009) is associated with pathogen recognition, including utilizing reactive oxygen species as an initial defense mechanism and synthesis of pathogenesis-related proteins (Mamidi et al., 2016) Given the complex infection process of white mold, a multi-faceted quantitative resistance mechanism is needed for dry bean (Mamidi et al., 2016). Unfortunately, completely resistant varieties are unavailable and most known sources of genetic resistance to white mold are of Andean origin, usually from unadapted landraces and wild relatives, and from secondary gene pools (Schwartz & Singh, 2013; Singh et al., 2014; Vasconcellos et al., 2017). While low levels of resistance have been identified in Middle-American germplasm (Ender & Kelly, 2005; Hoyos-Villegas et al., 2015; Mkwaila et al., 2011), progress to develop white mold resistant cultivars has been hindered by the lack of high levels of resistance (Schwartz & Singh, 2013), difficulty in pyramiding resistance genes (Singh et al., 2014), low heritability for the trait (Fuller et al., 1984; Miklas et al., 2004; Park et al., 2001), and environmental dependency for field evaluation requiring highly managed disease nurseries under frequent overhead irrigation (Ender & Kelly, 2005). Furthermore, greenhouse studies provide an alternate method of measuring physiological resistance, but previous studies have failed to show strong correlation between field and greenhouse screening methods (Terán & Singh, 2009).

The complex quantitative inheritance mechanism of this trait has been demonstrated by the identification of at least 35 quantitative trait loci (QTL) with minor effects for resistance to white

mold in dry bean (Singh et al., 2014). This genetic architecture has posed challenges to dry bean breeders aiming to deploy marker-assisted selection (MAS) for QTL conferring partial resistance and low accuracy of genomic intervals resulting in negative linkage drag for yield and other traits (Miklas, 2007; Vasconcellos et al., 2017). To provide a better resolution, Vasconcellos et al., (2017) conducted a meta-QTL analysis and identified a total of nine-QTL as potential targets for MAS for partial resistance. While this study determined 9 major gene candidates that contribute to white mold resistance, the integration of an adequate level of white mold resistance in adapted dry bean germplasm is difficult because backcrossing many genes is inefficient and takes time even with the assistance of molecular markers (Lee, 1995).

Unlike breeding for qualitative resistance, breeding for quantitative resistance is more challenging because it requires multiple cycles of breeding and screening, leading to a gradual improvement of resistance in a breeding population over time. With the sheer number of QTL identified previously conferring many environmentally dependent resistance traits, it is no surprise that there has been difficulty pyramiding genes into one robustly resistant genotype with the desired agronomic traits. An integrated screening and recurrent selection method that considers all aspects of resistance and agronomic performance would greatly assist future cultivar development. Multiple breeding tools such as recurrent selection and the use of alternative populations such as multiparent intercrosses are one way to assist the identification and pyramiding of resistance genes (Escobar et al., 2022; Osorno et al., 2018). Recently, Escobar et al. explored integrated multi-parent crosses and gamete selection using a Multiparent Advanced Generation Inter-Cross (MAGIC) population, to facilitate mapping and breeding efforts, resulting in multiple partially resistant lines. This method crosses together multiple founder lines and cycles through several additional generations of crossing, resulting in offspring with multiple recombination events, often

leading to improved results that maximize diversity. This and other emerging breeding tools will assist future breeding for complex traits.

One such tool is genomic prediction/selection (GP/GS). Genomic prediction creates a unique opportunity to select and pyramid major and minor alleles conferring resistance to complex diseases (Merrick et al., 2021; Poland & Rutkoski, 2016; Tiede & Smith, 2018). This established tool in Marker Assisted Selection (MAS) utilizes genome wide markers and phenotypic data to train a linear statistical model to predict and make selections based on genomic estimated breeding values (GEBVs) for a trait. In contrast to MAS, genomic prediction does not identify significant markers and estimate individual marker effects. Instead, all marker effects are estimated simultaneously and used to accurately predict overall breeding values for a trait (Jannink et al., 2010). When properly implemented, genomic prediction has the potential to improve breeding program efficiency and reduce phenotyping costs when screening for complex traits. Genomic prediction has assisted breeding efforts for quantitative disease and pest resistance traits in many crop species including wheat: (Arruda et al., 2016; Juliana et al., 2017, 2022; Larkin et al., 2021; Merrick et al., 2021; Odilbekov et al., 2019; Rutkoski et al., 2012; Sarinelli et al., 2019) , dry bean (Diaz et al., 2021; Shi et al., 2021a), soybean (Bao et al., 2015; de Azevedo Peixoto et al., 2017; Đorđević et al., 2019; Duhnen et al., 2017; Hemingway et al., 2021; Shi et al., 2021b; Wen et al., 2018), and other crop species. Specifically, when compared to MAS or phenomic selection alone, genomic prediction has shown to be more effective for pyramiding quantitative traits controlled by many small effect QTL (Heffner et al., 2010, 2011; Massman et al., 2013; Merrick et al., 2021; Zhang et al., 2016).

Many previous studies of the genetic control of white mold in dry bean fail to account for the many small effect QTL unable to be detected by genome wide association studies (GWAS)

that are likely controlling resistance to this trait. To date, there have been no major resistance genes identified for white mold resistance, unlike some disease traits such as *Fusarium* head blight in wheat (Larkin et al., 2021). Since genomic prediction utilizes all markers spread across the entire genome to make selections, it has the power to detect small effect QTL conferring resistance that would otherwise be overlooked by GWAS (Jannink et al. 2010; Meuwissen et al. 2013). Genomic selection for white mold resistance has been studied in soybean diversity panels (de Azevedo Peixoto et al., 2017; Wen et al., 2018) with moderate to high prediction accuracies (0.4-0.7) suggesting that this warrants further research into the validity to assist breeding for resistance in dry bean.

Another benefit of genomic prediction in breeding for complex traits controlled by many major and minor QTL is that major QTL controlling a trait of interest can be identified using a GWAS and implemented in genomic prediction as fixed effects, allowing for further accuracy in selection (Merrick et al., 2021). Fixed QTL can be selected based on previous research or identified through *de novo* GWAS of the training population. GS + *de novo* GWAS involves two main stages where in the first stage GWAS is conducted on individuals in the training set to identify fixed QTL to be implemented in genomic prediction of the testing set (Bian and Holland, 2017; Haile et al., 2021; Sarinelli et al., 2019; Spindel et al., 2016). For example, Bian and Holland 2017 and Spindel 2016 both found that GS + *de novo* GWAS outperformed all other models tested. A simulation study by (Rice and Lipka, 2019) evaluated the effect of the number of QTL implemented across multiple genetic architectures and trait heritability and observed increased, decreased, and mixed (increase then decrease or decrease then increase) effect on prediction accuracy depending on the trait and number of fixed QTL. This study emphasizes that a varying number of fixed effect QTL should be tested for each trait before implementation.

Genomic prediction is more complex to implement when the crop of interest has multiple sub-classes. Dry beans are one such crop because their breeding programs are funneled into many smaller programs based on market standards for over 10 distinct seed classes. While there is some cross breeding between classes (ex: black by navy), this is discouraged due to the possibility to transmit deleterious seed traits. Due to distinct market class traits, the number and genetic composition of market classes added to the training population may have a significant effect on prediction accuracy. Since this is the first study to date evaluating genomic prediction as a tool to screen for white mold resistance in dry bean breeding lines, a major objective was to determine the ideal population composition to optimize prediction accuracy.

Given these factors, the primary objective of this study was to evaluate the potential of genomic prediction as a screening tool to breed for white mold resistance and determine how it could be efficiently used in a dry bean breeding program. To do so, we evaluated advanced breeding lines from the Michigan State University (MSU) dry bean breeding program belonging to three distinct major market classes in Michigan. Our specific objectives were to i) evaluate genomic prediction across and within major market classes of dry bean, ii) identify the optimal training population composition to increase prediction accuracy in the presence of distinct market classes and population structure, and iii) compare predictive abilities between GP alone and GP + *de novo* GWAS.

MATERIALS AND METHODS

Plant material

A set of 303 lines were used to evaluate the potential of using genomic prediction for white mold resistance. This panel consisted of advanced breeding lines from four market classes (black, navy, small red, and pink) developed by the MSU dry bean breeding program, 7 commercially

available lines grown commonly in Michigan, and the partially white mold resistant lines USPT-WM-12 (pinto) and G122 (cranberry) (Phillip N. Miklas, USDA). Detailed line information can be found in (**Table 1.5**).

All lines were evaluated for white mold resistance in the field. During the 2021 field season 176 lines were evaluated and during the 2022 field season, 127 lines were evaluated. Finally, 55 lines were added to the study that were previously evaluated in the National Sclerotinia Initiative multi-state national trials and 43 lines were evaluated in multiple years. The final training set consisted of 144 black, 117 navy, 32 red, 7 pink, 1 pinto, 1 cranberry, and 1 great northern line.

Phenotype data collection

All lines were grown under natural white mold infestation in a disease nursery at Montcalm Research and Extension Center in Montcalm County, MI. This location has consistent yearly white mold disease pressure and an overhead pivot irrigation system to promote disease infection. The lines were planted in an alpha-lattice design with three replicates using four row plots 6.1 m in length with 50cm row spacing. The outer two rows were planted with the white mold susceptible black bean line (Black Bear) to encourage white mold infection. The resistant, moderately resistant, and susceptible checks, G122, Bunsi, and Beryl, were added to adjust for local disease variation.

Plants were evaluated for white mold multiple times per season between the R7-R9 growth stages, beginning when white mold disease was first observed (around pod fill) and continuing through dry down. The best white mold rating to be used for genomic prediction was chosen based on heritability and variance for disease score. A plot-wise visual disease rating system was used to screen for white mold resistance in the field. The visual rating consisted of incidence and severity on a scale of 1 to 9, where a rating of 1 indicates no diseased plants and a rating of 9

indicates 80 to 100% diseased plants or 60 to 100% infected tissue as described in Miklas et al., 2001. Plots were trimmed to 4.9m prior to harvest and the center two rows were harvested with a Wintersteiger Classic plot combine (Wintersteiger AG, Austria). Yield data was obtained utilizing a Harvestmaster H2 Classic Graingage (Juniper Systems, Logan, UT) to record plot weight and moisture. Prior to data analysis, seed yield was standardized to 18% moisture. Standard agronomic traits including plant height, lodging score, days to flower, and maturity were also collected in the field.

Genotypic data collection and processing

Single-nucleotide polymorphism (SNP) data was collected for all lines in the training population. The tissue collection, DNA extraction, and genotyping procedures were as follows. Ten first trifoliate leaves from each line were collected and bulked. Leaves were desiccated using liquid nitrogen, placed in a -80 $^{\circ}$C freezer, and lyophilized. DNA was extracted using the Qiagen DNeasy plant mini kit following the manufacturers protocols and concentration standardized to at least 50 ug/ul. SNP chip sequencing using the Illumina Infinium BARCBean12k Bead chip was performed at the USDA-ARS, Soybean Genomics and Improvement Lab in Beltsville, Maryland. SNP calling for 11,929 markers was performed in the Illumina Genomestudio software (Genomestudio Software, 2023). Markers were filtered in the TASSEL trait analysis software, which removed any SNP with a minor allele frequency of less than 0.10 or more than 50% missing individuals (Bradbury et al., 2007). A principal component analysis (PCA) and genomic relationship matrix were developed utilizing the remaining markers. Imputation was performed in the rrBLUP package in R using the A.mat function and the impute=mean parameter to input missing data according to the population mean at each marker (Endelman, 2011). Realized

relationship matrices were also calculated using the A.matrix function within rrBLUP. These steps resulted in a matrix of ~3,026 polymorphic markers used in this study.

Phenotypic statistical analysis

A linear mixed model was fit using the ASREML package in the R coding language to calculate Best Linear Unbiased Estimators (BLUEs) for white mold disease score (Butler et al., 2023). The model was as follows:

$$\mathbf{Y_{ijk} = \mu + Line_i + Env_j + Rep(Env)_{kj} + (Line\ x\ Env)_{ij} + e_{ijk}} \quad \text{Eq. 1}$$

Where Y_{ijk} is the observed phenotype. μ is the overall mean, $Line_i$ is the fixed effect of the *i*-th line, Env_j is the random effect of the *j*th environment, $Rep(Env)_{kj}$ is the random effect of the *k-th* rep nested within the *j-th* environment, $(Line\ x\ Env)_{ij}$ is the random effect of interaction between the *i*-th line and *j*-th environment,, and e_{ijklm} is the random residual term.

To estimate broad sense heritability (H^2) for white mold resistance on an entry means basis, variance components were extracted fitting Equation 1 with all terms random. Heritability was estimated as follows:

$$H^2 = \frac{\sigma_G^2}{\sigma_G^2 + \frac{\sigma_{GE}^2}{n} + \frac{\sigma_e^2}{rn}} \quad \text{Eq. 2}$$

Where σ^2_G ,σ^2_{GxE} ,and , σ^2_e are the genotype, genotype by environment interaction, and error variances, n is the number of environments and r is the number of reps. Genomic heritability for white mold resistance was also calculated by first using the marker matrix to generate a kinship matrix using the A.mat function in rrBLUP and then inputting the kinship matrix and the BLUEs for white mold disease score into the function marker_h2 means in the

AGHmatrix package (Amadeu et al., 2023; Endelman, 2011). Genomic heritability is the proportion of variance of phenotypes explained by a regression on a set of markers (De Los Campos et al., 2015).

Genomic predictions for white mold disease

The accuracy of genomic prediction was evaluated using two models (rrBLUP and GBLUP) implemented in the rrBLUP package in the R coding language (Endelman, 2011). All genomic prediction models and subsets were evaluated using 5-fold cross-validation (CV) repeated 20 times. For each round of CV, the datasets were divided into equal sets of five. Four of the sets were used as the training set, while the remaining set was used as a validation set. BLUEs were predicted for individuals in the validation set and prediction accuracy was evaluated as the correlation between the observed BLUEs and predicted genomic BLUEs. The rrBLUP model was expressed as follows:

$$\mathbf{Y = Xb + Zu + e} \qquad \text{Eq. 4}$$

Where **y** is the vector of BLUEs for disease score; **X** is the incidence matrix for the fixed effects **b** is the vector of fixed effects; **Z** is the incidence matrix for the genotype effects; **u** is the vector of marker effects (BLUEs), assumed to follow multivariate normal distribution such that u∼*N*(0,Iσ2u). Finally, **e** is a vector of residual errors assumed to be independent and identically distributed according to *N*(0,σ2e). GBLUP utilizes the same formula with the following differences. In GBLUP **u** is the vector of additive genotype effects (breeding values), assumed to follow multivariate normal distribution such that **u**∼*N*(0,Kσ^2a) where **K** is the realized kinship matrix and σ^2_a is the additive genetic variance.

Population Subsets

Dry bean breeding programs are unique because they consist of over 10 separate market class subsets with distinct characteristics (and therefore separate breeding populations). To simulate a breeding scenario of employing genomic prediction to screen for white mold disease resistance in a dry bean breeding program, multiple market class subsets were tested. For simplicity, red and pink beans are considered the same market class, both referred to as red. Four subsets (entire population, black, navy, and black + navy) used the respective market classes as the training and validation population for cross validation. The entire population subset included all lines in the training set (n=303) whereas the black, navy, and black + navy subsets included the respective market classes only. Red and pink beans were not tested as subsets as there were only 39 lines total. Two subsets (black predict navy and navy predict black) used one market class as the training set and another as the validation set.

Genome-Wide Association Analysis

Association analysis was performed on the entire training set to evaluate the effectiveness of including fixed effect markers on the accuracy of the genomic prediction through GP + *de novo* GWAS. With GEBVs for white mold disease score, GWAS was conducted in the Farm CPU model of the Genome Association and Prediction Integrated Tool package (GAPIT) version 3 in R studio (Wang & Zhang, 2021). The same SNP marker dataset and phenotype dataset was used for GWAS as for genomic prediction. To avoid bias in the calculation of prediction accuracy, the identification of markers to use as fixed effects was based on 20 cycles of 5-fold cross validated GWAS using each training set of lines only following a similar protocol to Haile 2021 and Sarinelli 2018. The Fixed and Random Circulating Probability Unification (Farm CPU) model was chosen because it uses the bin approach to avoid selecting markers from the same location and it accounts for

population structure using two principal components and kinship among individuals (Liu et al., 2016). P-value inflation was assessed using a QQ-plot. Significance was calculated using the Benjamini-Hochberg FDR procedure at p-value threshold of p=0.05. The average effect on white mold score for each significant QTL over all calls was also calculated within GAPIT. The top 1-7 QTL for each run of GWAS were called as fixed effects in each corresponding run of genomic prediction implemented in the rrBLUP model and mean prediction accuracies were obtained resulting from CV (100 runs for each QTL threshold).

RESULTS

White Mold Disease

A continuous distribution of BLUEs for white mold disease score was observed when evaluating all years of data combined, which ranged from 2.07 to 8.41 (**Figure 1.1a**). Disease scores ranged from 3.07 to 7.34 for the black market class, 2.07 to 8.41 for the Navy market class, and 2.29 to 7.95 for the Red and Pink market classes (**Figure 1.1b**). The two resistant, one moderately resistant, and two susceptible checks, USPT-WM-12, G122, Bunsi, Beryl, and Black Bear had scores of 4.83, 4.17, 6.16, 7.62, and 6.41, respectively (**Table 1.4**). Broad sense heritability (H^2) estimates for disease severity were moderate at 0.52. A significant correlation (-0.27) was identified between white mold resistance and yield in the combined dataset. Variance components for white mold and yield can be found in (**Table 1.6, Table 1.7**).

Training Population Structure

The population structure of the entire training set was evaluated with a principal component analysis (PCA) and a genomic relationship matrix (GRM). The first two principal components accounted for 15% of the phenotypic variation (**Figure 1.2a**). The PCA was able to separate three distinct clusters and showed admixture between two clusters. The black and navy market class

clusters overlap on the right side of the PCA, whereas the red/pink cluster is isolated to the left-hand side. This is supported by the GRM, where three darker color clusters correspond to the three major market classes used in the training population (**Figure 1.2b).** Genomic heritability was low-moderate at 0.31.

Genomic Prediction Accuracy

The accuracy of genomic prediction models was evaluated using rrBLUP and GBLUP across and within multiple market class subsets. Overall mean prediction accuracy ranged from 0.12-037 for rrBLUP and 0.03 to 0.38 for GBLUP when using the entire training set and across all subsets (**Figure 1.3, Table 1.1**). In the four sets utilizing the respective market classes as the training and testing set (Entire Population, black, navy, black + navy) prediction accuracy ranged from 0.30-0.38 and prediction accuracy was similar between rrBLUP and GBLUP. The navy bean subset had the highest prediction accuracy overall, followed by the entire training set. In the two subsets where different market classes were used for the training and testing sets (black predict navy and navy predict black) prediction accuracy ranged from 0.03-0.25. The black predict navy subset had higher prediction accuracies than the navy predict black subset. The black predict navy subset had higher prediction accuracy using GBLUP and the navy predict black subset had a higher prediction accuracy using rrBLUP. Variance in prediction accuracy across individual runs of genomic prediction was highest when the navy subset was used to predict the black subset and lowest when the entire population was used as the training and testing sets.

Genomic Prediction with Fixed Effect QTL

Association analysis was performed on the entire training set (n=303) to evaluate the effectiveness of fixed effect markers on the accuracy of the genomic prediction through GP + *de novo* GWAS. To avoid bias in the calculation of prediction accuracy, the identification of markers to use as fixed

effects was based on 20 cycles of 5-fold cross validated GWAS using the corresponding training set only. The top 1-7 QTL for each run of GWAS were called as fixed effects in each corresponding run of genomic prediction implemented in the rrBLUP model.

Significant QTL were identified on all chromosomes across GWAS cycles. The top 10 most frequently called QTL were identified on chromosomes 2,4,6,7,8, and 11 (**Table 1.2**). The most frequently called QTL was on chromosome 11 (sc00007ln1695141_1432256_A_C_13462529) with 44 calls total across cycles of CV. Prediction accuracy rose from 0.36 to 0.40 when 1-3 QTL were implemented as fixed effects in genomic prediction (**Figure 1.4**, **Table 1.3**). When 4-7 QTL were implemented, prediction accuracy decreased from 0.4 to 0.22. Mean effect of the top 10 most identified fixed QTL on phenotype over all cycles ranged from -0.81 - 0.7 with a negative sign referring to the minor allele being favorable. The most called SNP on chromosome 11 (sc00007ln1695141_1432256_A_C_13462529) also had the largest mean effect (-0.81) with the minor allele being favorable.

DISCUSSION

In this study, advanced dry bean breeding lines from distinct market classes of the MSU dry bean breeding program were used as a training population to evaluate the potential of genomic prediction as a screening tool for white mold resistance. Specifically, we aimed to understand whether random sets of four-fifths of the lines could be used to predict white mold disease resistance in the remaining one-fifth. Our results indicated moderate cross-validated prediction accuracies (0.3-0.38) given the small sample size in the entire training population and population subsets when the same market class was used as both the training and testing population, implying that genomic prediction for white mold resistance within Middle-American breeding panels is

promising and can be implemented by dry bean breeding programs. Although prediction accuracy is lower than previous studies utilizing the same cross validation methods, these studies had a larger training population size, different population composition, and were focused on different traits and crops. As the size of the training population increases, we expect prediction accuracy to rise as has been confirmed by multiple studies (Edwards et al., 2019; Fernández-González et al., 2023; Sarinelli et al., 2019).

Diversity and Population Structure

We observed moderate to severe white mold disease severity in the field yearly, which was aided by overhead irrigation and the presence of a spreader genotype. Broad sense heritability of white mold resistance under disease pressure in the field was moderate which suggests selections for white mold resistance on this population would be effective, considering the environmental dependance of a complex quantitative disease trait such as white mold. Our population had significant diversity for white mold resistance in the field, within and among market classes ranging from about 2-8 on a 1-9 scale. Three distinct clusters were observed via PCA which corresponded to the three market classes present in the population with red beans being the most distinct from black and navy beans. This relationship between individuals is likely due to distinct characteristics among the market classes and due to the crossing scheme used to develop the population. Red, pink, pinto and great northern beans belong to the Jalisco sub-race and black and navy beans belong to the Mesoamerican sub-race within the Middle-American gene pool (Mensack et al., 2010). Additionally, the MSU dry bean breeding and genetics program frequently performs black by navy crosses to develop new breeding lines, but red/pink beans are not commonly crossed with the two other classes, which could have contributed to the observed structure.

Genomic Predictions Across Models and Subsets

A secondary objective of this study was to identify the optimal training population to increase prediction accuracy in the presence of distinct dry bean market classes. When evaluating genomic prediction accuracies among market class subsets, we found that sample size and population structure both affected prediction accuracy. This is supported by the fact that the entire population subset and the navy subset had the two highest prediction accuracies across both the rrBLUP and GBLUP models. Population size was highest in the entire population subset and genetic relatedness was higher in the navy subset vs the entire population which included three market classes. Overall, population structure affected prediction accuracy more than population size, as reflected by the navy subset having the highest prediction accuracy, followed by the entire training population. This result is like previously published manuscripts, for example (Duhnen et al., 2017) in soybean, observed higher prediction accuracies when early and late genotypes were considered separately rather than when the entire population was considered. Another future direction would be to utilize a training population optimization algorithm or sparse selection index to select the optimal genotypes to increase prediction accuracy in the presence of multiple market classes (Fernández-González et al., 2023; Isidro et al., 2015; Lopez-Cruz & de los Campos, 2021). Furthermore, across most subsets the difference in prediction accuracy between rrBLUP and GBLUP was minimal. These genomic prediction models are similar, with the main difference being the incorporation of the genomic relationship matrix in GBLUP in replacement of the pedigree relationship matrix. If the pedigree relationship matrix is equal to the genomic relationship matrix, then the two models should have the same accuracy. The findings from this research show that the pedigree relationship and genomic relationship are similar in this population.

To this effect, when one market class was used to predict another market class, prediction accuracy dropped significantly. This indicates that to obtain a high genomic prediction accuracy in a dry bean breeding program, the training population and validation population should consist of the same market classes. Even in the case of black and navy beans, where the two market classes are frequently crossed, there are distinct characteristics of each market class that lead to insufficient predictions.

Integration of Fixed Effect QTL

Another secondary objective was to compare prediction accuracies between genomic prediction and GP + *de novo* GWAS. We observed increased prediction accuracy when 1-3 QTL were integrated as fixed effects through GP + *de novo* GWAS and a decrease in prediction accuracy when 4+ QTL were integrated. Previous studies have observed an increase in prediction accuracy when utilizing fixed effect QTL in GP + *de novo* GWAS such as in rice (Spindel et al., 2016) and wheat (Haile et al., 2021). Similar to results observed in the simulation study by Rice and Lipka 2019, prediction accuracy initially increased then decreased when 4+ QTL were added. Interestingly, although significant QTL were identified, all had a relatively low contribution to overall variation in resistance (**Table 1.2**) highlighting the likelihood that this trait is controlled by many small effect QTL rather than large effect R genes.

Limitations

One primary limitation in our study to note is the small size of the training population. Many public breeding programs are limited by sample population due to the number of lines moving through the breeding pipeline in a generation. In the MSU dry bean breeding and genetics program ~100-150 lines are advanced to the preliminary and advanced trial stage yearly. This training population will be updated with these lines and prediction accuracies are expected to

increase as a result. Additionally, our training population contained the three most important market classes in Michigan, prediction accuracies and genetic architecture for other market classes require further investigation.

The low to moderate prediction accuracy in all population subsets could have been due to multiple factors including sample size, marker density, and low heritability of the trait. One other important aspect of the fixed effect QTL identified using GWAS is the high possibility that our current population of breeding lines isn't sufficient to cover the breadth of resistance sources for white mold in dry bean. It is also likely that rare alleles in our population were filtered out. As the population size increases, more QTL conferring resistance will likely be identified and prediction accuracy will increase. The genotyping method used was a high throughput, cost-effective whole genome method that would allow the program to process all individuals in each year's preliminary yield trial stage. Any reduction in prediction accuracy due to marker density was likely outweighed by the ability to develop a larger training population.

Applications to Dry Bean Breeding

Genomic selection is a useful tool for breeding and can outperform phenotypic selection and MAS when screening and pyramiding complex traits. White mold in dry bean is one such trait where genomic selection could be extremely beneficial to breed for resistance if implemented properly. Progress to develop white mold resistant cultivars has been greatly hindered by difficulty in pyramiding resistance genes and complexity of field evaluation. A major advantage of implementing genomic selection in a breeding program is the reduction of phenotyping. This is an important aspect for white mold resistance in dry bean especially because field screening requires a specialized disease nursery with overhead pivot irrigation and natural white mold infection. If genomic prediction accuracy in the training population is sufficient, genomic data could be

collected for members of the validation population and breeding values could be predicted, thus allowing for more lines to be screened per year. Another advantage of genomic prediction is it allows for earlier accurate selection of parents for the next generation of crosses therefore reducing breeding cycle time and increasing gain from selection. As selection for quantitative traits occurs in the advanced testing stages for many crops (including dry bean) this can have a large effect on genetic gain, effectively skipping 1-4 years of testing.

CONCLUSION

Complex fungal diseases such as white mold are a major constraint in dry bean production and the identification of QTL and development of resistant cultivars for white mold resistance in dry bean has been historically slow due to many factors. This study aimed to test genomic prediction as a novel method for the development of resistant cultivars. Implementation of genomic prediction in a plant breeding program relies on the development of a robust training population which requires consideration of multiple factors including size, marker density, and population structure. Results from this analysis indicate that it is possible to obtain moderate prediction accuracy in dry bean in the presence of population structure and multiple market classes, if sample size is adequate. The training population will continue to be updated, modified, and validated yearly to further increase prediction accuracy. The results from this research will assist plant breeders in the development of training populations for genomic prediction of highly quantitative disease resistance traits.

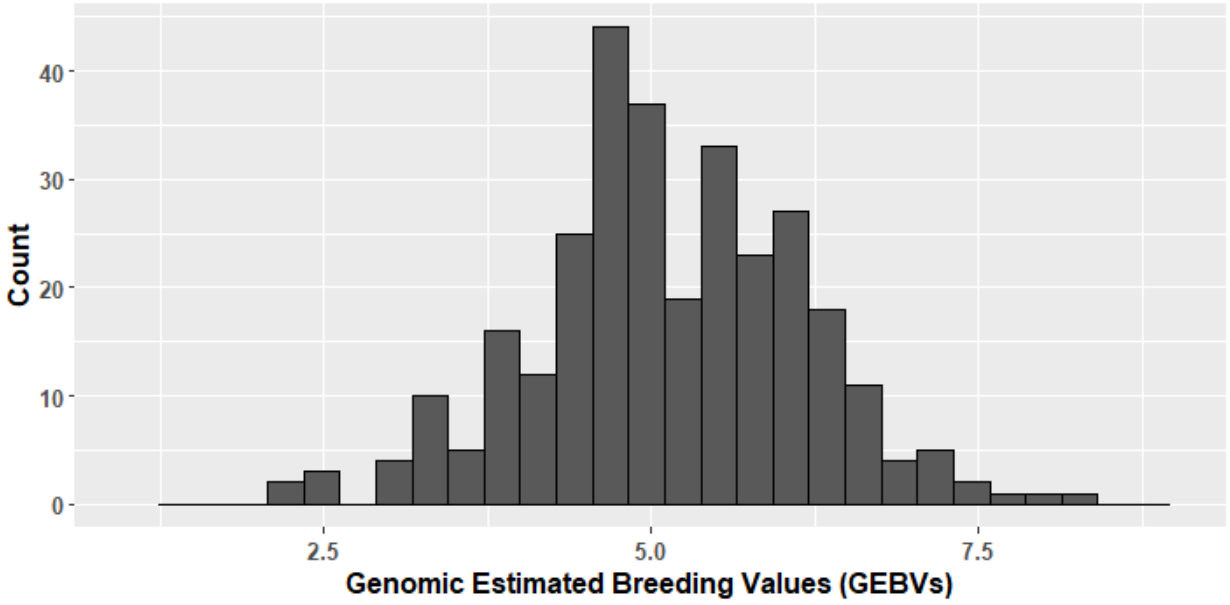

Figure 1.1a: Phenotypic distribution of genomic estimated breeding values (GEBVs) for the entire training set.

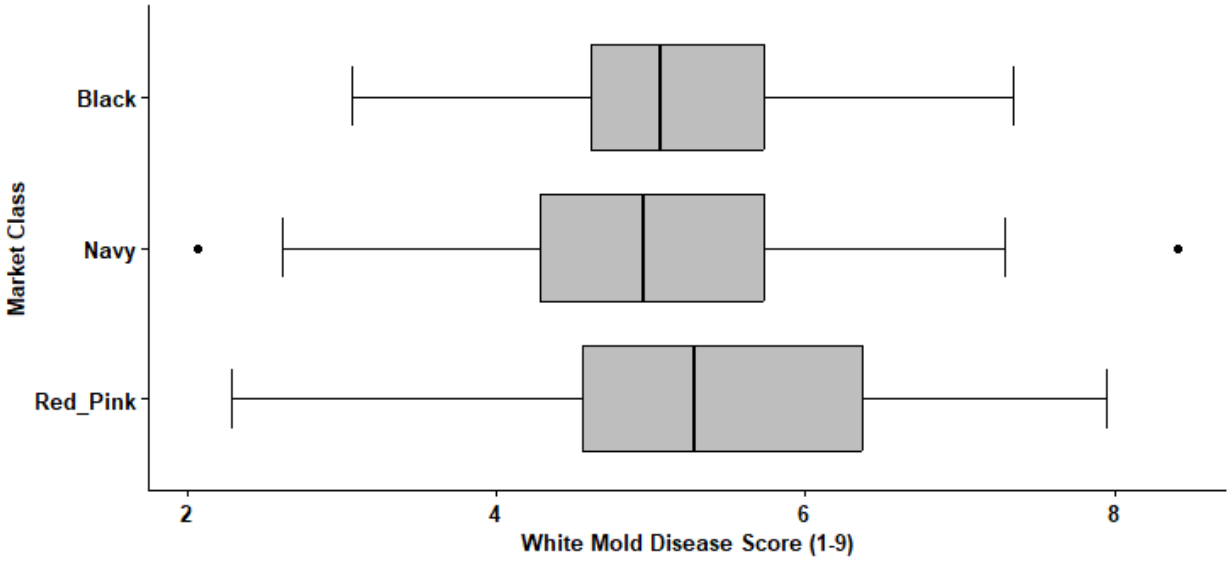

Figure 1.1b: Phenotypic distribution of genomic estimated breeding values (GEBVs) for the entire training set by market class.

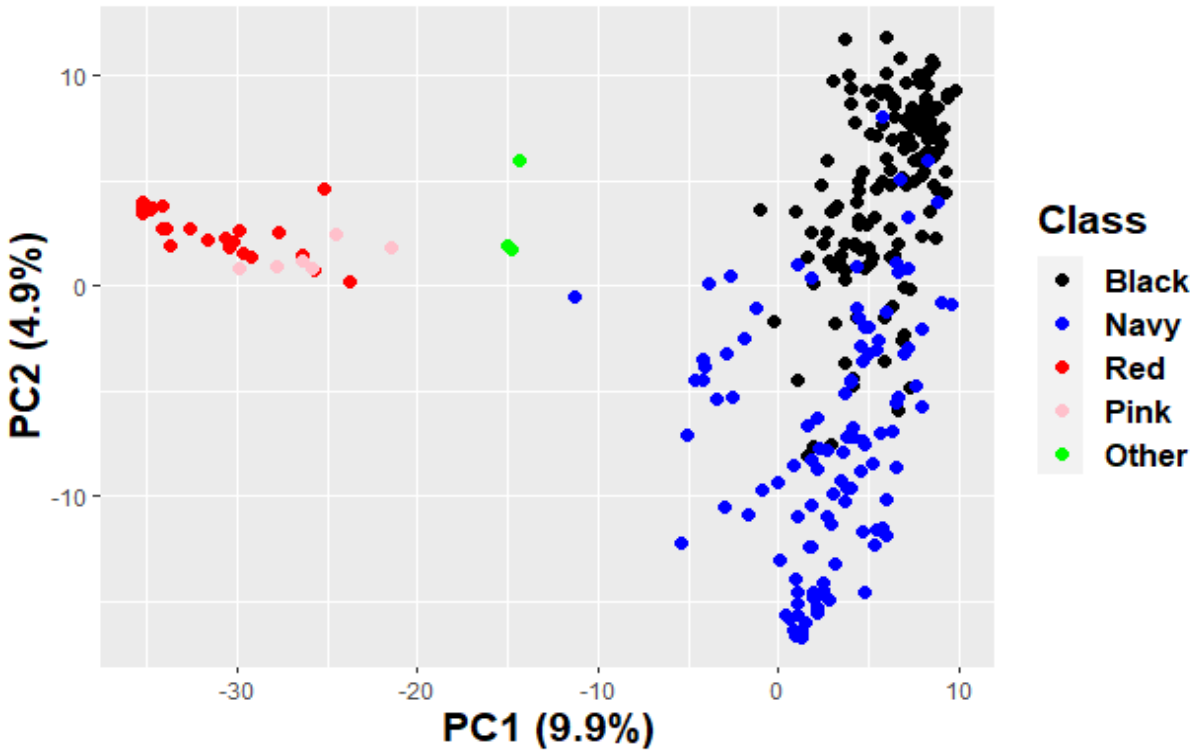

Figure 1.2a: Principal component analysis. The first two principal components accounted for 15% of the phenotypic variation. The PCA was able to separate three distinct clusters and showed admixture between two clusters. The black and navy market class clusters overlap on the right side of the PCA, whereas the red/pink cluster is isolated to the left-hand side.

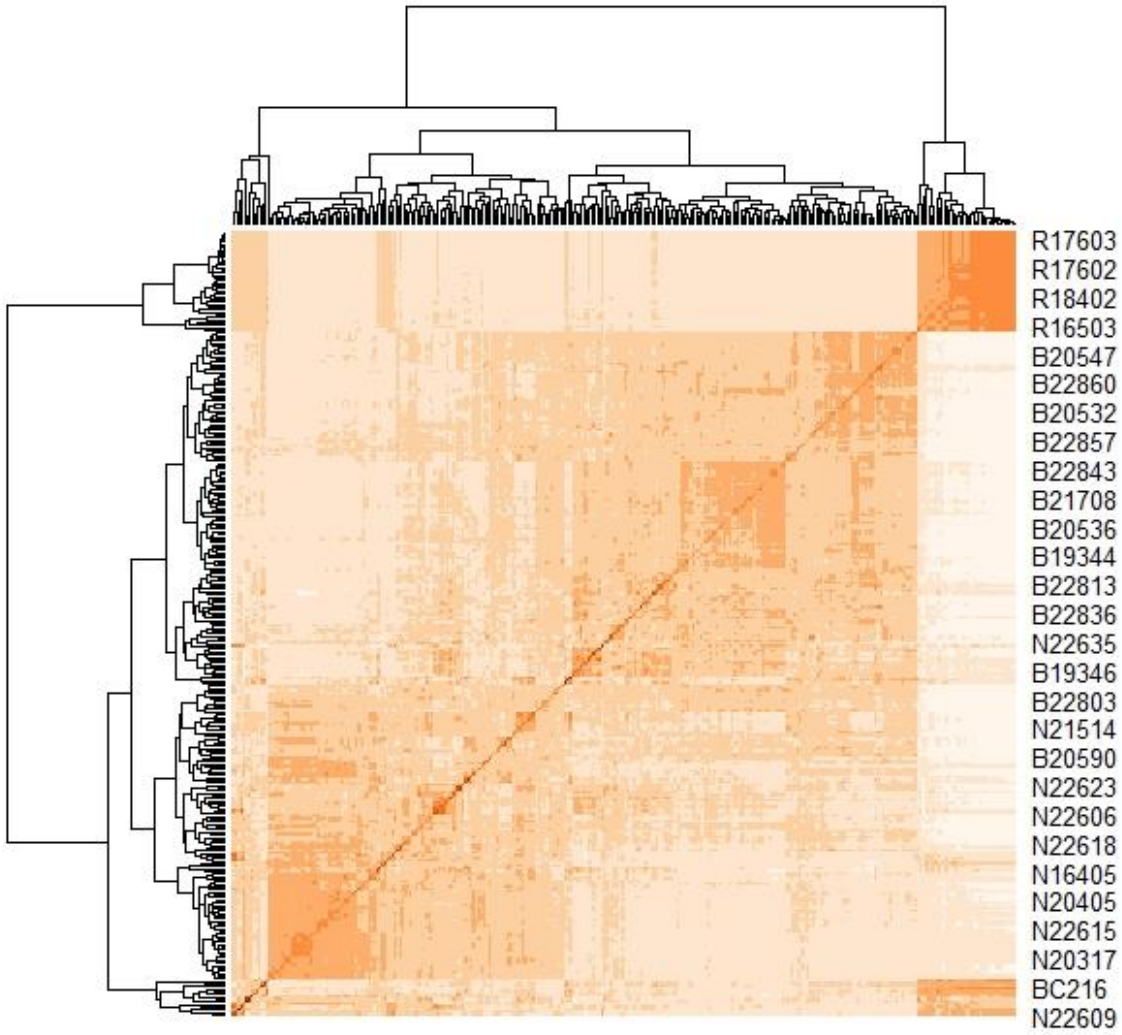

Figure 1.2b: Genomic relationship (proportion of the genome shared) among individuals in the training population (n=303). The Y axis represents the genotyped lines ordered by market class with red/pink, followed by black and navy. The darker the color, the more highly related the lines, with white = no relationship to dark orange (e.g. the diagonal) genomic relationship = 1.

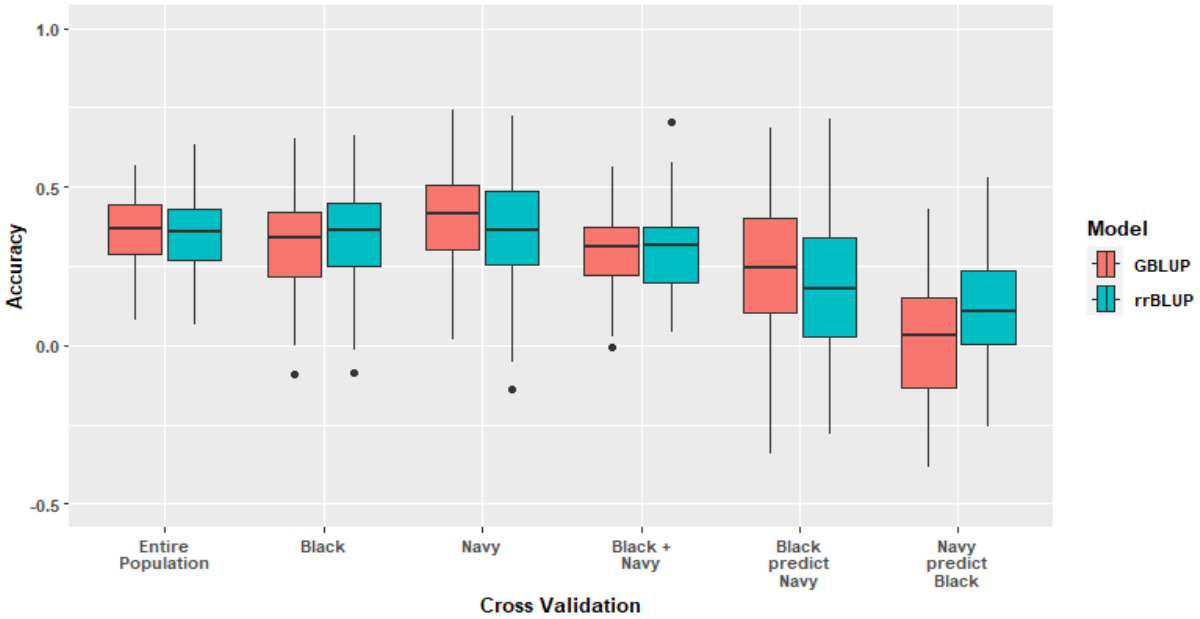

Figure 1.3: Comparison of genomic prediction accuracy of 5-fold CV repeated 20 times, for two models rrBLUP and GBLUP, over all training population subsets (entire population, black, navy, black + navy, black predict navy, and navy predict black).

Table 1.1: Average genomic prediction accuracies for 5-fold cross validation repeated 20 times across models and subsets.

Subset	Model	Average Accuracy
Entire Population	RrBLUP	0.35
	GBLUP	0.36
Black	RrBLUP	0.33
	GBLUP	0.32
Navy	RrBLUP	0.37
	GBLUP	0.38
Black + Navy	RrBLUP	0.30
	GBLUP	0.30
Black predict Navy	RrBLUP	0.18
	GBLUP	0.25
Navy Predict Black	RrBLUP	0.12
	GBLUP	0.03

Table 1.2: Top 20 most frequently called SNPs over 5-fold cross validated GWAS repeated 20 times by chromosome and position. Mean effect is the average effect on phenotype over runs of cross validation.

SNP	Chromosome	Position (Mb)	Mean Effect	# of Calls
Chr02_41657167_A_G	2	41657167	0.71	13
sc00687ln167302_103360_G_A_266954597	4	13213188	0.52	22
Chr06_12181540_G_A	6	12181540	-0.61	6
Chr06_16668700_G_T	6	16668700	-0.61	9
Chr07_3905254_C_T	7	3905254	0.61	4
sc00394ln266395_95537_T_C_204849189	7	32697386	0.38	13
sc00093ln620690_104379_A_G_86490862	7	33306574	0.35	5
sc00146ln499601_116735_C_T_115838631	8	1525152	0.41	14
sc00187ln435150_54957_C_T_135207180	8	62919922	0.34	6
sc00007ln1695141_1432256_A_C_13462529	11	4741435	-0.81	44

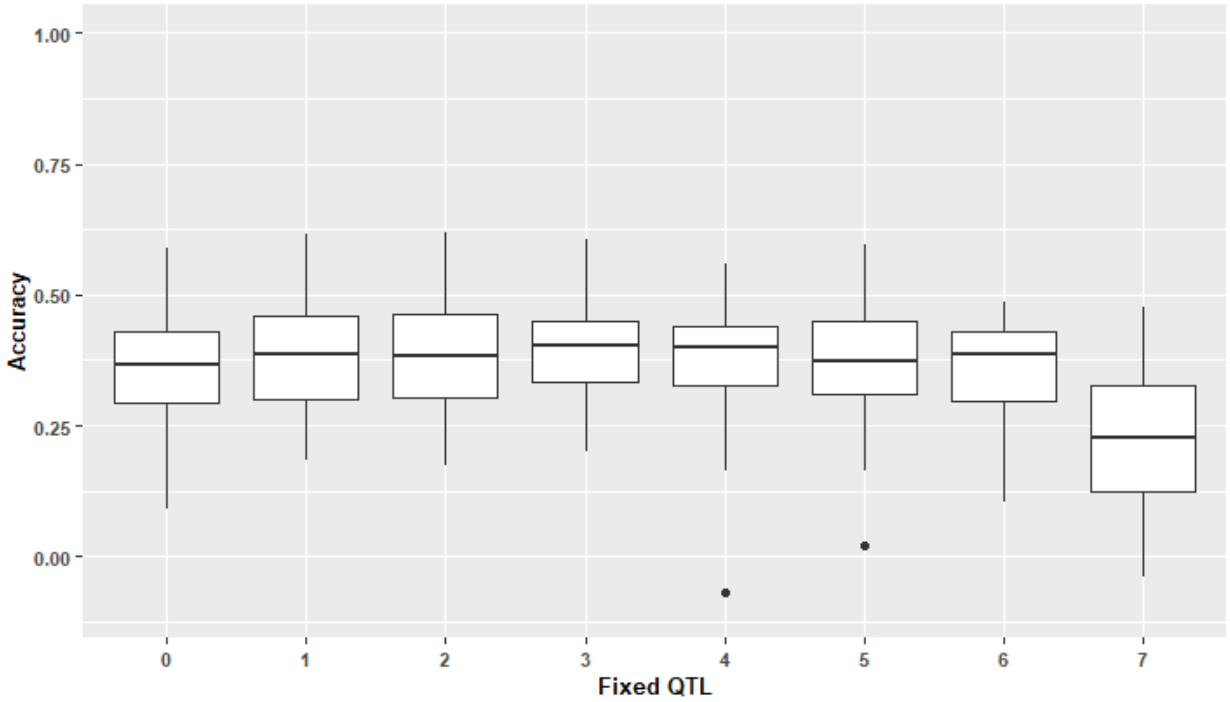

Figure 1.4: GWAS + de novo genomic prediction prediction accuracies over 20 replicates of 5-fold cross validation for 1-7 integrated fixed QTL implemented in rrBLUP.

Table 1.3: Average GWAS + de novo genomic prediction prediction accuracies over 20 replicates of 5-fold cross validation for 1-7 integrated fixed QTL implemented in rrBLUP.

# of QTL	**0**	**1**	**2**	**3**	**4**	**5**	**6**	**7**
Average Prediction Accuracy	0.36	0.39	0.39	0.40	0.37	0.37	0.35	0.22

Table 1.4: Ranking of BLUEs for White Mold and Yield.

Line	White Mold	Yield (CWT_Acre)	Yield Ranking
N22631	2.07	27.72	25
S08418	2.29	26.32	34
N21514	2.62	25.55	47
N19277	2.62	25.48	49
N21519	2.62	15.42	247
N21521	2.95	19.13	179
N21529	2.95	17.22	220
B22855	3.07	23.04	82
B22865	3.07	19.7	162
B18201	3.17	22.52	94
B20590	3.29	27.32	28
B20642	3.29	26.09	38
N20384	3.29	19.35	172
N21520	3.29	18.41	199
N19226	3.29	16.32	230
N21528	3.29	15.58	244
N21511	3.34	23.84	68
N22622	3.4	28.85	16
N18117	3.41	22.52	94
B20547	3.51	23.7	72
N17504	3.56	24.64	57
N21503	3.62	18.79	186
N21531	3.62	16.82	224

Table 1.4 (cont'd)

N21534	3.62	16.18	235
B22861	3.74	28.29	19
N22605	3.74	22.08	109
N22637	3.74	20.61	148
B16506	3.8	20.9	137
B19345	3.82	20.61	148
N18103	3.9	19.16	178
R17603	3.92	33.58	2
B20532	3.95	28.36	18
R18402	3.95	25.59	45
B20602	3.95	25.41	50
N20405	3.95	22	110
S19307	3.95	20.12	155
B20542	3.95	17.41	217
N21506	3.95	16.05	237
N20317	3.95	14.65	254
R17604	3.96	29.63	13
N20404	4.01	20.79	141
B17220	4.06	17.28	219
B22827	4.07	30.75	8
B22831	4.07	29.15	15
B22840	4.07	22.5	95
N22638	4.07	15.15	250
B17691	4.13	34.12	1

Table 1.4 (cont'd)

B16507	4.13	33.49	3
B15430	4.14	31.41	6
B20591	4.18	21.96	111
R16503	4.19	16.5	228
B18204	4.22	26.25	36
R20629	4.29	28.07	22
N21515	4.29	24.67	55
S20405	4.29	24.41	63
B21707	4.29	23.79	69
R20667	4.29	23.25	78
B21715	4.29	22.14	106
N19253	4.29	20.73	144
N21522	4.29	20.61	148
N21505	4.29	20.55	150
N20401	4.29	19.93	158
B20616	4.29	19.58	165
N21524	4.29	17.78	214
B20627	4.29	14.12	259
N17506	4.33	24.07	66
B20599	4.34	25.67	43
B15451	4.34	22.63	92
B22826	4.4	30.9	7
B22857	4.4	21.76	117
B22834	4.4	21.52	123

Table 1.4 (cont'd)

N22632	4.4	17.15	222
N14229	4.43	26.11	37
I96417	4.48	13.03	269
I09203	4.49	28.02	23
N19285	4.49	18.67	191
B17536	4.54	21.33	126
I11264	4.59	23.53	74
B21706	4.62	30.74	9
N20352	4.62	30.49	10
R17602	4.62	30.24	11
R20639	4.62	25.87	40
B21720	4.62	23.07	81
B21709	4.62	22.68	90
N21507	4.62	22.58	93
N21509	4.62	22.58	93
B20538	4.62	22.32	103
B19339	4.62	21.71	118
N20391	4.62	21.68	119
B21705	4.62	21.16	131
B21714	4.62	20.88	138
B21724	4.62	19.33	174
N21533	4.62	18.87	182
N21502	4.62	18.83	184
N18105	4.62	18.75	188

Table 1.4 (cont'd)

N20335	4.62	18.7	190
N21526	4.62	18.13	204
N19223	4.62	17.94	209
N19252	4.62	14.54	255
B21716	4.62	13.79	262
B20536	4.68	27.45	26
N19248	4.68	22.52	94
B22823	4.74	27.99	24
B22825	4.74	27.4	27
B22862	4.74	24.99	52
N22619	4.74	24.45	62
B22838	4.74	23.1	80
N22616	4.74	22.65	91
B22863	4.74	22.41	100
B22835	4.74	22.11	108
B22841	4.74	21.87	113
N22630	4.74	21.64	121
N22610	4.74	21.54	122
B22853	4.74	21.21	130
B22815	4.74	20.55	150
N22621	4.74	19.91	159
B22839	4.74	18.81	185
N22617	4.74	16.77	225
N17505	4.82	20.95	134

Table 1.4 (cont'd)

S18904	4.82	20.77	143
B19330	4.82	18.57	193
USPT-WM-12	4.83	23.48	76
B21708	4.84	22.8	88
B04554	4.84	22.52	94
B18231	4.91	22.52	94
B10244	4.93	27.26	30
B21713	4.95	32.45	4
B20632	4.95	25.8	42
B18504	4.95	24.09	65
R20659	4.95	23.86	67
R20669	4.95	23.73	71
B21702	4.95	21.81	115
B20597	4.95	20.95	134
B21718	4.95	19.85	160
N21525	4.95	19.17	177
B19340	4.95	18.77	187
B21703	4.95	18.7	190
N21504	4.95	18.47	196
N20341	4.95	17.44	215
B21721	4.95	15.95	238
N21518	4.95	15.9	240
N21510	4.95	12.23	271
N21532	4.95	11.6	273

Table 1.4 (cont'd)

B16501	4.96	18.52	195
B17922	4.97	22.52	94
B19309	5.01	26.55	33
B16504	5.04	31.99	5
B22850	5.07	26.64	32
B22860	5.07	25.83	41
B22820	5.07	25.66	44
B22854	5.07	25.06	51
B22814	5.07	24.95	53
N22624	5.07	24.56	61
B22844	5.07	22.95	84
B22836	5.07	22.13	107
B22866	5.07	21.12	132
N22607	5.07	20.01	156
B22859	5.07	19.2	176
R12844	5.11	22.49	96
B15442	5.16	30.2	12
N14218	5.17	17.97	208
N19246	5.18	18.07	206
BC269	5.18	18	207
N20388	5.18	17.92	210
R20683	5.29	26.55	33
R20637	5.29	24.78	54
R20632	5.29	24.61	60

Table 1.4 (cont'd)

B21712	5.29	23.46	77
R20627	5.29	22.82	86
N21517	5.29	18.59	192
B20527	5.29	17.88	211
N21512	5.29	16.21	233
B20549	5.29	14.46	256
B20639	5.29	13.59	265
B19332	5.32	19.55	166
N18130	5.32	15.53	245
N20395	5.34	19.34	173
B22804	5.4	25.58	46
B22876	5.4	25.5	48
N22602	5.4	24.66	56
B22848	5.4	23.07	81
B22828	5.4	23	83
N22634	5.4	22.74	89
B22812	5.4	22.42	99
B22830	5.4	22.39	101
N22609	5.4	22.36	102
B22875	5.4	20.86	139
B22873	5.4	20.49	151
B22813	5.4	18.88	181
N22629	5.4	18.84	183
N22633	5.4	16.19	234

Table 1.4 (cont'd)

B19344	5.46	15.75	242
N11283	5.48	18.17	203
B18236	5.49	14.24	258
N15331	5.58	26.78	31
R20633	5.62	26.29	35
R20614	5.62	24.3	64
B21723	5.62	23.73	71
B20623	5.62	23.65	73
B21717	5.62	23.5	75
R20636	5.62	22.43	98
B20579	5.62	21.79	116
B19341	5.62	21.67	120
N19290	5.62	20.82	140
B21722	5.62	19.22	175
N21530	5.62	18.42	198
N20343	5.62	15.6	243
B20617	5.62	13.33	267
B21719	5.62	13.16	268
B21711	5.62	12.16	272
B21710	5.68	15.53	245
B22842	5.74	22.45	97
N22623	5.74	22.26	105
B22837	5.74	21.46	124
B22856	5.74	21.25	128

Table 1.4 (cont'd)

B22818	5.74	21.06	133
N22608	5.74	20.92	135
B22816	5.74	20.91	136
B22846	5.74	20.78	142
B22821	5.74	20.69	145
B22868	5.74	20.67	146
N22636	5.74	20.62	147
N22620	5.74	20.47	152
N22601	5.74	19.85	160
B22867	5.74	18.83	184
N18122	5.74	18.67	191
N22606	5.74	18.59	192
N22613	5.74	18.55	194
N22603	5.74	18.46	197
B22833	5.74	17.2	221
B22802	5.74	16.27	232
N19239	5.82	15.82	241
I81010	5.92	18.23	202
R20612	5.95	23.2	79
R19502	5.95	22.32	103
N21527	5.95	20.24	154
N19284	5.95	18.38	200
N20336	5.95	18.08	205
B19302	5.95	17.8	213

Table 1.4 (cont'd)

B20582	5.95	16.64	226
B20629	5.95	16.3	231
S18907	5.95	14.11	260
B20621	5.95	13.65	264
R17605	5.96	28.26	20
B22807	6.07	24.78	54
B22870	6.07	23.75	70
B22874	6.07	22.88	85
B22843	6.07	21.91	112
B22822	6.07	21.39	125
B22806	6.07	21.3	127
B22811	6.07	20.56	149
B22829	6.07	19.79	161
B22801	6.07	19.45	168
B22803	6.07	19.39	171
B22805	6.07	18.33	201
N22627	6.07	17.87	212
N22615	6.07	16.52	227
N22612	6.07	13.74	263
N15341	6.15	13.82	261
N16405	6.16	28.68	17
R20625	6.29	22.81	87
N20346	6.29	15.95	238
N19243	6.29	15.6	243

Table 1.4 (cont'd)

B20620	6.29	15.03	252
N21523	6.29	11.07	274
N19269	6.29	10.91	275
I17501	6.34	19.7	162
B19346	6.35	22.52	94
N16401	6.37	19.4	170
B22832	6.4	21.86	114
B22847	6.4	19.61	164
B22845	6.4	19.46	167
N22618	6.4	17.32	218
B22819	6.4	16.15	236
B22852	6.4	15.37	248
N22626	6.4	15.07	251
N22614	6.4	14.95	253
R98026	6.45	17.43	216
R20684	6.62	28.24	21
R20624	6.62	24.63	58
B21704	6.62	24.62	59
R20635	6.62	22.29	104
B21701	6.62	19.97	157
R20652	6.62	15.43	246
N20376	6.62	14.43	257
N21516	6.62	12.75	270
B22810	6.74	29.49	14

Table 1.4 (cont'd)

N22635	6.74	18.71	189
N22639	6.74	15.94	239
N18109	6.91	22.52	94
R20604	6.95	21.24	129
N21535	6.95	19.66	163
N21508	6.95	13.37	266
B22817	7.07	15.27	249
R20653	7.29	27.29	29
N21501	7.29	20.28	153
R20642	7.29	19.41	169
N21513	7.29	16.83	223
BC216	7.34	19.06	180
I13401	7.46	25.91	39
I89011	7.62	10.64	276
S20420	7.95	16.33	229
N18102	8.41	22.52	94

Table 1.5: Line, class, origin, and trial information for the entire training set.

Line	Class	Origin	Trial
B04554	Black	MSU	Historic/2021/2022
B10244	Black	MSU	Historic/2021/2022
B15430	Black	MSU	Historic
B15442	Black	MSU	Historic
B15451	Black	MSU	Historic
B16501	Black	MSU	Historic/2021
B16504	Black	MSU	Historic/2021
B16506	Black	MSU	Historic
B16507	Black	MSU	Historic
B17220	Black	MSU	Historic
B17536	Black	MSU	Historic
B17691	Black	MSU	Historic
B17922	Black	MSU	Historic
B18201	Black	MSU	Historic
B18204	Black	MSU	Historic/2021
B18231	Black	MSU	Historic
B18236	Black	MSU	Historic/2021
B18504 (Adams)	Black	MSU	Historic/2021/2022
B19302	Black	MSU	2021
B19309	Black	MSU	2021/2022
B19330	Black	MSU	Historic/2021
B19332	Black	MSU	Historic/2021
B19339	Black	MSU	2021

Table 1.5 (cont'd)

B19340	Black	MSU	2021
B19341	Black	MSU	2021
B19344	Black	MSU	Historic/2021/2022
B19345	Black	MSU	Historic/2021
B19346	Black	MSU	Historic
B20527	Black	MSU	2021
B20532	Black	MSU	2021
B20536	Black	MSU	2021/2022
B20538	Black	MSU	2021
B20542	Black	MSU	2021
B20547	Black	MSU	2021/2022
B20549	Black	MSU	2021
B20579	Black	MSU	2021
B20582	Black	MSU	2021
B20590	Black	MSU	2021
B20591	Black	MSU	2021/2022
B20597	Black	MSU	2021
B20599	Black	MSU	2021/2022
B20602	Black	MSU	2021
B20616	Black	MSU	2021
B20617	Black	MSU	2021
B20620	Black	MSU	2021
B20621	Black	MSU	2021
B20623	Black	MSU	2021

Table 1.5 (cont'd)

B20627	Black	MSU	2021
B20629	Black	MSU	2021
B20632	Black	MSU	2021
B20639	Black	MSU	2021
B20642	Black	MSU	2021
B21701	Black	MSU	2021
B21702	Black	MSU	2021
B21703	Black	MSU	2021
B21704	Black	MSU	2021
B21705	Black	MSU	2021
B21706	Black	MSU	2021
B21707	Black	MSU	2021
B21708	Black	MSU	2021/2022
B21709	Black	MSU	2021
B21710	Black	MSU	2021/2022
B21711	Black	MSU	2021
B21712	Black	MSU	2021
B21713	Black	MSU	2021
B21714	Black	MSU	2021
B21715	Black	MSU	2021
B21716	Black	MSU	2021
B21717	Black	MSU	2021
B21718	Black	MSU	2021
B21719	Black	MSU	2021

Table 1.5 (cont'd)

B21720	Black	MSU	2021
B21721	Black	MSU	2021
B21722	Black	MSU	2021
B21723	Black	MSU	2021
B21724	Black	MSU	2021
B22801	Black	MSU	2022
B22802	Black	MSU	2022
B22803	Black	MSU	2022
B22804	Black	MSU	2022
B22805	Black	MSU	2022
B22806	Black	MSU	2022
B22807	Black	MSU	2022
B22810	Black	MSU	2022
B22811	Black	MSU	2022
B22812	Black	MSU	2022
B22813	Black	MSU	2022
B22814	Black	MSU	2022
B22815	Black	MSU	2022
B22816	Black	MSU	2022
B22817	Black	MSU	2022
B22818	Black	MSU	2022
B22819	Black	MSU	2022
B22820	Black	MSU	2022
B22821	Black	MSU	2022

Table 1.5 (cont'd)

B22822	Black	MSU	2022
B22823	Black	MSU	2022
B22825	Black	MSU	2022
B22826	Black	MSU	2022
B22827	Black	MSU	2022
B22828	Black	MSU	2022
B22829	Black	MSU	2022
B22830	Black	MSU	2022
B22831	Black	MSU	2022
B22832	Black	MSU	2022
B22833	Black	MSU	2022
B22834	Black	MSU	2022
B22835	Black	MSU	2022
B22836	Black	MSU	2022
B22837	Black	MSU	2022
B22838	Black	MSU	2022
B22839	Black	MSU	2022
B22840	Black	MSU	2022
B22841	Black	MSU	2022
B22822	Black	MSU	2022
B22823	Black	MSU	2022
B22825	Black	MSU	2022
B22826	Black	MSU	2022
B22827	Black	MSU	2022

Table 1.5 (cont'd)

B22828	Black	MSU	2022
B22829	Black	MSU	2022
B22830	Black	MSU	2022
B22831	Black	MSU	2022
B22832	Black	MSU	2022
B22833	Black	MSU	2022
B22834	Black	MSU	2022
B22835	Black	MSU	2022
B22836	Black	MSU	2022
B22837	Black	MSU	2022
B22838	Black	MSU	2022
B22839	Black	MSU	2022
B22840	Black	MSU	2022
B22841	Black	MSU	2022
B22842	Black	MSU	2022
B22843	Black	MSU	2022
B22844	Black	MSU	2022
B22845	Black	MSU	2022
B22846	Black	MSU	2022
B22847	Black	MSU	2022
B22848	Black	MSU	2022
B22850	Black	MSU	2022
B22852	Black	MSU	2022
B22853	Black	MSU	2022

Table 1.5 (cont'd)

B22854	Black	MSU	2022
B22855	Black	MSU	2022
B22856	Black	MSU	2022
B22857	Black	MSU	2022
B22859	Black	MSU	2022
B22860	Black	MSU	2022
B22861	Black	MSU	2022
B22862	Black	MSU	2022
B22863	Black	MSU	2022
B22865	Black	MSU	2022
B22866	Black	MSU	2022
B22867	Black	MSU	2022
B22868	Black	MSU	2022
B22870	Black	MSU	2022
B22873	Black	MSU	2022
B22874	Black	MSU	2022
B22875	Black	MSU	2022
B22876	Black	MSU	2022
BC216	Black	MSU	2021/2022
BC269	Pink	MSU	2021/2022
USPT-WM-12	Pinto	Miklas, USDA	Historic/2021/2022
SR9-5	Small Red	Miklas, USDA	Historic/2021/2022
Merlin	Navy	Provita	Historic/2021/2022
Viper	Small Red	Provita	Historic/2021

Table 1.5 (cont'd)

Black Bear	Black	Provita	2021/2022
Bunsi	Navy	Tu, J.C., and W.D. Beversdorf. 1982.	Historic/2021/2022
Beryl	Great Northern	Syngenta	Historic/2021/2022
G122	Cranberry	Landrace	Historic/2021/2022
N11283	Navy	MSU	Historic/2021/2022
N14218	Navy	MSU	Historic
N14229	Navy	MSU	Historic
N15331	Navy	MSU	Historic
N15341	Navy	MSU	Historic
N16401	Navy	MSU	Historic
N16405	Navy	MSU	Historic
N17504	Navy	MSU	Historic
N17505	Navy	MSU	Historic/2021
N17506	Navy	MSU	Historic
N18102	Navy	MSU	Historic
N18103	Navy	MSU	Historic/2021
N18105	Navy	MSU	2021
N18109	Navy	MSU	Historic
N18117	Navy	MSU	Historic
N18122	Navy	MSU	Historic/2021
N18130	Navy	MSU	Historic/2021
N19223	Navy	MSU	2021

Table 1.5 (cont'd)

N19226	Navy	MSU	2021
N19239	Navy	MSU	Historic/2021
N19243	Navy	MSU	2021
N19246	Navy	MSU	2021/2022
N19248	Navy	MSU	Historic
N19252	Navy	MSU	2021
N19253	Navy	MSU	2021
N19269	Navy	MSU	2021
N19277	Navy	MSU	2021
N19284	Navy	MSU	2021
N19285	Navy	MSU	Historic/2021
N19290	Navy	MSU	2021
N20317	Navy	MSU	2021
N20335	Navy	MSU	2021
N20336	Navy	MSU	2021
N20341	Navy	MSU	2021
N20343	Navy	MSU	2021
N20346	Navy	MSU	2021
N20352	Navy	MSU	2021
N20376	Navy	MSU	2021
N20384	Navy	MSU	2021
N20388	Navy	MSU	2021/2022
N20391	Navy	MSU	2021
N20395	Navy	MSU	2021/2022

Table 1.5 (cont'd)

N20401	Navy	MSU	2021
N20404	Navy	MSU	2021/2022
N20405	Navy	MSU	2021
N21501	Navy	MSU	2021
N21502	Navy	MSU	2021
N21503	Navy	MSU	2021
N21504	Navy	MSU	2021
N21505	Navy	MSU	2021
N21506	Navy	MSU	2021
N21507	Navy	MSU	2021
N21508	Navy	MSU	2021
N21509	Navy	MSU	2021
N21510	Navy	MSU	2021
N21511	Navy	MSU	2021/2022
N21512	Navy	MSU	2021
N21513	Navy	MSU	2021
N21514	Navy	MSU	2021
N21515	Navy	MSU	2021
N21516	Navy	MSU	2021
N21517	Navy	MSU	2021
N21518	Navy	MSU	2021
N21519	Navy	MSU	2021
N21520	Navy	MSU	2021
N21521	Navy	MSU	2021

Table 1.5 (cont'd)

N21522	Navy	MSU	2021
N21523	Navy	MSU	2021
N21524	Navy	MSU	2021
N21525	Navy	MSU	2021
N21526	Navy	MSU	2021
N21527	Navy	MSU	2021
N21528	Navy	MSU	2021
N21529	Navy	MSU	2021
N21530	Navy	MSU	2021
N21531	Navy	MSU	2021
N21532	Navy	MSU	2021
N21533	Navy	MSU	2021
N21534	Navy	MSU	2021
N21535	Navy	MSU	2021
N22601	Navy	MSU	2022
N22602	Navy	MSU	2022
N22603	Navy	MSU	2022
N22605	Navy	MSU	2022
N22606	Navy	MSU	2022
N22607	Navy	MSU	2022
N22608	Navy	MSU	2022
N22609	Navy	MSU	2022
N22610	Navy	MSU	2022
N22612	Navy	MSU	2022

Table 1.5 (cont'd)

N22613	Navy	MSU	2022
N22614	Navy	MSU	2022
N22615	Navy	MSU	2022
N22616	Navy	MSU	2022
N22617	Navy	MSU	2022
N22618	Navy	MSU	2022
N22619	Navy	MSU	2022
N22620	Navy	MSU	2022
N22621	Navy	MSU	2022
N22622	Navy	MSU	2022
N22623	Navy	MSU	2022
N22624	Navy	MSU	2022
N22626	Navy	MSU	2022
N22627	Navy	MSU	2022
N22629	Navy	MSU	2022
N22630	Navy	MSU	2022
N22631	Navy	MSU	2022
N22632	Navy	MSU	2022
N22633	Navy	MSU	2022
N22634	Navy	MSU	2022
N22635	Navy	MSU	2022
N22636	Navy	MSU	2022
N22637	Navy	MSU	2022
N22638	Navy	MSU	2022

Table 1.5 (cont'd)

N22639	Navy	MSU	2022
R12844	Red	MSU	Historic/2021
R16503	Red	MSU	Historic
R17602	Red	MSU	2021
R17603	Red	MSU	Historic
R17604	Red	MSU	Historic/2021
R17605	Red	MSU	2021
R18402	Red	MSU	2021
R19502	Red	MSU	2021
R20604	Red	MSU	2021
R20612	Red	MSU	2021
R20614	Red	MSU	2021
R20624	Red	MSU	2021
R20625	Red	MSU	2021
R20627	Red	MSU	2021
R20629	Red	MSU	2021
R20632	Red	MSU	2021
R20633	Red	MSU	2021
R20635	Red	MSU	2021
R20636	Red	MSU	2021
R20637	Red	MSU	2021
R20639	Red	MSU	2021
R20642	Red	MSU	2021
R20652	Red	MSU	2021

Table 1.5 (cont'd)

R20653	Red	MSU	2021
R20659	Red	MSU	2021
R20667	Red	MSU	2021
R20669	Red	MSU	2021
R20683	Red	MSU	2021
R20684	Red	MSU	2021
R98026	Red	MSU	Historic/2021
S08418	Pink	MSU	2021
S18904	Pink	MSU	Historic/2021
S18907	Pink	MSU	2021
S19307	Pink	MSU	2021
S20405	Pink	MSU	2021
S20420	Pink	MSU	2021

Table 1.6: ANOVA table for white mold resistance in the combined analysis (2021-2022) of the field trial.

	Variance	Standard Error
Genotype	0.45*	0.10
Environment:Genotype	0.24*	0.10
Error	1.82*	0.08

Values marked with an asterisk (*) are significant at the p=0.05 level.

Table 1.7: ANOVA table for yield under white mold disease pressure in the combined analysis (2021-2022) of the field trial.

	Variance	Standard Error
Genotype	13.51*	3.22
Environment:Genotype	11.47*	2.83
Error	13.90*	0.73

Values marked with an asterisk (*) are significant at the p=0.05 level.

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CHAPTER TWO: SCREENING DRY BEAN BREEDING LINES FOR RESISTANCE TO COMMON MICHIGAN ROOT ROT PATHOGENS

ABSTRACT

Root rot is a major yield limiting disease of dry bean (*Phaseolus vulgaris*) production in the US and worldwide. Specifically, disease symptoms conferred by the soil borne fungal pathogens *Fusarium oxysporum* and *Rhizoctonia solani* cause up to 84% yield loss in susceptible dry bean cultivars through damage of root biomass, reduced vigor, and plant death. This study evaluated a diverse set of breeding lines and diversity panel lines from 2021 to 2022 for field resistance to root rots conferred by these pathogens. Secondary objectives were to establish correlations between field and greenhouse screens and non-destructive traits correlated to root rot resistance to *Fusarium oxysporum* for ease of phenotyping. All trials were successful in identifying significant variation for root rot resistance. Significant correlations were found between the field trial and the greenhouse trials (P=0.03, 0.71). Significant trait correlations were also identified between root rating and fresh (P=0.01, -0.67) and dry root (P=0.01, -0.64) and dry shoot (P=0.04, -0.43) weights in the greenhouse. Ultimately multiple lines are recommended as parents for future root rot resistance breeding efforts.

INTRODUCTION

Dry bean (*Phaseolus vulgaris* L.) is one of the most important grain legumes for human consumption worldwide, providing an important source of key dietary nutrients such as protein and fiber (Uebersax et al., 2023). Dry beans are a dietary staple in Latin America and sub-Saharan Africa (Leterme & Carmenza Muũoz, 2002; Paparu et al., 2018) and an important agricultural commodity in the US (primarily North Dakota, Minnesota, and Michigan), Latin America, and other regions. However, biotic stress via fungal disease infection is among the main constraints

that limit yield and increase annual management cost of dry bean production. Among the most yield limiting fungal borne diseases affecting dry bean production is root rot, which is conferred by a soilborne disease complex of multiple fungal and oomycete pathogens including members of *Fusarium sp.*, *Rhizoctonia sp.*, *Pythium sp.*, *Macrophomina sp.*, and *Alternaria sp*. (Bilgi et al., 2011; Harveson et al., 2005; Schwartz, 2011; Sendi et al., 2020; Singh & Schwartz, 2010). Root rots are widespread in dry bean production regions and cause significant yield losses in the US, Africa, and Central and South America (Abawi and Pastor Corrales, 1990) For example, root rot is responsible for losses estimated at 221.000 metric tons per year in sub-Saharan Africa (Paparu et al., 2018).

Dry beans have two primary gene pools resulting from two independent domestication events in the Andes and Middle-Americas (Gepts, and Debouck, 1991; Mensack et al., 2010). The Andean gene pool primarily consists of large-seeded beans from the kidney (dark red, light red, and white), yellow, and cranberry market classes. The Middle-American gene pool consists of small to medium seeded market classes from the navy, black, small red, pink, pinto and great northern market classes. Previous research has established susceptibility to root rot in both dry bean gene pools, but susceptibility to individual pathogens differs. Large-seeded dry bean cultivars from the Andean gene pool are generally more susceptible to *Fusarium* species while the small seeded types from the Middle-American gene pool are generally more susceptible to *Rhizoctonia* species, with Andean types being more susceptible to root rots overall (Conner et al., 2014; Schneider et al., 2001). Andean genotypes in particular, tend to have less robust root systems which is thought to be why they have lower levels of resistance to root rots (Cichy et al., 2007; Román-Avilés & Kelly, 2005; Schneider et al., 2001).

Michigan is the second largest dry bean producer in the United States, producing over 400 million pounds of dry edible beans per year and contributing to a farm gate value of over $139 million (USDA Crop Production Summary 2022). It also leads the nation in the black, small red, and navy bean market classes in terms of acreage (USDA Crop Production Summary 2022). Michigan's temperate climate, with warm humid summers, are highly conducive to fungal pathogens. Over the past few years Michigan growers have rated root rot as the second most important disease and it is considered the first most impactful disease in large seeded Andean bean varieties (MBC, 2022). In Michigan, the most common root rot pathogens belong to the *Fusarium* and *Rhizoctonia* species as well as multiple oomycete species and species diversity varies by region (Jacobs et al., 2019). Root rots conferred by these fungal pathogens cause up to 84% yield loss in susceptible dry bean cultivars through damage of root biomass, reduced vigor, and whole plant death (Jacobs et al., 2019).

Root rot disease symptoms vary depending on the pathogen(s) involved, but are primarily characterized by root lesions, root and foliar biomass loss, and reduced stand. *Fusarium* and *Rhizoctonia* infections of dry bean are both primarily characterized by water-soaked, dark brown to rust colored lesions on the root and death of lateral roots (Hall et al. 1991; Hagedorn et al. 1994). *Fusarium* root rot develops slowly in response to prolonged cool and wet environmental conditions, and generally leads to an overall vigor and yield decline. Entire plant death is rare and usually occurs later in the season in only the most severe infections. Conversely, *Rhizoctonia* root rot tends to affect plants early in the season primarily through reduced stand resulting from seed rot, seedling blight, and pre/post emergence damping off (Gossen et al., 2016). *Rhizoctonia* root rot infection is characterized by the formation of deep sunken lesions at the soil level (Conner et

al., 2014). Cool wet weather or flooding events can increase infection severity of both pathogens due to additional plant stress (Kumar and Kudada, 2018).

Managing root rot can be challenging due to the durability and extended viability of chlamydospores in soil and plant residue and broad host range (Katan, 2017). Current agronomic management practices include fungicidal seed and soil treatments, reduced irrigation, crop rotation, cover crops, seedbed preparation, and other agronomic practices are currently used to combat yield loss from root rots (Abawi and Pastor Corrales, 1990; Gossen et al., 2016; Harveson et al., 2005; Rubiales et al., 2015). However, they are often not sustainable or economically feasible and reduce productivity of dry bean fields. For example, control of root rots through reduced irrigation limits yield per acre. Fungicidal seed and soil treatments are expensive and have adverse effects on the environment. Furthermore, these practices do not entirely prevent root rot infection due to its virulence and ability for thick-walled spores, hyphae, or sclerotia to overwinter in the soil for multiple years (Schwartz, 2011). Biocontrols have emerged as a significant research objective for control of root rot pathogens, but there are currently no commercial biocontrols available that significantly reduce infection (Hassan Dar et al., 1997; Sendi et al., 2020). Therefore, the most sustainable, durable, and cost-effective method to ensure protection against infection would be to develop resistant dry bean cultivars, along with appropriate management practices.

Genetic resistance to root rots in dry bean is quantitatively inherited, primarily through root architecture traits such as root weight, root length and root mass (Haus et al., 2020; Kamfwa et al., 2013; Snapp et al., 2003; Wang et al., 2018) and physiological avoidance (Kamfwa et al., 2013; Mukankusi et al., 2011; Román-Avilés et al., 2004; Snapp et al., 2003). The quantitative inheritance of these traits has been demonstrated by the low narrow-sense heritability estimates (26%-44%) reported and many small effect quantitative trait loci (QTL) found for root rot (Román-

Avilés et al., 2011; Nakedde et al., 2016; Kamfwa et al., 2018; Wang et al., 2018; Zitnick-Anderson et al., 2020). Previous research has established the importance of high root biomass cultivars with high density of lateral roots, high basal root number, and many adventitious roots for root rot avoidance (Haus et al., 2020; Román-Avilés et al., 2004; Snapp et al., 2003). These traits have been identified as significant targets for improving resistance. Resistance to root rot is primarily found in the small-seeded Middle-American bean germplasm compared to the highly susceptible large-seeded beans of Andean origin and has served as the only source of resistance (Cichy et al., 2007; Mukankusi et al., 2011; Román-Avilés & Kelly, 2005; Schneider et al., 2001)

Although few studies to date have evaluated genetic resistance to *Fusarium oxysporum* or *Rhizoctonia solani*, multiple studies have established genomic regions associated with resistance to root rots conferred by other *Fusarium* sp. (Hagerty et al., 2015; Kamfwa et al., 2013; Nakedde et al., 2016; Román-Avilés & Kelly, 2005; Schneider et al., 2001; Wang et al., 2018; Zitnick-Anderson et al., 2020). These studies often identify many different QTL which highlights the highly quantitative inheritance method of resistance. QTL studies of resistance to *Fusarium sp.* in dry bean have identified genomic regions associated with root traits, plant immune/defense mechanisms, and other disease resistance genes (Hagerty et al., 2015; Nakedde et al., 2016; Wang et al., 2018; Zitnick-Anderson et al., 2020). One study to date has evaluated genetic resistance to *Rhizoctonia solani*- (Oladzad et al. 2019). In this study, researchers evaluated the Andean and Middle American diversity panels and observed multiple QTL associated with gene models encoding proteins like known disease resistance genes. Other *Rhizoctonia solani* resistance studies have focused on screening and ranking breeding lines and establishing trait correlations (Adesemoye et al., 2018; Conner et al., 2014; Peña et al., 2013).

Progress to introgress root rot resistance has been hindered by the difficulty of pyramiding resistance genes given the quantitative nature of inheritance of root rot, inconsistent screening methods, and screening dependence on the presence of the pathogen under suitable environmental conditions (Hagerty et al., 2015; Nakedde et al., 2016; Wang et al., 2018). Phenotyping for root rot disease in the field often requires laborious and destructive “shovelomics" techniques (Burridge et al., 2016; Trachsel et al., 2011) that involve digging up the root system of individual plants to evaluate root rot disease severity or manually counting stands multiple times per season to evaluate post-emergence damping off. There have been multiple phenotyping methods proposed for the greenhouse and field evaluation of *Fusarium* root rots including the liquid inoculum method (Schneider & Kelly, 2000), inoculum layer method (Chaudhary et al., 2006), and nutrient culture (Boomstra et al., 1977). Inoculated grain planted alongside the crop seed or natural infection are common methods for field evaluation (Haus et al., 2020; Pandey et al., 2020; Wang et al., 2018). However, the presence of multiple resistance mechanisms through both physiological resistance and architectural avoidance complicates screening. Greenhouse trials are preferred by researchers because they provide a controlled environment, have higher heritability, ease of screening, and less environmental dependance, but they do not screen for some avoidance mechanisms that can only be observed in the field. Conversely, field screens allow for measurement of both architectural traits and physiological resistance, but are often hampered by low heritability, high coefficient of variation (CV), high error variances, and natural pathogen presence (Guzman, 2016.; Hagerty et al., 2015; Nakedde et al., 2016; Román-Avilés et al., 2004; Román-Avilés & Kelly, 2005). Furthermore, these destructive phenotyping methods require the whole plant to be dug up with a shovel in the field or removed from the pot in the greenhouse to be evaluated for root rot symptoms, which is laborious and prevents additional measurements of yield and other agronomic traits

throughout the season. Establishments of aboveground traits related to root rot disease severity would enable high-throughput methods of phenotyping such as UAS imaging to assist breeders in screening for root rot resistant varieties (Guo et al., 2021; Lu et al., 2019; Manganiello et al., 2021; Marzougui et al., 2019).

Therefore, the primary objective of this study was to screen advanced breeding lines and a subset of an Andean diversity panel for *Fusarium oxysporum* and *Rhizoctonia solani* under field and greenhouse conditions. Specific objectives were to ii) evaluate a set of panels for *F. oxysporum* and *R. solani resistance* across multiple years, ii) compare a subset of the *Fusarium* lines grown in the field and in the greenhouse, and iii) compare the rankings between artificial inoculation and natural infestation in the field, and iv) determine agronomic traits correlated to *Fusarium oxysporum* root rot resistance that could provide an alternative phenotyping method.

MATERIALS AND METHODS

Plant Material

A set of three panels were used to evaluate *Fusarium* and *Rhizoctonia* resistance during the 2021 and 2022 field season (**Table 2.1, Table 2.2**). Specifically, two panels were used to evaluate *F. oxysporum* root rot resistance and one for *R. solani*. The *F. oxysporum* panels included a set 2021 and 2022 advanced kidney bean breeding lines from the Michigan State University dry bean breeding program (KDB) and a subset of 38 lines from the Andean Diversity Panel (ADP) (Cichy et al., 2015). The KDB trial consisted of 53 advanced breeding lines (AYT) from the large-seeded Andean gene pool (kidney and yellow market classes) of the MSU breeding program and 13 commercial varieties. 29 lines were evaluated in 2021 and 27 lines were evaluated in 2022, with 20 AYT lines conserved in both years. The motivation for this approach was to eliminate inferior breeding lines discarded from the MSU Advanced Yield Trial (AYT) after year one and replace

them with newer breeding lines that had performed well in the Preliminary Yield Trials (PYTs). The commercial varieties Coho, Clouseau, Denali, Red Cedar, Red Hawk, and Snowdon were added as performance checks. Clouseau was chosen as it has been widely grown in Michigan, while the others represent recently released MSU developed light red, dark red, or white kidney varieties that have shown superior yield potential despite ambient root rot disease pressure. All lines in the ADP trial were evaluated in both years. The check lines used in the ADP trial included the resistant line VAX 3 (Bilgi et al., 2011; Guzman, 2016), Cabernet (susceptible), Dynasty (moderately resistant), and Talon (moderately resistant).

The third panel (MAB) consisted of 65 AYT breeding lines from the small-seeded Middle American gene pool (great northern, pinto, small red, pink, black, and navy market classes) was used to evaluate *R. solani* root rot resistance in the field during the 2021 and 2022 growing seasons. The 2021 set consisted of 24 AYT lines and 19 commercial lines while the 2022 set consisted of 31 AYT lines and 22 commercial lines. Across the panel 16 breeding lines and 15 commercial lines were conserved in both years. There currently aren't any established performance checks for *Rhizoctonia solani* resistance in dry bean. Commercial lines used for comparison are listed in **Table 2.**

Field Experiment

Three field trials (ADP, KDB, and MAB) field were planted in a disease nursery in East Lansing, MI using a randomized-complete block design (RCBD) with four replicates. Entries were planted in four-row plots, 160 seeds per replicate (40 per row), 10 ft in length with 30 in row spacing, where two rows contained non inoculated plants, and two rows contained plants inoculated with *F. oxysporum* isolate F_14-38 or *R. solani* AG2-2 isolate Rs_14-17 colonized grain. Isolates used for the inoculum were collected from dry bean plants in Michigan. Inoculum

consisted of millet (*F. oxysporum*) or barley (*R. solani*) kernels colonized with mycelium at a concentration of 4.23 x 10^6 per gram and air dried in a drying oven. In the *F. oxysporum* trials (ADP and KDB) the colonized millet seed was planted in the furrow with the dry bean seed at a rate of 34.45 ml/linear meter in 2021 and increased to 42.65 ml/linear meter in 2022 following the protocol described in (Haus et al., 2020; Jacobs et al., 2018) In the *R. solani* (AM) trial the colonized barley seed was planted in the furrow with the dry bean seed at a rate of 4.27 ml/linear meter in 2021 and 3.28 ml/linear meter in 2022.

The KDB trial was also planted at Montcalm Research Farm in the 2021 and 2022 growing season near Entrican in Montcalm County, Michigan which is a location previously established to have substantial root rot disease pressure. Previous literature on root rot disease in Michigan and visual observation has established *F. solani fsp. phaseoli* present in this site which makes it ideal to compare natural infection conditions with artificial inoculation (Jacobs et al., 2019; Román-Avilés et al., 2004). The experimental design was an alpha-lattice design with three replications using four row plots 6.1 m in length with 50 cm row spacing.

Phenotypic Evaluation

Plants were collected and rated for disease severity in the ADP and KDB *F. oxysporum* trials and the KDB natural infection trial. One month after germination (V3-V4 stage), five plants from each line, replicate, and treatment were uprooted with a shovel, washed to remove soil, and evaluated for root rot disease severity using the 1 to 7 scale, developed by Schneider and Kelly where 1 indicates no disease and 7 indicates a non-functional, completely rotted root system (Schneider & Kelly, 2000). **Figure 1.1** shows an image of dry bean plants used to calibrate disease scores corresponding to the 1-7 rating scale. In 2022, the five plants from each replication were bulked and weighed (g) after root rot disease severity was recorded. Multiple vigor and stand count

measurements were taken throughout the growing season both years. Vigor was measured by visually observing overall plot wise canopy biomass and closure on a scale of 1-9 following the rating system in Van Schoonhoven, 1987.

In the MAB *R. solani* trial, stand count per row for the inoculated and non-inoculated treatments was collected at multiple time points during the growing season. Post-emergence damping off was defined as the difference in stand count between the first and last measurement of the season.

In the natural infection trial of the KDB lines, root rot disease severity was evaluated as outlined previously at 6-8 weeks after planting. Standard agronomic traits including plant height, lodging score, days to flower, and maturity were also collected in the field. Plots were trimmed to 4.9m prior to harvest and the center two rows were harvested with a Wintersteiger Classic plot combine (Wintersteiger AG, Austria). Yield data was obtained utilizing a Harvestmaster H2 Classic Graingage (Juniper Systems, Logan, UT) to record plot weight and moisture. Prior to data analysis, seed yield was standardized to 18% moisture.

Greenhouse Experiment

A trial run of 5 genotypes was used to identify optimal inoculum concentration and harvest date to elucidate differences in resistance to *Fusarium oxysporum* in the greenhouse and compare rankings for similar genotypes to the field trials. Inoculated grain was prepared using the same procedures as the field trial. The containers used were 354-mL coffee cups with three holes on the bottom for drainage. Different quantities of inoculum (2, 3.5, and 5 grams) were tested to determine the correct ratio of inoculum to best separate resistance among lines in the main trial. Inoculum was mixed thoroughly with vermiculite medium in a plastic bag. Bean seeds were placed on the top and covered with another layer of vermiculite. Non-inoculated replicates followed the

same methods, minus the inoculum step. Plants were arranged in a RCBD with 3 replicates. Two harvest dates (3 and 4 weeks) were also tested to determine the best time to evaluate differences in root symptoms.

A subset of the 11 most resistant and susceptible lines (n=22) and the resistant check VAX3 obtained from the 2021 KDB and ADP field trials were selected to evaluate root rot disease severity under greenhouse conditions. Based on results of the test trial, a 3 week harvest date and an inoculum concentration of 2 grams per cup was chosen. Production of inoculum and planting methods followed the same procedure as in the test greenhouse trial. For each genotype twelve seeds were planted, one in each coffee cup, using a randomized complete block experimental design with three inoculated and three non-inoculated replicates of 6 plants each. All plants were uprooted, washed with water, and evaluated for root rot disease severity using the 1-7 root rot disease severity scale mentioned previously. Fresh and dry shoot and root weights (g) for each plant were also taken.

Data Analysis

The statistical analysis for all experiments were conducted in the ASReml-R package in the R programming language (Butler et al., 2023). The normality of residuals was evaluated using quantile plots and residual plots and outliers were removed as necessary. For all experiments a linear mixed model was fit and LSmeans for root rot disease severity (ADP, KDB, GH, and KDB Natural) or post-emergence damping off (MAB) were calculated using the predict function of the ASReml-R package (Butler et al., 2023) utilizing the inoculated treatment only as follows:

ADP, KDB, and MAB field trials

$$\mathbf{Y_{ijk} = \mu + Line_i + Env_k + Rep(Env)_{jk} + (Line\ x\ Env)_{ik} + e_{ijk}} \qquad \text{Eq. 1}$$

Where Y_{ijk} is the observed phenotype. μ is the overall mean, $Line_i$ is the fixed effect of the *i*-th line, Env_k is the fixed effect of the *k*th environment, $Rep(Env)_{jk}$ is the random effect of the interaction between the the *j*-th rep within the *k*-th environment, $(Line \times Env)_{ik}$ is the fixed effect of interaction between the *i*-th line and *k*-th environment, and e_{ijk} is the random residual term.

ADP and KDB greenhouse trial

$$\mathbf{Y_{ij} = \mu + Line_i + Rep_j + e_{ij}} \quad \text{Eq. 2}$$

Where Y_{ij} is the observed phenotype. μ is the overall mean, $Line_i$ is the fixed effect of the *i*-th line, Rep_j is the random effect of the *j*th rep, and e_{ij} is the random residual term.

Natural Infection Trial (KDB)

$$\mathbf{Y_{ijkl} = \mu + Line_i + Env_j + iBlock(Rep)_{kl} + Rep(Env)_{jl} + (Line \times Env)_{ij} + e_{ijkl}} \quad \text{Eq. 3}$$

Where Y_{ijkl} is the observed phenotype (root rot disease severity score). μ is the overall mean, $Line_i$ is the fixed effect of the *i*-th line, Env_j is the fixed effect of the *j*th environment, $iBlock(Rep)_{kl}$ is the random effect of the interaction between the the *k*-th incomplete block within the *l*-th rep, $Rep(Env)_{jl}$ is the random effect of the interaction between the the *l*-th rep within the *j*-th environment, $(Line \times Env)_{ij}$ is the fixed effect of interaction between the *i*-th line and *j*-th environment, and e_{ijkl} is the random residual term.

To estimate broad sense heritability (H^2) for on an entry means basis, variance components were extracted fitting the previous equations with all terms random. Heritability was estimated as follows:

$$H^2 = \frac{\sigma_G^2}{\sigma_G^2 + \frac{\sigma_{GE}^2}{n} + \frac{\sigma_e^2}{rn}}$$ Eq. 4

Where σ^2_G ,σ^2_{GxE} ,and , σ^2_e are the genotype, genotype by environment interaction, and error variances, n is the number of environments and r is the number of reps. Pairwise comparisons between lines were calculated using Fisher's LSD using the assigned LSD value resulting from the allDifferences.data.frame function in the ASRemlPlus package (Brien et al. 2023). The effect of inoculum was validated using a student's t-test of root rot disease severity scores or post-emergence damping off between the inoculated and non-inoculated treatments. Population coefficient of variation (CV) was calculated as ((σ/μ) * 100) = CV%.

Correlations

Correlations between trials were evaluated to compare phenotyping in the greenhouse vs field as well as artificial vs natural disease pressure using the LSmeans for lines present in both trials. Additionally, correlations between root rot rating (or post emergence damping off) and various agronomic traits were calculated using trait LSmeans. Correlations between trials were performed using the cor.test function in R and correlations between traits were visualized using the corrplot function in the R package corrplot (Wei et al., 2021).

RESULTS

In this study we evaluated three diverse dry bean panels (ADP, KDB, MAB) in the field for variance in resistance to two prevalent Michigan root rot pathogens. Additionally, a subset of the ADP and KDB trials were evaluated in the greenhouse and the entire KDB trial was also evaluated in natural root rot infection conditions. Significant differences were found between inoculated and non-inoculated plots using a student's t-test which indicated significant differences

in mean value between treatments in all trials and years tested **(Table 2.3)**. Overall, genetic variation was low with the lowest being for the *F. oxysporum* trials compared to the *R. solani* trial. This led to a lower heritability (0.12-0.29) in the *F. oxysporum* trials (KDB, ADP, GH), low to moderate in the KDB natural infection trials (0.11-0.43) and moderate in the MAB *R. solan*i trial (0.49-0.57) with the greenhouse having the lowest heritability overall (**Table 2.4**). Significant genotypic variation was found for all *F. oxysporum* trials across all years except the 2022 ADP trial and Greenhouse trial (Table 2.4). There was no significant genotype by year effect across any of the field trials. Coefficient of variation (CV) was low to moderate for all *F. oxysporum* trials (31.04-49.26%), low in the KDB natural infection trial (23.94-35.32%), and high for the MAB trials (65.11-71.06%) (**Table 2.4).**

Fusarium Oxysporum Field and Greenhouse Screens

After outlier removal and model fitting, LSmeans in the field for the ADP trial ranged from 1.8-3.9 in 2021, 2.3-4.6 in 2022, and 2.05-4 in the combined analysis **(Figure 2.2a)**. The five most resistant lines in the combined analysis were ADP99, ADP444, ADP481, Dynasty, and ADP462 with scores ranging from 2.05-2.7 (**Table 2.5, Figure 2.3**). While the checks, Dynasty, Talon, Cabernet, and VAX3 had disease severity scores of 2.65, 2.87, 3.3, and 3.47 respectively. In 2021, the five most resistant lines were ADP99, ADP444, ADP462, ADP43, and ADP612 with scores ranging from 1.8-2.3 (**Table 2.6, Figure 2.4**). While the checks, Dynasty, Talon, Cabernet, and VAX3 had disease severity scores of 2.6, 2.7, 3.3, and 3.7 respectively. In 2022, the five most resistant lines were ADP99, ADP111, ADP481, ADP15, and ADP4 with scores ranging from 2.3-2.65 (**Table 2.7, Figure 2.5**). While the checks, Dynasty, Talon, Cabernet, and VAX3 had disease severity scores of 2.7, 3.05, 3.3, and 3.25, respectively.

The LSmeans in the field for the KDB trial ranged from 2.2-3.3 in 2021, 2.1-4.3 in 2022, and 2.4-4 in the combined analysis (**Figure 2.2b**). The five most resistant lines in the combined analysis were K20730, Y19817, Beluga, K19832, and Clouseau with scores ranging from 2.39-2.73 (**Table 2.8, Figure 2.6**). While the checks, Clouseau, Denali, Snowdon, Coho, and Red Cedar had disease severity scores of 2.7, 2.73, 2.84, 2.75, and 3.35, respectively. In 2021, the five most resistant lines were K19610, K20712, Y19817, K16911, and K18907 with scores ranging from 2.3-2.5 (**Table 2.9, Figure 2.7**). While the checks, Clouseau, Denali, Snowdon, Coho, and Red Cedar had disease severity scores of 2.73, 2.85, 2.8, 2.75, and 3.35, respectively. In 2022, the five most resistant lines were K20730, K19832, Y19808, Beluga, and Y19817 with scores ranging from 2.09-2.48 (**Table 2.10, Figure 2.8**). While the checks, Clouseau, Denali, Snowdon, Coho, and Red Cedar had disease severity scores of 2.75, 2.6, 2.89, 3.25, and 3.5, respectively. The LSmeans for the greenhouse trial ranged from 4-6 (**Figure 2.2c**). The five most resistant lines in the greenhouse were ADP481, K20730, VAX3, ADP462, and K20712 with scores ranging from 4-4.78 (**Table 2.11, Figure 2.9**). VAX 3 had a disease score of 4.5 and Dynasty had a disease score of 6.

Natural Infection

The LSmeans in the field for the KDB natural infection trial ranged from 3.45-5.41 in 2021, 2.47-5.58 in 2022, and 3.17-5.06 in the combined analysis (**Figure 2.2b**). The five most resistant lines in the combined analysis were K19832, K19817, K20745, K20717, and K20730 with scores ranging from 3.17-3.57 (**Table 2.12, Figure 2.10**). While the checks, Clouseau, Denali, Snowdon, Coho, and Red Cedar had disease severity scores of 4.11, 4.15, 4.26, 4.23, and 5.06 respectively. In 2021, the five most resistant lines were K19832, K20730, K20743, K21904, K16136 with scores ranging from 3.45-3.9 (**Table 2.13, Figure 2.11**). While the checks, Clouseau, Denali, Snowdon,

Coho, and Red Cedar had disease severity scores of 4.44, 4.17, 4.41, 4.17, and 4.53, respectively. In 2022, the five most resistant lines were K19817, K19832, K20745, K20717, and K20730 with scores ranging from 2.47-3.44 (**Table 2.14, Figure 2.12**). While the checks, Clouseau, Denali, Snowdon, Coho, and Red Cedar had disease severity scores of 3.8, 4.14, 4.12, 4.28, and 5.58, respectively.

Rhizoctonia solani Screen

The LSmeans in the field for the MAB trial ranged from 2.00-13.50 in 2021, 1.25-16.25 in 2022, and 3.31-12.25 in the combined analysis (**Figure 2.2d**). The five most resistant lines in the combined analysis were B19344, N20395, N19246, Spectre, and N20404 with values ranging from 3.31-3.69 (**Table 2.15, Figure 2.13**). In 2021, the five most resistant lines were N19226, N20404, G19611, P19103, and N20395 with values ranging from 2-3.87 (**Table 2.16, Figure 2.14**). In 2022, the five most resistant lines were Spectre, G21811, Merlin, N19246, and B19344 with values ranging from 1.25-2.5 (**Table 2.17, Figure 2.15**).

Trial and Trait Correlations

Most correlations between *Fusarium* root rot disease scores among genotypes in the field, greenhouse, and natural infection trials were found to be statistically non-significant except for between the 2022 Kidney bean trial and the greenhouse trial with a significant correlation (P =0.03) of 0.71(**Table 2.18**). Additional measurements were conducted in all trials to establish possible traits correlated to root rot disease severity. In the KDB trials, no significant correlations were found between root rot rating and other traits that were measured (vigor, stand count, bulk weights), but significant correlations were identified among vigor, stand count, and bulk weight measurements (**Figure 2.16, Table 2.19)**. In the KDB trials, significant correlations were found between root rot rating and the first and last vigor measurements in 2021. Significant correlations

were also identified between other agronomic traits that were measured (vigor, stand count, bulk weights) **Figure 2.17, Table 2.20.** In the greenhouse trial significant correlations were found between root rot rating and fresh/dry root and dry shoot measurements (**Figure 2.18, Table 2.21**). Significant correlations were also found among fresh/dry root and shoot measurements. In the KDB natural infection trial, significant correlations were observed between root rot rating and yield and maturity date (**Figure 2.19, Table 2.22**).

DISCUSSION

This diverse study aimed to screen MSU Andean breeding lines and diversity panel lines for field and greenhouse-based resistance to *Fusarium oxysporum* and screen MSU Middle-American breeding lines for field resistance to *Rhizoctonia solani*, two pathogens that are particularly detrimental to dry bean production in Michigan. The levels of disease pressure in all experiments were sufficient to examine differences in plant response in inoculated conditions under root rot disease pressure, although low genetic variation limits analysis. Root rot resistance in dry bean is notoriously difficult to screen for due to many environmental factors that contribute to severity of the infection, and the ability of the plant to overcome the pathogen through avoidance or physiological resistance conferred by numerous small effect genetic loci. Although high standard error (SE) limits distinguishing between two similarly resistant genotypes in all trials tested, conclusions can be drawn in distinguishing between the most and least resistant lines, which is ultimately the most important objective when making selections in a plant breeding program.

Fusarium oxysporum Study

In the ADP trial the commercial checks Dynasty, Cabernet, and Talon behaved as expected based on results from previous literature (Oladzad et al., 2019; Zitnick-Anderson et al., 2020), but VAX3 consistently behaved as susceptible. In the greenhouse, VAX3 behaved as resistant as

expected, but Dynasty was the most susceptible line, supporting the observation that there was low correlation between ratings in the field vs greenhouse studies. In the ADP trial multiple lines stood out as possible sources of root rot resistance including ADP444, ADP481, ADP462, and ADP391. In the KDB inoculated and natural infection trials, multiple MSU breeding lines consistently outperformed the commercial lines. K19832, K20717, and K20730 stood out in particular as parents for future root rot resistance breeding efforts as they were top resistant lines in the KDB inoculated and natural infection trials. These lines have been noted previously as vigorous, robust plants with high yield. Their high root rot resistance may contribute to higher stands, vigor, and observed overall yield under ambient disease pressure in Michigan.

Root rot rating was highest overall in the greenhouse, which was particularly interesting because during the evaluation we noted multiple "escapes" or inoculated plants that avoided disease infection. This could have also contributed to the lower heritability. In the field, the natural infection condition trial had the highest root rot rating. This is likely due to the effect of the root rot species complex in Montcalm County where these lines were tested, versus the artificial inoculation with one species in the KDB trial. Genetic variation in root rot disease score within trials was relatively small with the ADP trial having the largest variation. Identifying and integrating sources of unadapted resistance such as what is found in the ADP, may be a valid approach for integrating higher levels of resistance in breeding lines.

Rhizoctonia solani Study

The MAB trial was the first screen of commercial and MSU breeding lines for resistance to post-emergence damping off due to *Rhizoctonia solani* infection to date. In contrast to the *Fusarium oxysporum* trials these trials had a higher heritability, likely alluding to the objective phenotyping measurement used (damping off) vs the subjective 1-7 scale or the nature of genetic

resistance to the specific species. Significant differences were observed between genotypes. Many MSU breeding lines were highly resistant to post-emergence damping off including B19344, N20395, N19246, N20404, and N19246. B19344 stands out particularly as a promising line to use as a parent in future breeding efforts as it was the most resistant line identified in the combined analysis. Like the resistant lines present in the KDB inoculated and natural trials, this line has been previously identified as high yielding, which may be partially due to its ability to overcome ambient root rot in Michigan. The commercial small red line Viper was consistently the most susceptible line to post-emergence damping off.

Field vs Greenhouse Correlations for Fusarium Root Rot

Screening for root rots in the field is a challenge due to the potential of other root rot pathogens to confound the study. Furthermore, field trials require large plots of land and in many cases pivot irrigation for inducing root rot. Therefore, developing a greenhouse assay would improve the challenges with field trials and improve the throughput of phenotyping for this trait. The only significant correlation identified was between the KD2022 and GH (0.71) (**Table 7**). However, there was an almost significant correlation of 0.65 between the combined KDB trial and the greenhouse trial. Increasing inoculum and environmental conditions during the 2022 season could have contributed to this higher correlation. Previous studies identified varying significance between field and greenhouse screens depending on which isolate, and inoculation method was used (Bilgi et al., 2008; Chaudhary et al., 2006; Mukankusi et al., 2010; Nicoli et al., 2012; Schneider & Kelly, 2000) with significant correlations of 0.57-0.92 being found with spore suspension greenhouse methods and natural infection field conditions. Greenhouse screening would be ideal since it takes less time and resources and more lines could be phenotyped in a season, but it is ultimately important that the screening method mimics field conditions in order to

make accurate selections. Further exploration to establish correlations between greenhouse and field screening utilizing different methods including the inoculated sorghum seed method is warranted.

When comparing results from the greenhouse and field *Fusarium oxysporum* trials, it is significant to note that although correlations between ratings in the greenhouse and field were not significant, two genotypes (ADP481 and K20730) were identified among the top five most resistant lines in both screening methods. K20730 was also a top five resistant line in the natural infection trial. It is possible that highly resistant lines are identified as resistant no matter the inoculation method used, but further research is needed to establish screening methods with better correlation.

Evaluating secondary traits associated with Fusarium oxysporum root rot

To score for Fusarium root rot, roots need to be harvested, washed, and scored. This process takes time and is labor intensive. Identifying secondary traits associated with root rot would allow for indirect evaluation of this trait in a non-destructive manner. Several traits were collected for this purpose. Root rot rating was found to be significantly negatively correlated to fresh and dry root weight in the greenhouse, supporting the hypothesis that this disease has significant negative impacts on the plant root biomass (Haus et al., 2020; Nakedde et al., 2016; Román-Avilés et al., 2004; Wang et al., 2018). This is contrary to a previous study by Bilgi et al. 2010 where there was no correlation identified between *F. solani* f. sp. *phaseoli* root rot rating and root weight, root-shoot ratio, or yield using the spore suspension method.

This observation also supports the finding in the natural infection trial where yield was highly negatively correlated to root rot disease score. In the natural infection trial, standard agronomic traits were measured. Both yield and maturity date were identified as significantly

negatively correlated to root rot disease score with correlations of -0.62 and -0.43, respectively. This was encouraging as it suggests that routinely selecting for higher yield and reasonable maturity under natural root rot pressure in breeding trials can be effective at eliminating the most susceptible breeding lines and advancing more tolerant ones. However, there were no significant correlations identified between root rot disease score and stand count or vigor measurements for the ADP and KDB field trials indicating that these may not be reliable measurements for non-destructively measuring root rot resistance. Another method for measuring root rot disease severity that would be beneficial to investigate is UAS based measurement of aboveground biomass. This would be a similar measurement as the ground-truth vigor that was taken in this study, but would be a uniform, high-throughput, unbiased plot wise aerial measurement that would eliminate any scorer error. There is previous research supporting this method for measuring disease traits indicating that it may be beneficial for accurately and non-destructively measuring root rot resistance score (Guo et al., 2021; Marzougui et al., 2019; Moreira et al., 2020).

Limitations

Effective screening for quantitatively inherited traits is challenging, and root rot disease traits are no exception. Field based root rot screens are preferable to the breeder because they most accurately reflect agronomic conditions, but they are labor and land intensive and confounded by natural pathogen presence, as well as large environmental and error variances. Conversely, greenhouse evaluations provide a controlled environment, cost, and labor effective screening alternative but may not accurately predict resistance in field conditions, which can lead to conflicting results between screening methods.

Similar to multiple previous root rot disease studies (Guzman, 2016; Hagerty et al., 2015; Nakedde et al., 2016; Román-Avilés et al., 2004; Román-Avilés & Kelly, 2005), the current study

was also hampered by large environmental and error variances, large standard error, and low genetic effect, leading to a low overall heritability and difficulty distinguishing between resistant genotypes for root rot disease rating (*Fusarium oxysporum* trials) and post emergence damping off (*Rhizoctonia solani* trials). This is primarily due to low innate heritability and the complex inheritance mechanism of the trait which prevents the separation of genetic and environmental effects and low innate resistance in breeding populations. Our greenhouse trial was also limited by large error variance, low genetic effect, and high CVs. The greenhouse screening method we used was chosen because it was previously shown to reduce environmental variation, decrease CV, and accurately mimic field conditions compared to other inoculation methods (Haus et al., 2020; Nakedde et al., 2016; Sendi et al., 2020; Wang et al., 2018). A consensus has not formed for the best inoculation method for root rot disease studies and various studies utilize different greenhouse screening methods which provide varying results among studies. As stated before, unbiased measurements such as UAS may improve this currently unavoidable aspect of root rot resistance studies as well as identifying the best universal greenhouse and field measurement systems.

VAX 3, a line previously established to have moderate resistance to *Fusarium brasiliense* and *Fusarium solani* in the greenhouse, behaved moderately resistant in the greenhouse and susceptible in the field, which raises the question of its reliability as a resistant check for *Fusarium oxysporum* screening in the field. An external factor that could have contributed to variable results in the field is the presence of untested natural pathogens. Natural pathogen presence is virtually unavoidable in the field, especially in a disease nursery, and could have contributed to the observed results. Previous studies have identified *Fusarium, Rhizoctonia,* and oomycete species as endemic to Michigan growing regions (Jacobs et al., 2019). Another factor that complicates analysis is the presence of avoidance mechanisms and variable environmental conditions in the field that could

contribute to resistance or susceptibility that would not be identified under controlled greenhouse conditions. Further research is needed to confirm the validity of this line as a resistant check to screen for *Fusarium* root rot resistance in the field. In the interim, a better approach would be to use the most resistant lines such as Dynasty as local disease resistant checks for future field studies in Michigan.

CONCLUSION

In the present study, 98 diverse dry bean lines from the Andean gene pool were screened for *Fusarium oxysporum* resistance in the field and (a subset) in the greenhouse and 64 lines from the Middle-American gene pool were screened for *Rhizoctonia solani* resistance in the field. The primary objective of this study was successful. Variance in root rot disease score/damping-off for a diverse set of dry bean lines was identified and the most resistant lines are recommended for future breeding efforts. All trials were successful in identifying significant variation for root rot resistance. Significant correlations were found between the field trial and the greenhouse trials (P=0.03, 0.71). Significant trait correlations were also identified between root rating and fresh (P=0.01, -0.67) and dry root (P=0.01, -0.64) and dry shoot (P=0.04, -0.43) weights in the greenhouse. Ultimately the lines ADP444, ADP481, ADP462, ADP391, K19832, K20717, K20730, B19344, N20395, N19246, N20404, and N19226 are recommended as parents for future root rot resistance breeding efforts. Similar to previous root rot resistance studies, high standard error (SE), error and environmental variance, and low heritability limited interpretation of the results. Ultimately, conclusions can be drawn in distinguishing between the most and least resistant lines, which is the most important objective when making selections in a plant breeding program.

Moving forward, objective high-throughput methods such as UAS based phenomics may provide an opportunity for accurate, unbiased, and reproducible screening of root rot resistance if

aboveground symptoms can be accurately measured and correlated to root rot rating as was suggested by the correlation between root and shoot biomass in the greenhouse trial. A standardized inoculation method and continued establishment of consistent resistant/susceptible checks under field conditions will also aid future studies. Ultimately, while the current studies presented here will be useful in making selections between the most resistant and most susceptible individuals, further optimization of screening methods is warranted for this highly complex trait.

TABLES AND FIGURES

Table 2.1: Population description, isolate information, sample number, years tested, and measurements collected for all populations evaluated for root rot resistance in this study.

Population Description	Isolate	Sample Number	Years	Trait Measurements
Andean Diversity Panel (ADP)	*F. oxysporum*	38	2021/2022	Root Rot Disease Score Vigor Stand Count Bulk Fresh Weight (2022)
Kidney Breeding Lines (KDB)	*F. oxysporum*	66	2021/2022	Root Rot Disease Score Vigor Stand Count Bulk Fresh Weight (2022)
Greenhouse trial (GH)	*F. oxysporum*	23	2022	Root Rot Disease Score Fresh Weight Dry Weight Stand Count
Natural Infection	*F. oxysporum*	42	2021/2022	Root Rot Disease Score Flowering Date Maturity Date Yield (CWT Acre) Lodging Score Bulk Fresh Weight (2022)
Middle American Breeding Lines (MAB)	*R. solani*	65	2021/2022	Stand Count Vigor

Table 2.2: List of lines evaluated in this study including the trials present, market class information, origin, and years tested.

Line	Trial(s)	Market Class / Seed Coat	Origin	Year(s)
ADP001	ADP	Red Mottled	ADP	2021/2022
ADP002	ADP	Cranberry	ADP	2021/2022
ADP004	ADP	Small Red	ADP	2021/2022
ADP015	ADP	Dark Red Kidney	ADP	2021/2022
ADP021	ADP	Small Red	ADP	2021/2022
ADP042	ADP	Purple	ADP	2021/2022
ADP043	ADP/GH	Yellow	ADP	2021/2022
ADP081	ADP	Black	ADP	2021/2022
ADP088	ADP	Light Red Kidney	ADP	2021/2022
ADP091	ADP	Cranberry	ADP	2021/2022
ADP094	ADP	Yellow	ADP	2021/2022
ADP099	ADP/GH	Dark Red Kidney	ADP	2021/2022
ADP111	ADP	Pink Cranberry	ADP	2021/2022
ADP112	ADP	Dark Red Kidney	ADP	2021/2022
ADP186	ADP/GH	Red Mottled	ADP	2021/2022
ADP214	ADP	Yellow	ADP	2021/2022
ADP391	ADP/GH	Red Mottled	ADP	2021/2022
ADP392	ADP	Red Mottled	ADP	2021/2022
ADP429	ADP	Yellow	ADP	2021/2022
ADP444	ADP/GH	Yellow	ADP	2021/2022
ADP462	ADP/GH	Yellow	ADP	2021/2022
ADP474	ADP/GH	Cranberry	ADP	2021/2022

Table 2.2 (cont'd)

ADP481	ADP/GH	Light Red Kidney	ADP	2021/2022
ADP511	ADP	Dark Red Kidney	ADP	2021/2022
ADP513	ADP	Yellow	ADP	2021/2022
ADP514	ADP	Light Red Kidney	ADP	2021/2022
ADP519	ADP/GH	White Kidney	ADP	2021/2022
ADP602	ADP	Pink Mottled	ADP	2021/2022
ADP612	ADP/GH	Dark Red Kidney	ADP	2021/2022
ADP621	ADP	Purple Speckled	ADP	2021/2022
ADP626	ADP	Purple Speckled	ADP	2021/2022
ADP640	ADP	Manteca	ADP	2021/2022
ADP683	ADP	Yellow	ADP	2021/2022
ADP684	ADP	Dark Red Kidney	ADP	2021/2022
Cabernet	ADP	Dark Red Kidney	Seminis	2021/2022
Dynasty	ADP/GH	Dark Red Kidney	Guelph	2021/2022
Talon	ADP	Dark Red Kidney	NDSU	2021/2022
VAX3	ADP/GH	Small Red	U of ID	2021/2022
Beluga	KDB/Natural (2021)	White Kidney	MSU	2021/2022
Clouseau	KDB/Natural	Light Red Kidney	Seminis	2021/2022
Coho	KDB/Natural	Light Red Kidney	MSU	2021/2022
Denali	KDB/Natural	White Kidney	MSU	2021/2022
SVS-0863	KDB	Yellow	Seminis	2021/2022
K16136	KDB/Natural	Dark Red Kidney	MSU	2021
K16640	KDB/GH	Light Red Kidney	MSU	2021
K16911	KDB	White Kidney	MSU	2021

Table 2.2 (cont'd)

K17201	KDB	Dark Red Kidney	MSU	2021
K17702	KDB/Natural	Light Red Kidney	MSU	2021
K17703	KDB/Natural	Light Red Kidney	MSU	2021
K17704	KDB/Natural	Light Red Kidney	MSU	2021
K18312	KDB/Natural	Dark Red Kidney	MSU	2021
K18907	KDB	White Kidney	MSU	2021
K19111	KDB	Dark Red Kidney	MSU	2021
K19120	KDB/Natural	Dark Red Kidney	MSU	2021
K19608	KDB/Natural	Light Red Kidney	MSU	2021
K19610	KDB/GH/Natural	Light Red Kidney	MSU	2021/2022
K19817	KDB/GH/Natural	White Kidney	MSU	2021/2022
K19830	KDB/Natural	White Kidney	MSU	2021/2022
K19831	KDB/Natural	White Kidney	MSU	2021/2022
K19832	KDB/GH/Natural	White Kidney	MSU	2021/2022
K20210	KDB/Natural	Dark Red Kidney	MSU	2021
K20212	KDB/Natural	Dark Red Kidney	MSU	2021/2022
K20217	KDB/Natural	Dark Red Kidney	MSU	2021/2022
K20221	KDB/Natural	Dark Red Kidney	MSU	2021/2022
K20234	KDB/Natural	Dark Red Kidney	MSU	2021
K20235	KDB/Natural	Dark Red Kidney	MSU	2021
K20239	KDB/Natural	Dark Red Kidney	MSU	2021/2022
K20712	KDB/GH/Natural	Light Red Kidney	MSU	2021
K20715	KDB/GH/Natural	Light Red Kidney	MSU	2021/2022
K20717	KDB/Natural	Light Red Kidney	MSU	2021/2022

Table 2.2 (cont'd)

K20720	KDB/Natural	Light Red Kidney	MSU	2021
K20721	KDB/GH/Natural	Dark Red Kidney	MSU	2021/2022
K20728	KDB/Natural	Light Red Kidney	MSU	2021
K20730	KDB/GH/Natural	Light Red Kidney	MSU	2021/2022
K20732	KDB/GH/Natural	Light Red Kidney	MSU	2021/2022
K20734	KDB/Natural	Light Red Kidney	MSU	2021/2022
K20742	KDB/Natural	Light Red Kidney	MSU	2021/2022
K20743	KDB/Natural	Light Red Kidney	MSU	2021/2022
K20744	KDB/Natural	Light Red Kidney	MSU	2021/2022
K20745	KDB/Natural	Light Red Kidney	MSU	2021/2022
K20749	KDB/Natural	Light Red Kidney	MSU	2021
Montcalm	KDB/Natural (2021)	Dark Red Kidney	MSU	2021/2022
ND Whitetail	KDB/Natural (2021)	White Kidney	NDSU	2021/2022
Patron	KDB	Yellow	OSU	2021/2022
Red Cedar	KDB/Natural	Dark Red Kidney	MSU	2021/2022
Red Hawk	KDB/GH/Natural (2021)	Dark Red Kidney	MSU	2021/2022
Rosie	KDB	Light Red Kidney	NDSU	2021/2022
Snowdon	KDB/Natural	White Kidney	MSU	2021/2022
Y17502	KDB	Yellow	MSU	2022
Y18703	KDB	Yellow	MSU	2022
Y19801	KDB	Yellow	MSU	2022
Y19804	KDB	Yellow	MSU	2022
Y19808	KDB	Yellow	MSU	2022
Y19810	KDB	Yellow	MSU	2022

Table 2.2 (cont'd)

Y19815	KDB	Yellow	MSU	2022
Y19817	KDB	Yellow	MSU	2021/2022
Yellowstone	KDB/GH	Yellow	MSU	2021/2022
Adams	MAB	Black	MSU	2022
Armada	MAB	Navy	Provita	2021/2022
B18504	MAB	Black	MSU	2022
B19309	MAB	Black	MSU	2021/2022
B19330	MAB	Black	MSU	2022
B19332	MAB	Black	MSU	2022
B19344	MAB	Black	MSU	2021/2022
B20536	MAB	Black	MSU	2022
B20547	MAB	Black	MSU	2021/2022
B20549	MAB	Black	MSU	2022
B20591	MAB	Black	MSU	2021/2022
B20597	MAB	Black	MSU	2021/2022
B20599	MAB	Black	MSU	2022
B20602	MAB	Black	MSU	2022
B21708	MAB	Black	MSU	2022
B21710	MAB	Black	MSU	2022
B21714	MAB	Black	MSU	2022
Black Bear	MAB	Black	Provita	2021/2022
Black Beard	MAB	Black	Provita	2021/2022
Black Tails	MAB	Black	Provita	2021/2022
Blizzard	MAB	Navy	Provita	2021/2022

Table 2.2 (cont'd)

Caldera	MAB	Small Red	Provita	2021/2022
Cayenne	MAB	Small Red	MSU	2022
Charro	MAB	Pinto	MSU	2022
Coral	MAB	Pink	MSU	2022
Eiger	MAB	Great Northern	MSU	2022
G19611	MAB	Great Northern	MSU	2021
G19613	MAB	Great Northern	MSU	2021/2022
G21811	MAB	Great Northern	MSU	2022
HMS Bounty	MAB	Navy	Provita	2021/2022
Liberty	MAB	Navy	Provita	2022
Medalist	MAB	Navy	Provita	2021/2022
Merlin	MAB	Navy	Provita	2021/2022
N18103	MAB	Navy	MSU	2022
N19226	MAB	Navy	MSU	2021/2022
N19239	MAB	Navy	MSU	2021
N19246	MAB	Navy	MSU	2021/2022
N19285	MAB	Navy	MSU	2021
N20388	MAB	Navy	MSU	2021/2022
N20395	MAB	Navy	MSU	2021/2022
N20404	MAB	Navy	MSU	2021/2022
N21511	MAB	Navy	MSU	2022
N21525	MAB	Navy	MSU	2022
Nimbus	MAB	Black	Provita	2021
P16901	MAB	Pinto	MSU	2021

Table 2.2 (cont'd)

P19103	MAB	Pinto	MSU	2021/2022
P19707	MAB	Pinto	MSU	2021
P19713	MAB	Pinto	MSU	2021/2022
R12844	MAB	Small Red	MSU	2021
R20627	MAB	Small Red	MSU	2021/2022
R20639	MAB	Small Red	MSU	2021
R20652	MAB	Small Red	MSU	2021
R20659	MAB	Small Red	MSU	2021/2022
R20667	MAB	Small Red	MSU	2021/2022
R20669	MAB	Small Red	MSU	2022
R20683	MAB	Small Red	MSU	2021
Rosetta	MAB	Pink	MSU	2022
Ruby	MAB	Small Red	Provita	2021/2022
S08418	MAB	Pink	MSU	2021
S18904	MAB	Pink	MSU	2021
Spectre	MAB	Black	Provita	2021/2022
Valiant	MAB	Navy	Provita	2021/2022
Viper	MAB	Small Red	Provita	2021/2022
Zenith	MAB	Black	MSU	2021/2022
Zorro	MAB	Black	MSU	2021/2022

Figure 2.1: Screening scale used to evaluate *Fusarium oxysporum* inoculated lines for root damage. Lines were evaluated on a 1-7 scale developed by Schneider and Kelly 2001, where 1 indicates no disease and 7 indicates a non functional, completely rotted root system.

Table 2.3: T-test of the difference between Least squares means of the inoculated vs non-inoculated treatment to validate inoculum concentration.

Population	Isolate	2021	2022
KDB Lines- Field	*F. oxysporum*	<2.2 x 10^-16	<2.2 x 10^-16
ADP Lines- Field	*F. oxysporum*	<2.2 x 10^-16	<2.2 x 10^-16
ADP and KDB Lines- Greenhouse	*F. oxysporum*	-	<2.2 x 10^-16
Middle American Lines- Field	*R. solani*	<2.2 x 10^-16	<2.2 x 10^-16

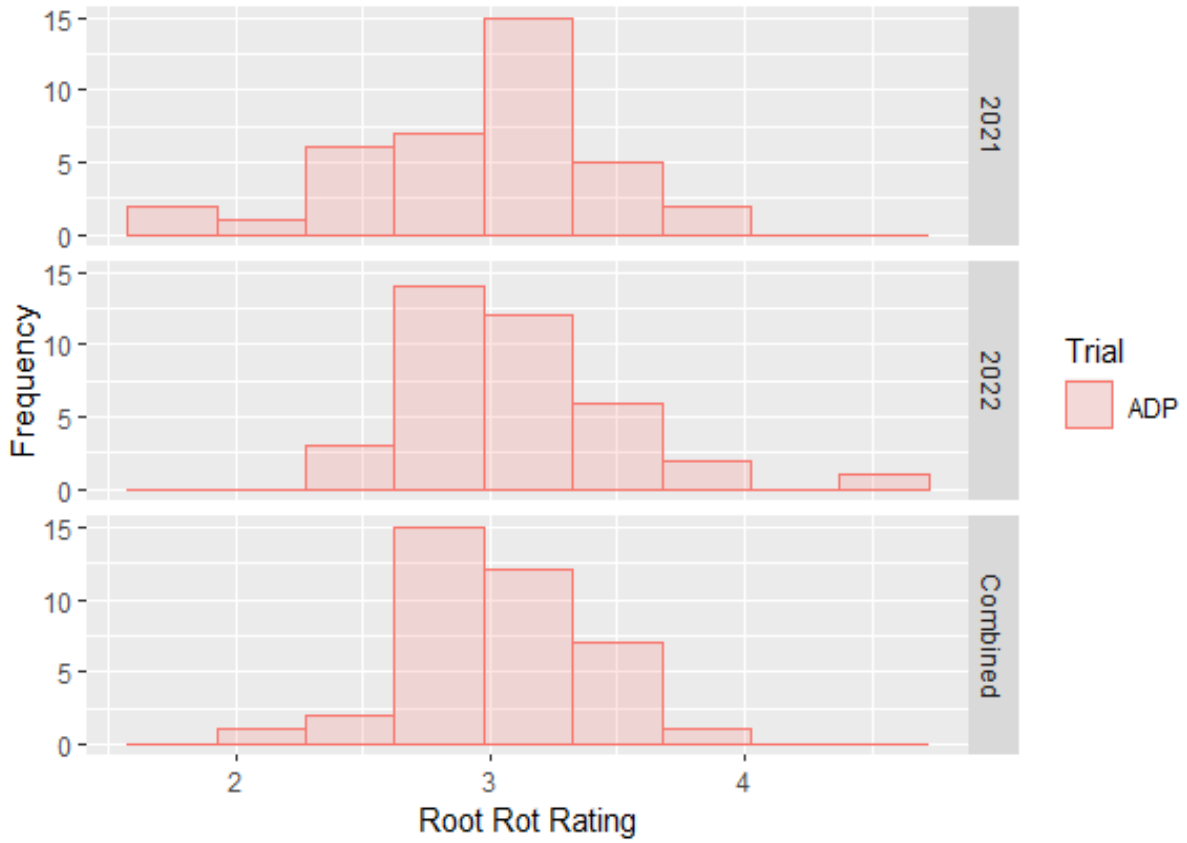

Figure 2.2a: Histogram of Least Squares means in the Andean diversity panel (ADP) *Fusarium oxysporum* trial (inoculated treatment only) using a 1-7 root rot disease severity score.

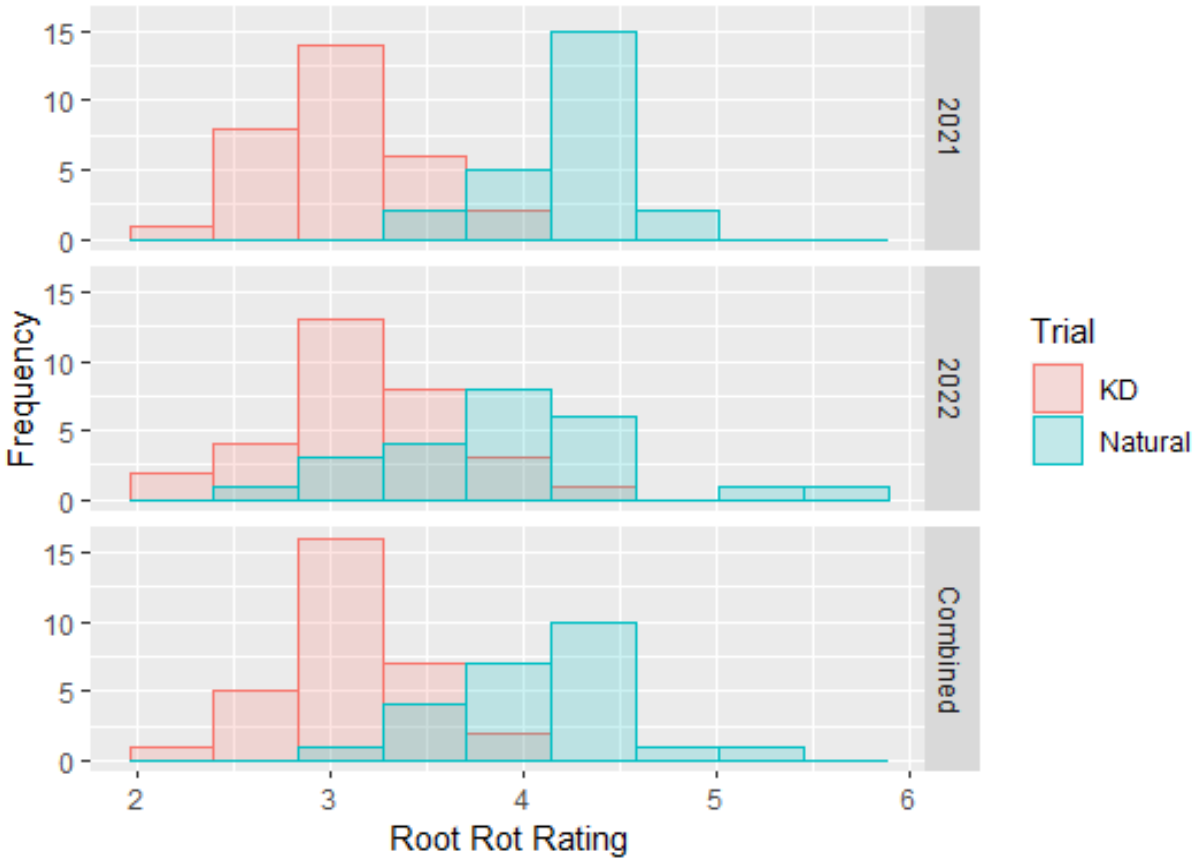

Figure 2.2b: Histogram of Least Squares means in the kidney breeding line (KDB) *Fusarium oxysporum* trials and Natural infection trials (inoculated treatment only) using a 1-7 root rot disease severity score.

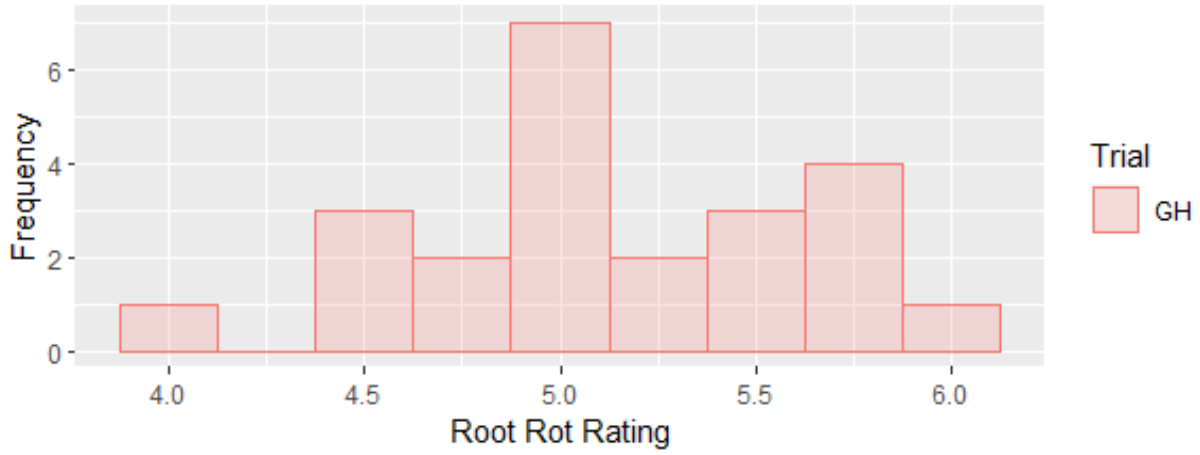

Figure 2.2c: Histogram of Least Squares means in the greenhouse *Fusarium oxysporum* trial (inoculated treatment only) using a 1-7 root rot disease severity score.

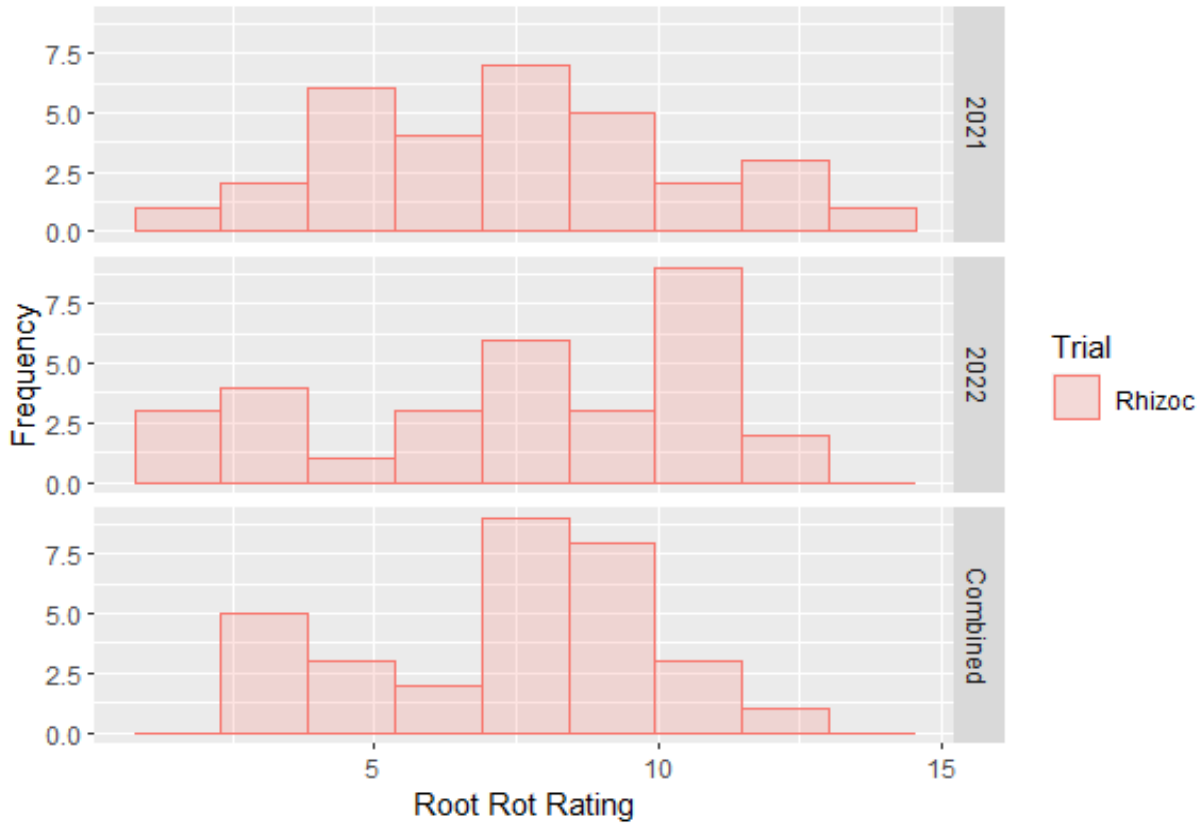

Figure 2.2d: Histogram plot of Least Squares means, for the Middle American breeding line trial inoculated with *Rhizoctonia solani* using the difference in post-emergence damping off in the inoculated treatment.

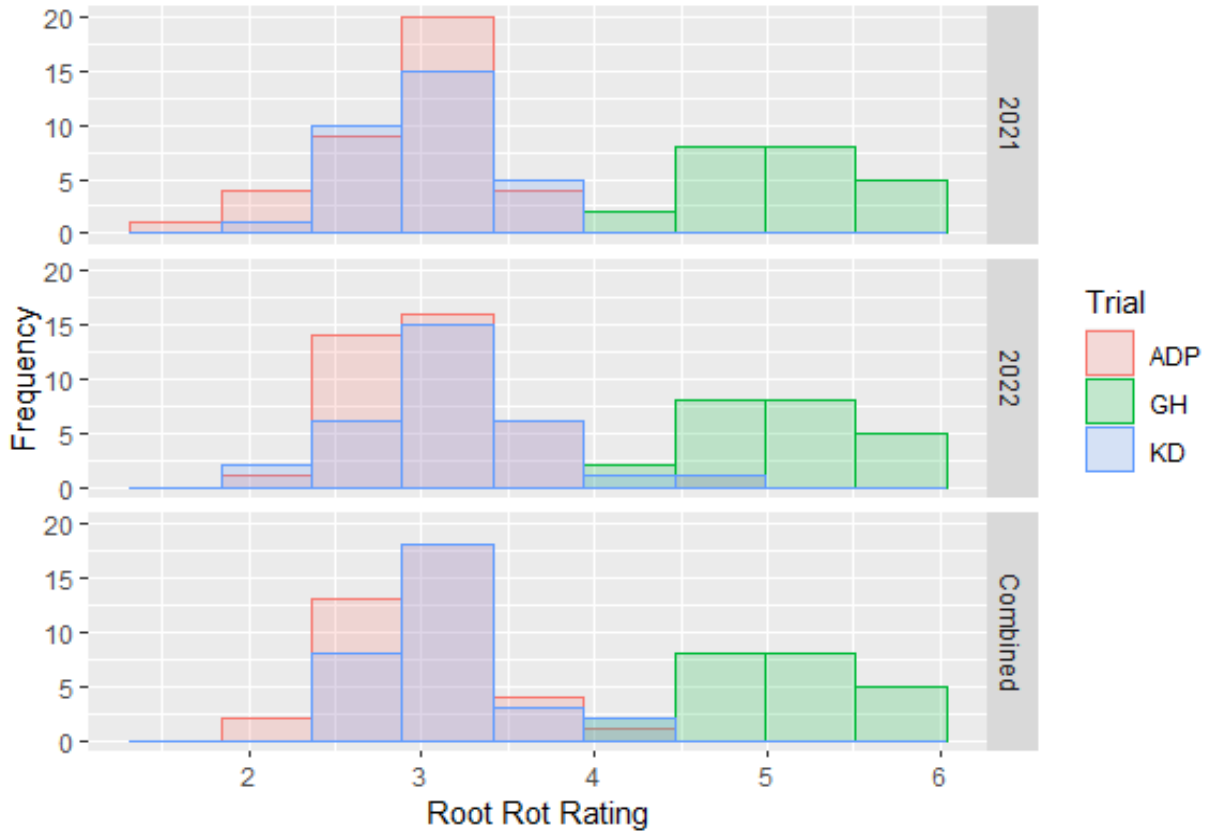

Figure 2.2e: Histograms of Least Squares means in the combined analysis of the Andean diversity panel and Kidney breeding line field trials and the greenhouse trial (2022 only) of the inoculated treatment only using a 1-7 root rot disease severity score.

Table 2.4: Table of variance components, heritability, and coefficient of variation for each population, utilizing the inoculated treatment only. Starred values are significant at a 0.05 significance level.

Population	Year	Isolate	σ^2_G	σ^2_{GxY}	σ^2_Y	σ^2_e	H^2	CV %
KDB Field Trial (KDB)	**Combined**	**F. oxysporum**	0.08*	0.04	>0.001	1.47*	0.29	42.13
	2021		0.09*	-	-	0.99*	0.26	34.99
	2022		0.17*	-	-	2.08*	0.24	49.26
ADP Field Trial (ADP)	**Combined**	***F. oxysporum***	0.07*	0.01	>0.001	1.80*	0.24	45.59
	2021		0.11*	-	-	1.21*	0.28	39.13
	2022		0.08	-	-	2.10*	0.14	48.19
Greenhouse Trial (GH)	**2022**	***F. oxysporum***	0.10	-	-	2.37*	0.12	31.04
Natural Infection (KDB)	**Combined**	**N/A**	0.06	0.08	0.05	1.13*	0.19	27.69
	2021		0.04	-	-	0.98*	0.11	23.94
	2022		0.38*	-	-	1.50*	0.43	35.32
Middle American Field Trial (MAB)	**Combined**	***R. solani***	3.31*	2.42	0.00002	1.75*	0.49	67.32
	2021		4.95*	-	-	16.31*	0.55	65.11
	2022		6.72*	-	-	20.1*	0.57	71.06

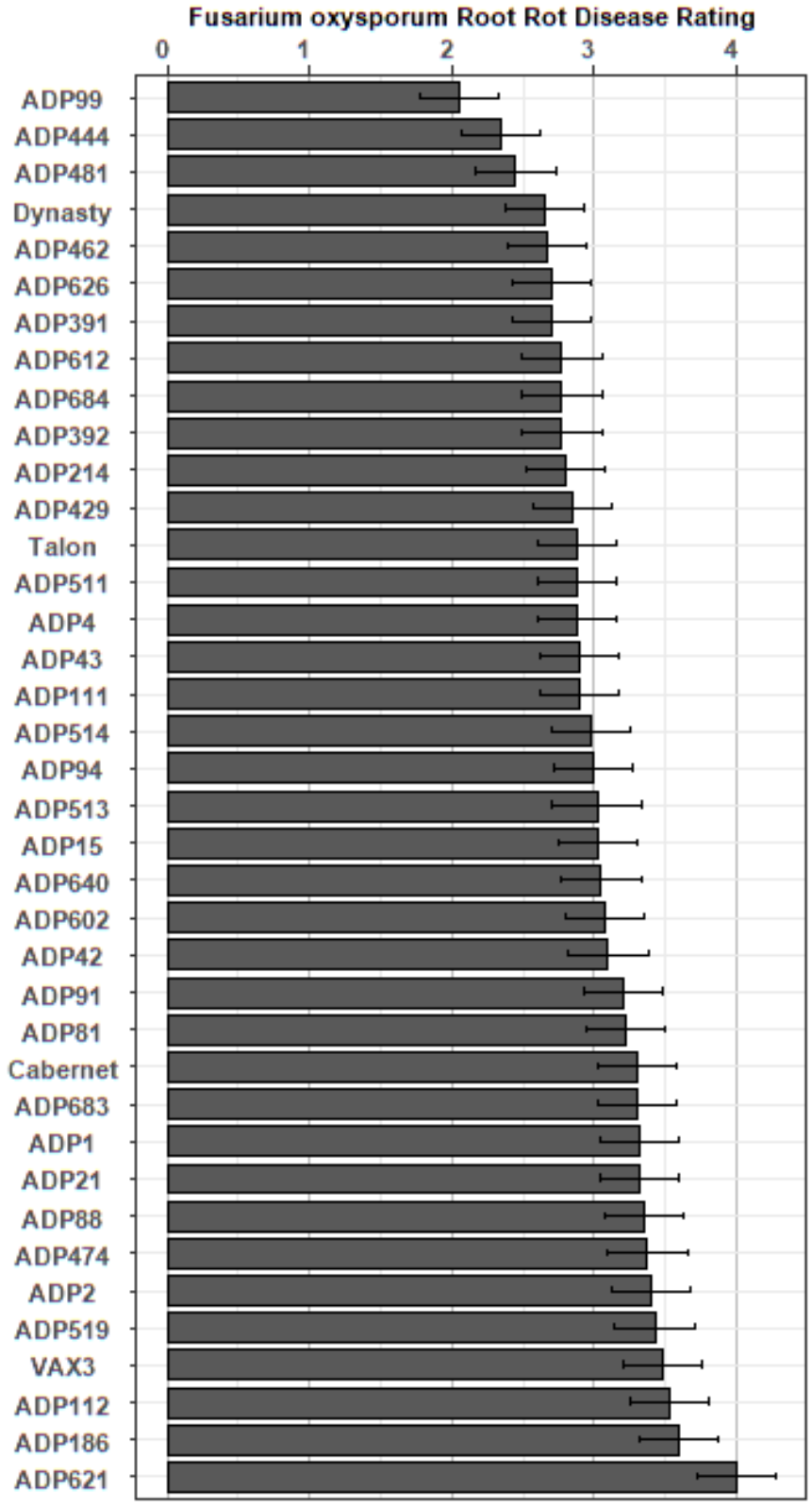

Figure 2.3: Histogram of Least Squares means by genotype with standard error bars, ADP trial combined analysis.

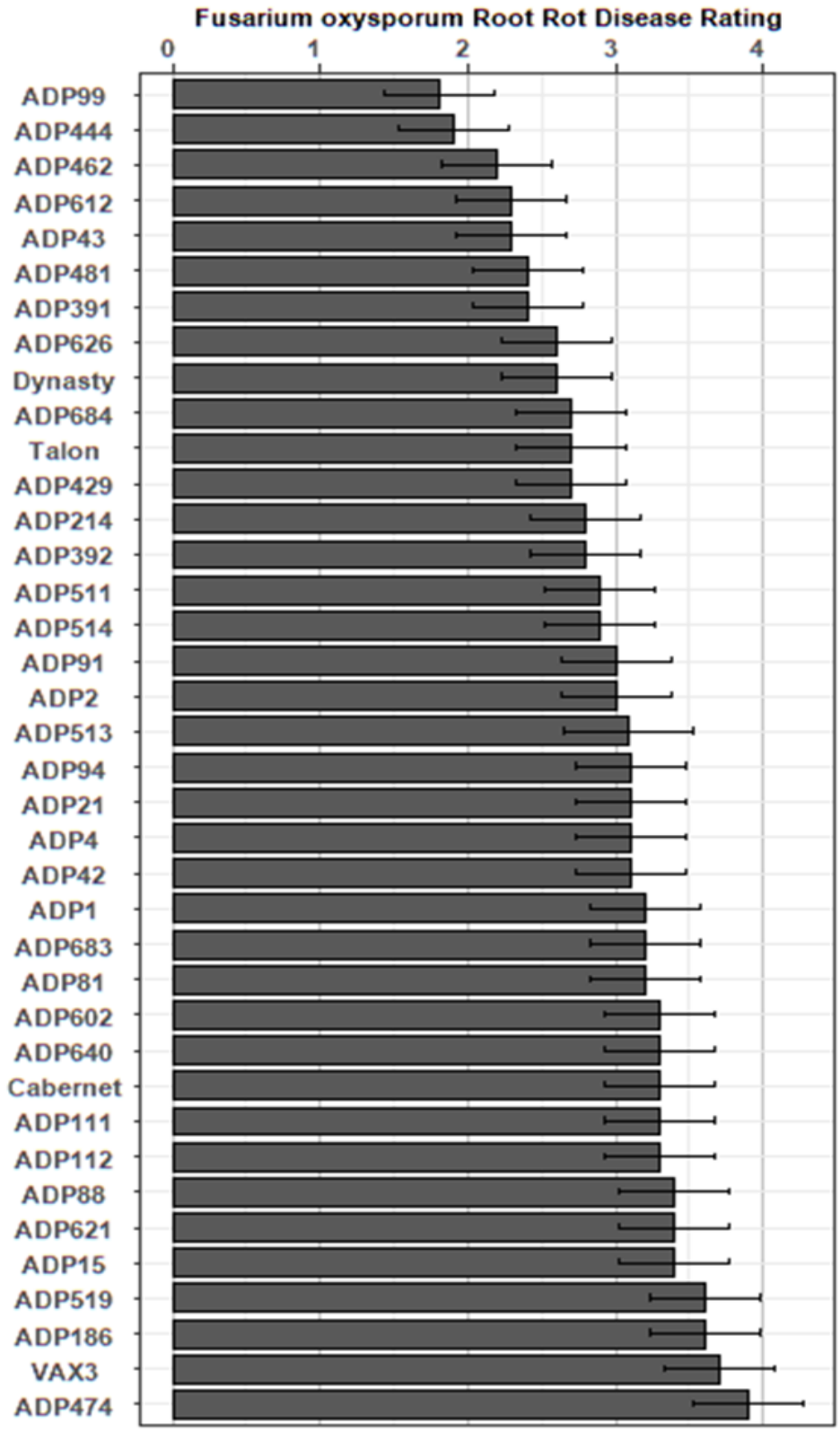

Figure 2.4: Histogram of Least Squares means by genotype with standard error bars, ADP trial 2021 analysis.

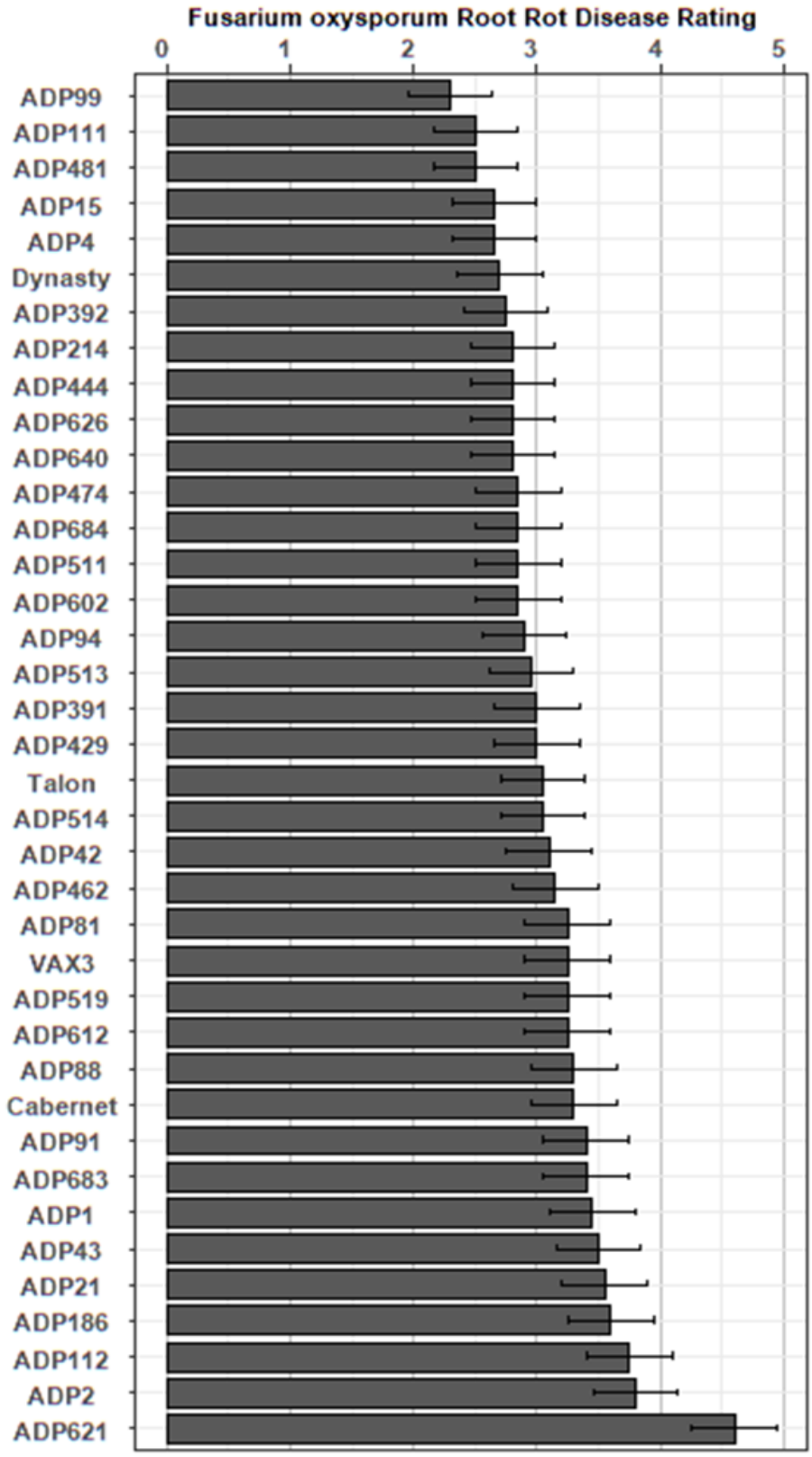

Figure 2.5: Histogram of Least Squares means by genotype with standard error bars, ADP trial 2022 analysis.

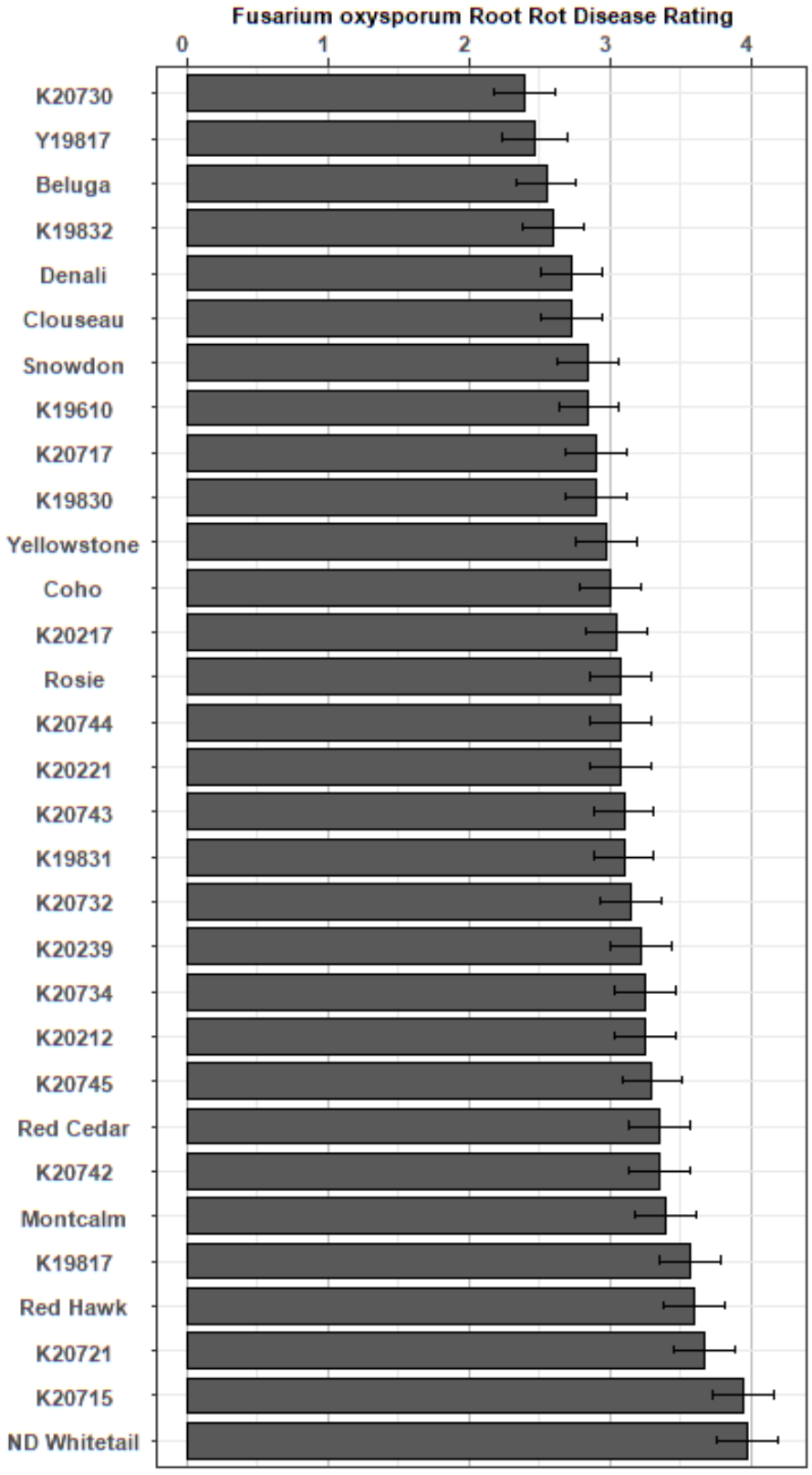

Figure 2.6: Histogram of Least Squares means by genotype with standard error bars, KDB trial combined analysis.

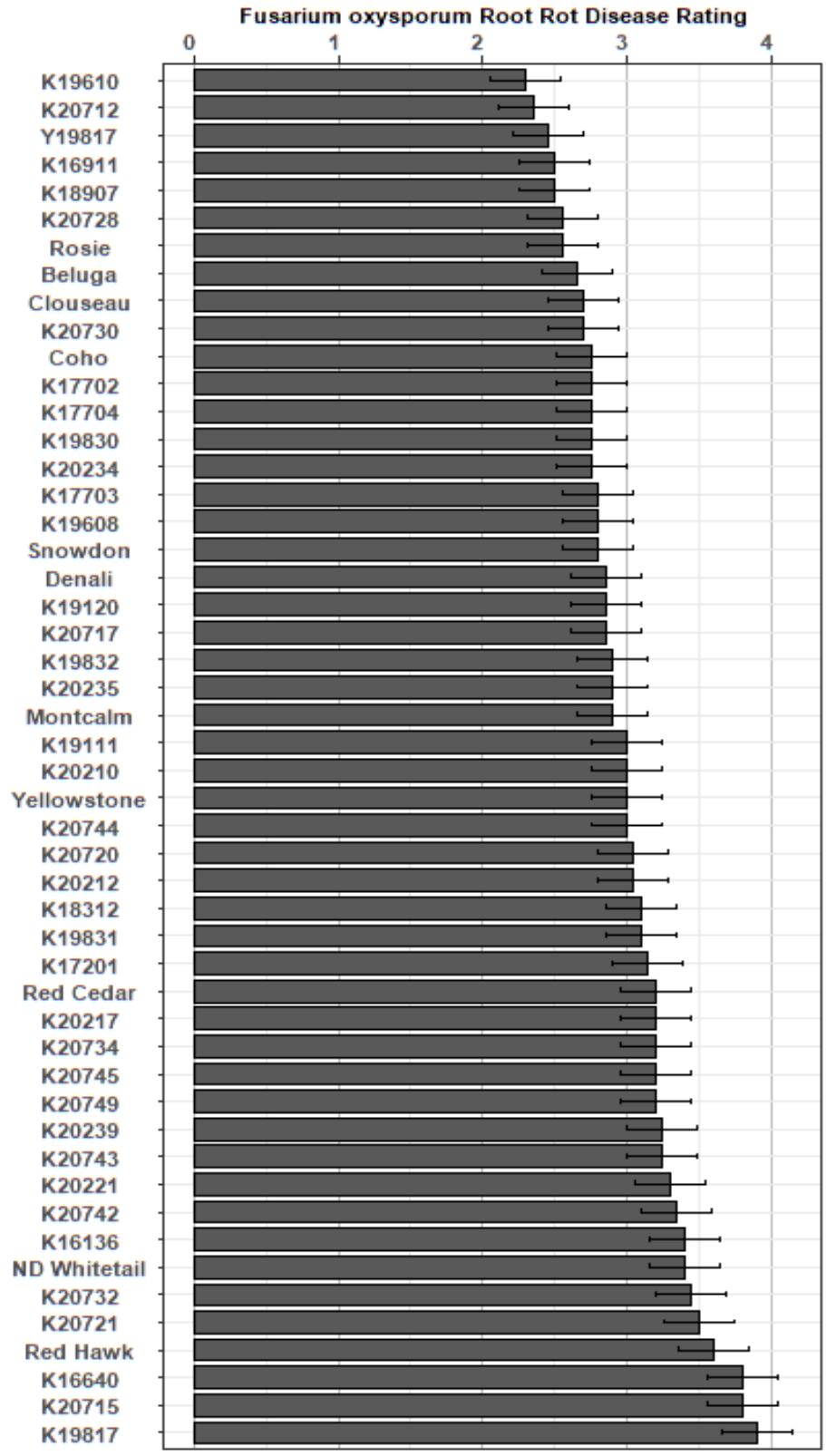

Figure 2.7: Histogram of Least Squares means by genotype with standard error bars, KDB trial 2021 analysis.

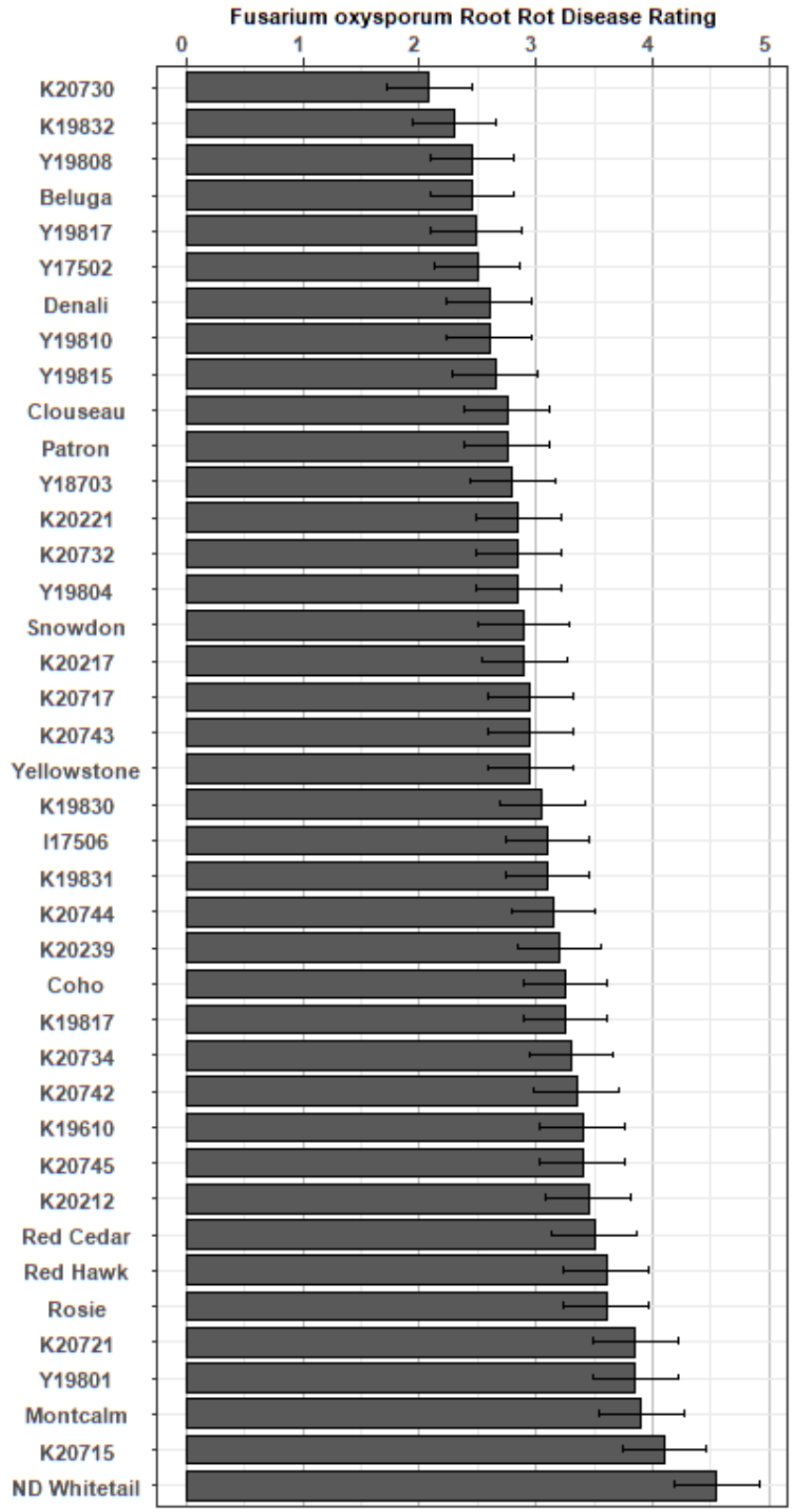

Figure 2.8: Histogram of Least Squares means by genotype with standard error bars, KDB trial 2022 analysis.

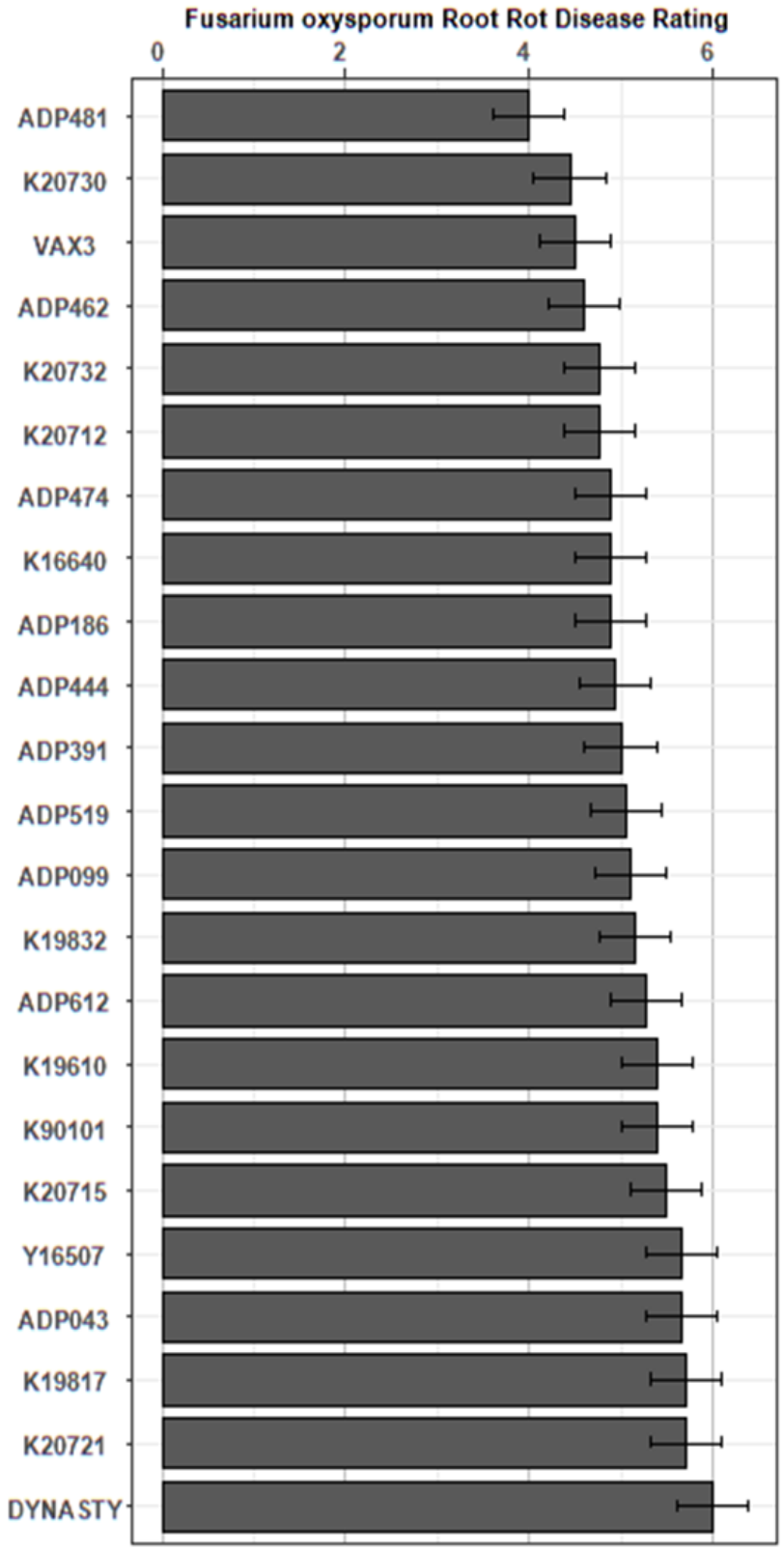

Figure 2.9: Histogram of Least Squares means by genotype with standard error bars, Greenhouse trial.

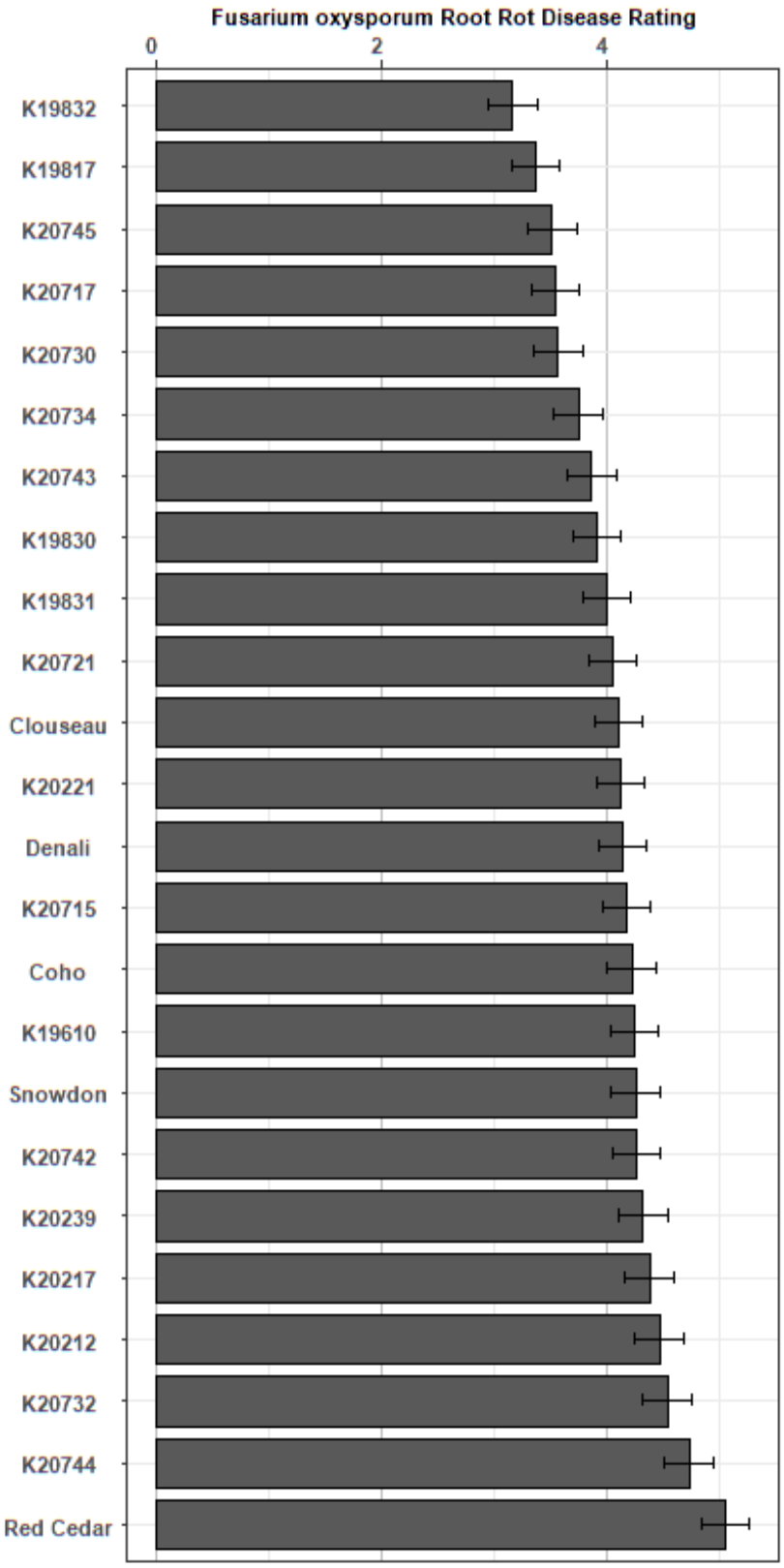

Figure 2.10: Histogram of Least Squares means by genotype with standard error bars, Natural infection trial combined analysis.

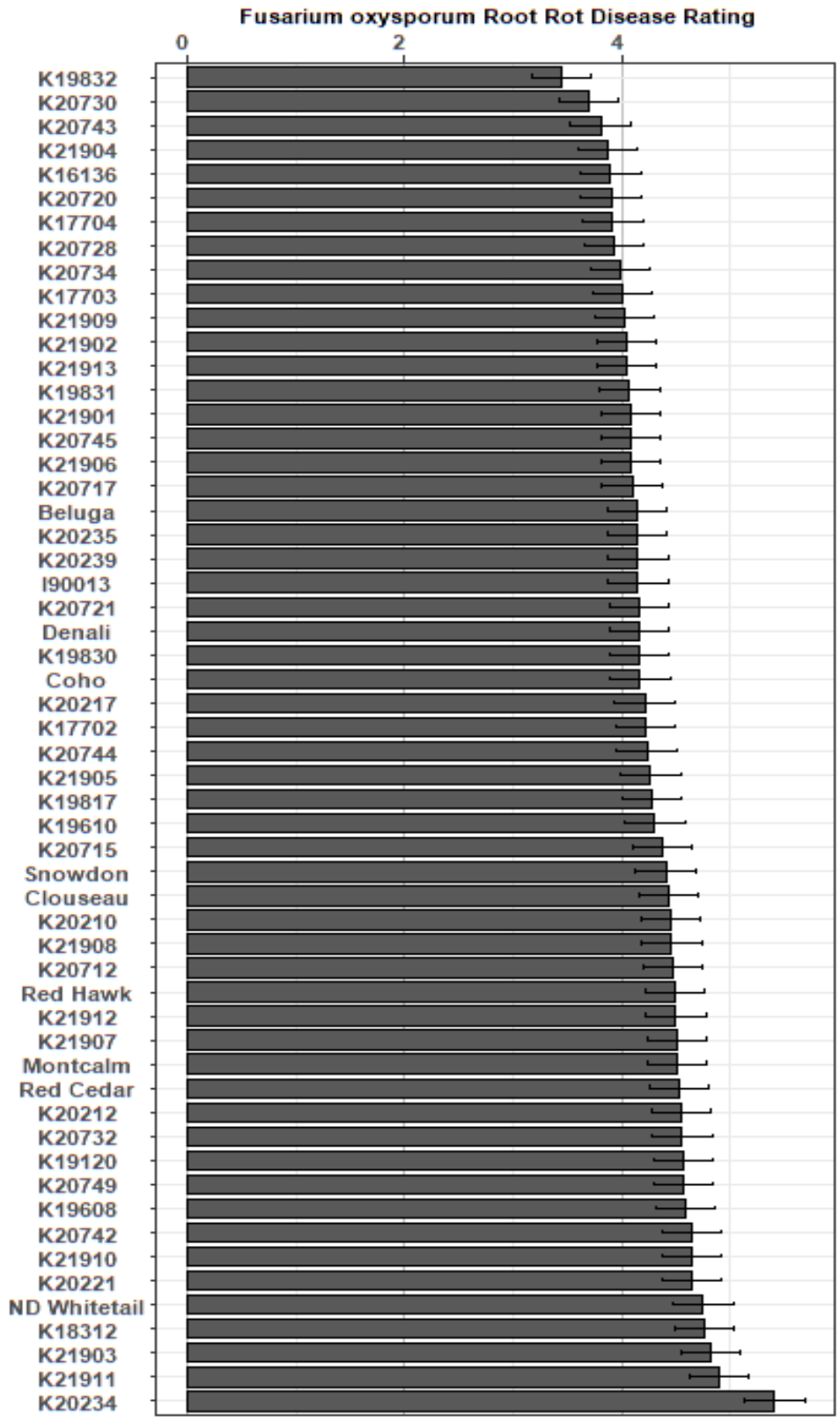

Figure 2.11: Histogram of Least Squares means by genotype with standard error bars, Natural infection trial 2021 analysis.

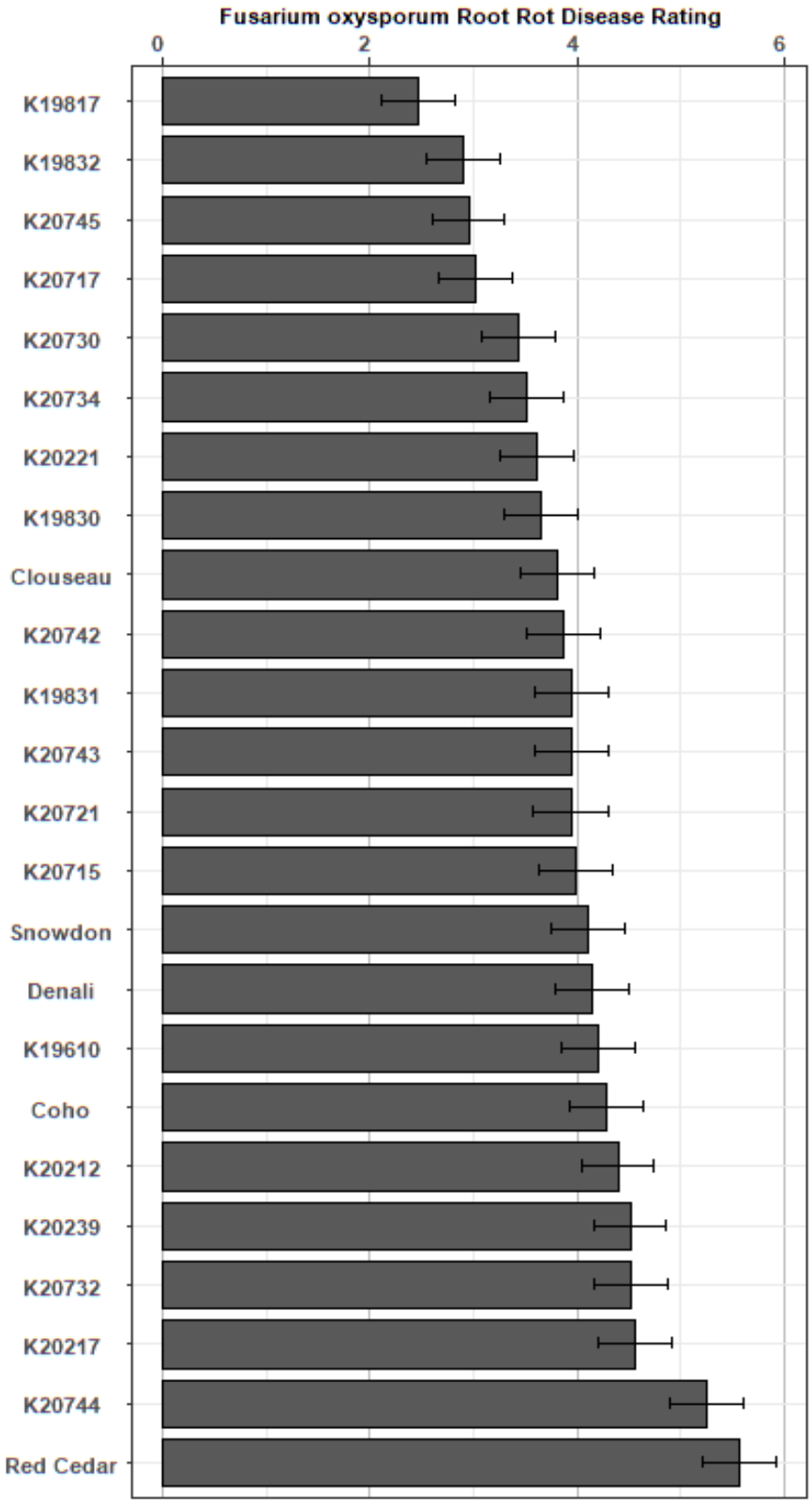

Figure 2.12: Histogram of Least Squares means by genotype with standard error bars, Natural infection trial 2022 analysis.

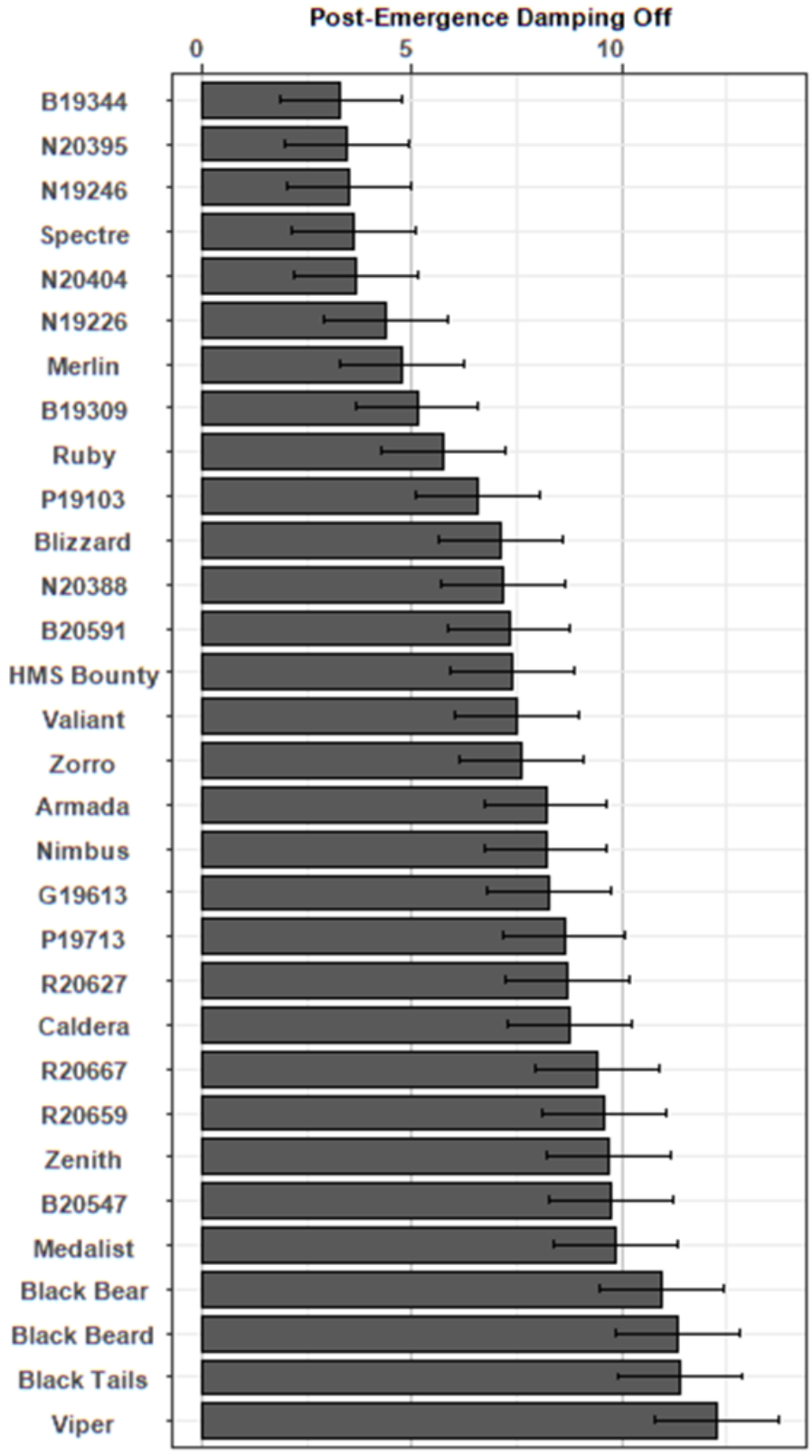

Figure 2.13: Histogram of Least Squares means by genotype with standard error bars, Middle-American trial combined analysis.

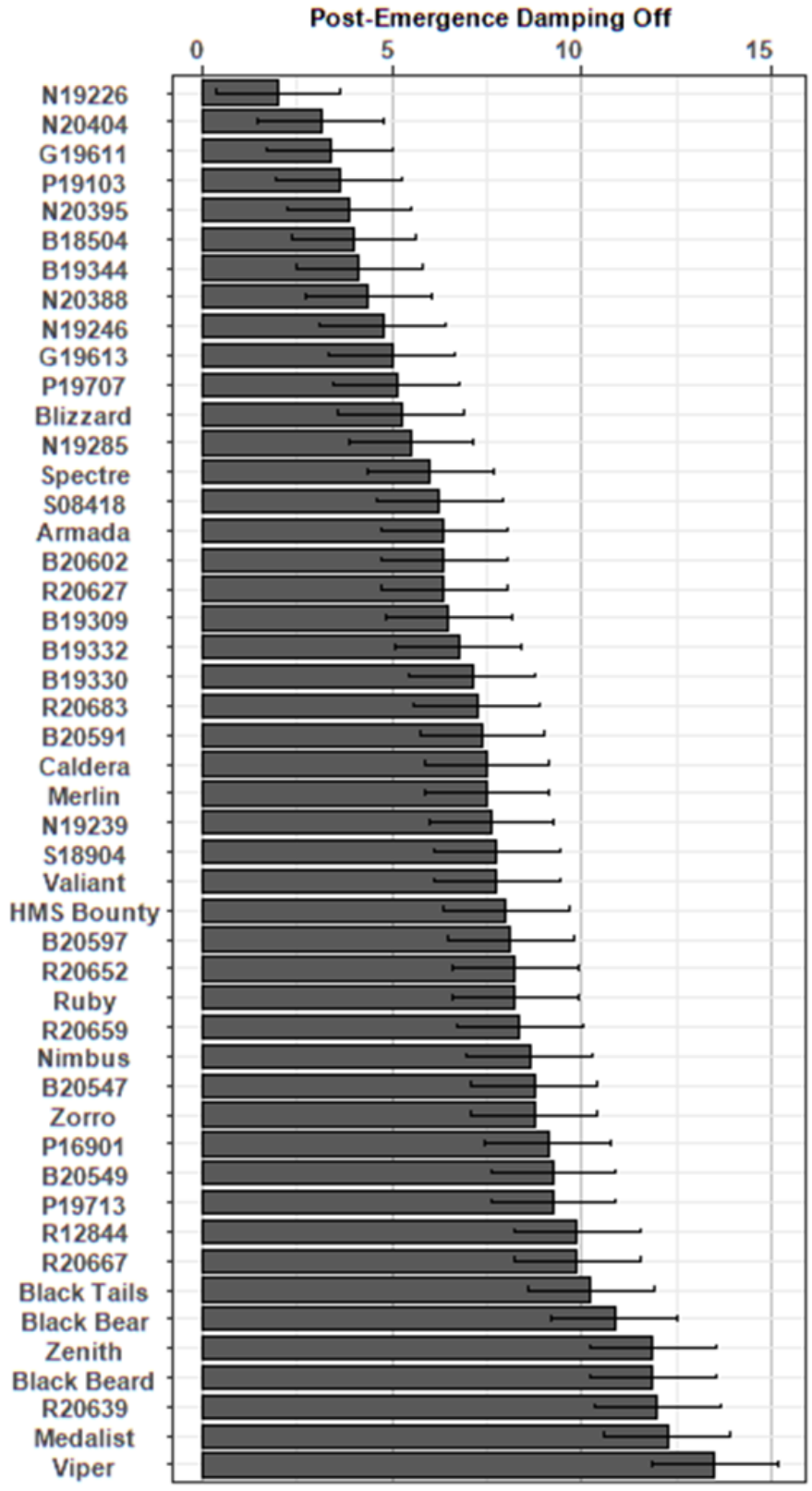

Figure 2.14: Histogram of Least Squares means by genotype with standard error bars, Middle-American trial 2021 analysis.

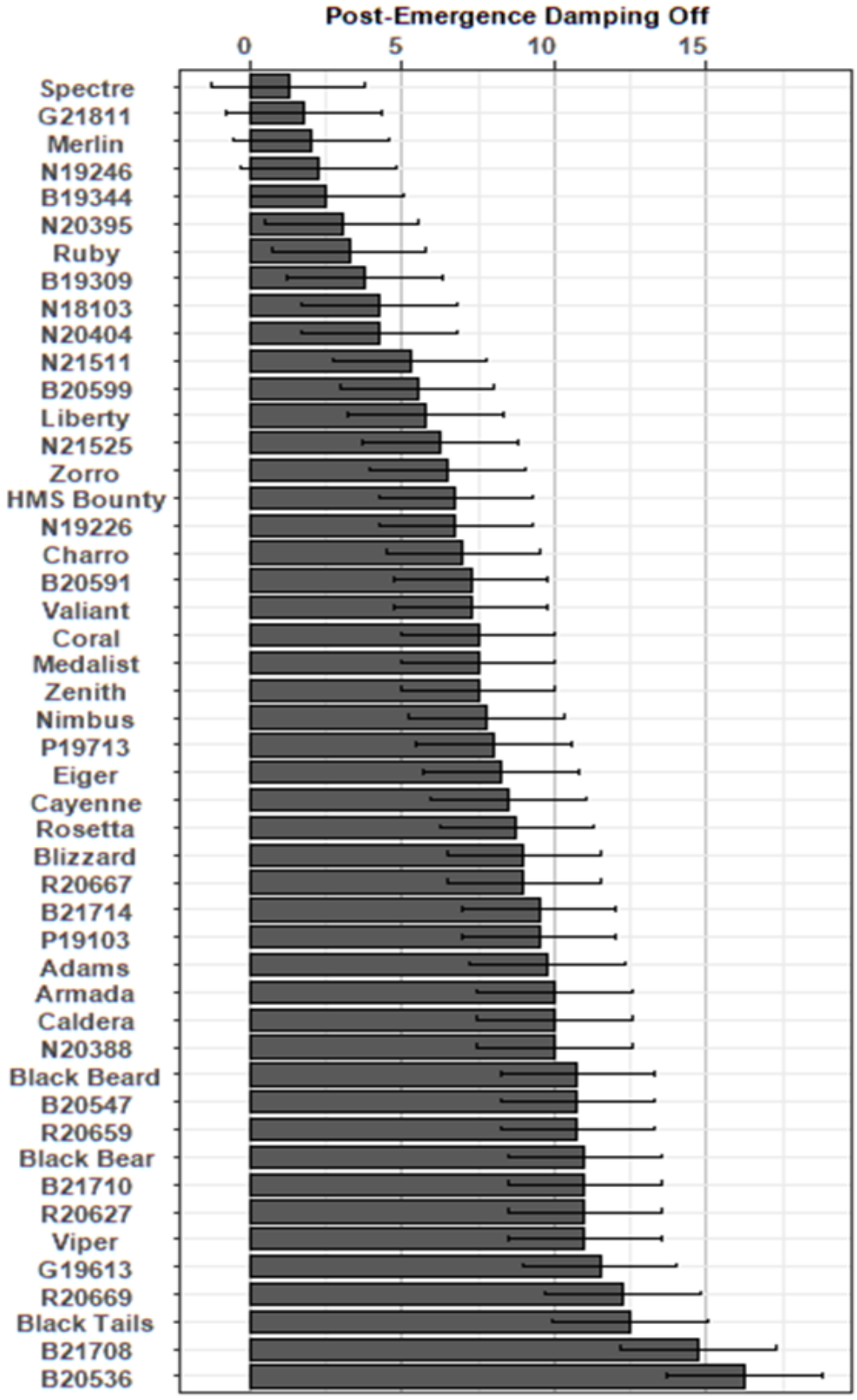

Figure 2.15: Histogram of Least Squares means by genotype with standard error bars, Middle-American trial 2022 analysis.

Table 2.5: Least squares means, least significant difference, and standard error by line for root rot rating of the ADP *Fusarium oxysporum* trial combined analysis. Asterisk (*) indicates lines that are not significantly different from the most resistant line.

Line	Lsmean	SE
ADP99*	2.05	0.28
ADP444*	2.35	0.28
ADP481*	2.45	0.28
Dynasty*	2.65	0.28
ADP462*	2.67	0.28
ADP391*	2.70	0.28
ADP626*	2.70	0.28
ADP392*	2.77	0.28
ADP612*	2.77	0.28
ADP684*	2.77	0.28
ADP214	2.80	0.28
ADP429	2.85	0.28
ADP4	2.87	0.28
ADP511	2.87	0.28
Talon	2.87	0.28
ADP111	2.90	0.28
ADP43	2.90	0.28
ADP514	2.97	0.28
ADP94	3.00	0.28
ADP15	3.02	0.28
ADP513	3.02	0.31
ADP640	3.05	0.28

Table 2.5 (cont'd)

ADP602	3.07	0.28
ADP42	3.10	0.28
ADP91	3.20	0.28
ADP81	3.22	0.28
ADP683	3.30	0.28
Cabernet	3.30	0.28
ADP1	3.32	0.28
ADP21	3.32	0.28
ADP88	3.35	0.28
ADP474	3.37	0.28
ADP2	3.40	0.28
ADP519	3.42	0.28
VAX3	3.47	0.28
ADP112	3.52	0.28
ADP186	3.60	0.28
ADP621	4.00	0.28
LSD	0.73	-

Table 2.6: Least squares means, least significant difference, and standard error by line for root rot rating of the ADP *Fusarium oxysporum* trial 2021 analysis. Asterisk (*) indicates lines that are not significantly different from the most resistant line.

Line	Lsmean	SE
ADP99*	1.80	0.37
ADP444*	1.90	0.37
ADP462*	2.20	0.37
ADP43*	2.30	0.37
ADP612*	2.30	0.37
ADP391*	2.40	0.37
ADP481*	2.40	0.37
ADP626*	2.60	0.37
Dynasty*	2.60	0.37
ADP429*	2.70	0.37
ADP684*	2.70	0.37
Talon*	2.70	0.37
ADP214	2.80	0.37
ADP392	2.80	0.37
ADP511	2.90	0.37
ADP514	2.90	0.37
ADP2	3.00	0.37
ADP91	3.00	0.37
ADP513	3.09	0.44
ADP21	3.10	0.37
ADP4	3.10	0.37
ADP42	3.10	0.37

Table 2.6 (cont'd)

ADP94	3.10	0.37
ADP1	3.20	0.37
ADP683	3.20	0.37
ADP81	3.20	0.37
ADP111	3.30	0.37
ADP112	3.30	0.37
ADP602	3.30	0.37
ADP640	3.30	0.37
Cabernet	3.30	0.37
ADP15	3.40	0.37
ADP621	3.40	0.37
ADP88	3.40	0.37
ADP186	3.60	0.37
ADP519	3.60	0.37
VAX3	3.70	0.37
ADP474	3.90	0.37
LSD	0.97	-

Table 2.7: Least squares means, least significant difference, and standard error by line for root rot rating of the ADP *Fusarium oxysporum* trial 2022 analysis. Asterisk (*) indicates lines that are not significantly different from the most resistant line.

Line	Lsmean	SE
ADP99*	2.30	0.34
ADP111*	2.50	0.34
ADP481*	2.50	0.34
ADP15*	2.65	0.34
ADP4*	2.65	0.34
Dynasty*	2.70	0.34
ADP392*	2.75	0.34
ADP214*	2.80	0.34
ADP444*	2.80	0.34
ADP626*	2.80	0.34
ADP640*	2.80	0.34
ADP474*	2.85	0.34
ADP511*	2.85	0.34
ADP602*	2.85	0.34
ADP684*	2.85	0.34
ADP94*	2.90	0.34
ADP513*	2.95	0.34
ADP391*	3.00	0.34
ADP429*	3.00	0.34
ADP514*	3.05	0.34
Talon*	3.05	0.34
ADP42*	3.10	0.34

Table 2.7 (cont'd)

ADP462*	3.15	0.34
ADP519	3.25	0.34
ADP81	3.25	0.34
VAX3	3.25	0.34
ADP612	3.25	0.34
ADP88	3.30	0.34
Cabernet	3.30	0.34
ADP683	3.40	0.34
ADP91	3.40	0.34
ADP1	3.45	0.34
ADP43	3.50	0.34
ADP21	3.55	0.34
ADP186	3.60	0.34
ADP112	3.75	0.34
ADP2	3.80	0.34
ADP621	4.60	0.34
LSD	0.90	-

Table 2.8: Least squares means, least significant difference, and standard error by line for root rot rating of the Kidney *Fusarium oxysporum* trial combined analysis. Asterisk (*) indicates lines that are not significantly different from the most resistant line.

Line	Lsmean	SE
K20730*	2.39	0.22
Y19817*	2.47	0.23
Beluga*	2.55	0.22
K19832*	2.6	0.22
Clouseau*	2.73	0.22
Denali*	2.73	0.22
Snowdon*	2.84	0.22
K19610*	2.85	0.22
K19830*	2.9	0.22
K20717*	2.9	0.22
Yellowstone	2.98	0.22
Coho	3	0.22
K20217	3.05	0.22
K20744	3.08	0.22
K20221	3.08	0.22
Rosie	3.08	0.22
K20743	3.1	0.22
K19831	3.1	0.22
K20732	3.15	0.22
K20239	3.23	0.22
K20734	3.25	0.22
K20212	3.25	0.22

Table 2.8 (cont'd)

K20745	3.3	0.22
Red Cedar	3.35	0.22
K20742	3.35	0.22
Montcalm	3.4	0.22
K19817	3.58	0.22
Red Hawk	3.6	0.22
K20721	3.68	0.22
K20715	3.95	0.22
ND Whitetail	3.98	0.22
LSD	0.53	-

Table 2.9: Least squares means, least significant difference, and standard error by line for root rot rating of the Kidney *Fusarium oxysporum* trial 2021 analysis. Asterisk (*) indicates lines that are not significantly different from the most resistant line.

Line	Lsmean	SE
K19610*	2.30	0.24
K20712*	2.35	0.24
Y19817*	2.45	0.24
K16911*	2.50	0.24
K18907*	2.50	0.24
K20728*	2.55	0.24
Rosie*	2.55	0.24
Beluga*	2.65	0.24
Clouseau*	2.70	0.24
K20730*	2.70	0.24
K17702*	2.75	0.24
K17704*	2.75	0.24
K19830*	2.75	0.24
K20234*	2.75	0.24
Coho*	2.75	0.24
K17703*	2.80	0.24
K19608*	2.80	0.24
Snowdon*	2.80	0.24
Denali*	2.85	0.24
K20717*	2.85	0.24
K19120*	2.85	0.24
Montcalm*	2.90	0.24

Table 2.9 (cont'd)

K19832*	2.90	0.24
K20235*	2.90	0.24
Yellowstone	3.00	0.24
K20744	3.00	0.24
K19111	3.00	0.24
K20210	3.00	0.24
K20720	3.05	0.24
K20212	3.05	0.24
K18312	3.10	0.24
K19831	3.10	0.24
K17201	3.15	0.24
K20734	3.20	0.24
K20745	3.20	0.24
K20749	3.20	0.24
K20217	3.20	0.24
Red Cedar	3.20	0.24
K20743	3.25	0.24
K20239	3.25	0.24
K20221	3.30	0.24
K20742	3.35	0.24
K16136	3.40	0.24
ND Whitetail	3.40	0.24
K20732	3.45	0.24
K20721	3.50	0.24

Table 2.9 (cont'd)

Red Hawk	3.60	0.24
K16640	3.80	0.24
K20715	3.80	0.24
K19817	3.90	0.24
LSD	0.62	-

Table 2.10: Least squares means, least significant difference, and standard error by line for root rot rating of the Kidney *Fusarium oxysporum* trial 2022 analysis. Asterisk (*) indicates lines that are not significantly different from the most resistant line.

Line	Lsmean	SE
K20730*	2.09	0.37
K19832*	2.30	0.36
Y19808*	2.45	0.36
Beluga*	2.45	0.36
Y19817*	2.48	0.4
Y17502*	2.50	0.36
Y19810*	2.60	0.36
Denali*	2.60	0.36
Y19815*	2.65	0.36
Patron*	2.75	0.36
Clouseau*	2.75	0.36
Y18703*	2.80	0.36
K20221*	2.85	0.36
K20732*	2.85	0.36
Y19804*	2.85	0.36
Snowdon*	2.89	0.39
K20217*	2.90	0.36
K20743*	2.95	0.36
K20717*	2.95	0.36
Yellowstone*	2.95	0.36
K19830	3.05	0.36
I17506	3.10	0.36

Table 2.10 (cont'd)

K19831	3.10	0.36
K20744	3.15	0.36
K20239	3.20	0.36
K19817	3.25	0.36
Coho	3.25	0.36
K20734	3.30	0.36
K20742	3.35	0.36
K20745	3.40	0.36
K19610	3.40	0.36
K20212	3.45	0.36
Red Cedar	3.50	0.36
Rosie	3.60	0.36
Red Hawk	3.60	0.36
Y19801	3.85	0.36
K20721	3.85	0.36
Montcalm	3.90	0.36
K20715	4.10	0.36
ND Whitetail	4.55	0.36
LSD	0.90	-

Table 2.11: Least squares means, least significant difference, and standard error by line for root rot rating of the Greenhouse *Fusarium oxysporum* trial analysis. Asterisk (*) indicates lines that are not significantly different from the most resistant line.

Line	Lsmean	SE
ADP481*	4.00	0.39
K20730*	4.44	0.39
VAX3*	4.50	0.39
ADP462*	4.61	0.39
K20712*	4.78	0.39
K20732*	4.78	0.39
ADP474*	4.89	0.39
K16640*	4.89	0.39
ADP186*	4.89	0.39
ADP444*	4.94	0.39
ADP391*	5.00	0.39
ADP519	5.06	0.39
ADP099	5.11	0.39
K19832	5.17	0.39
ADP612	5.28	0.39
K19610	5.39	0.39
Red Hawk	5.39	0.39
K20715	5.50	0.39
Yellowstone	5.67	0.39
ADP043	5.67	0.39
K19817	5.72	0.39
K20721	5.72	0.39

Table 2.11 (cont'd)

DYNASTY	6.00	0.39
LSD	1.01	-

Table 2.12: Least squares means, least significant difference, and standard error by line for root rot rating of the Natural infection trial combined analysis. Asterisk (*) indicates lines that are not significantly different from the most resistant line.

Line	Lsmean	SE
K19832*	3.17	0.21
K19817*	3.37	0.21
K20745*	3.52	0.21
K20717*	3.55	0.21
K20730*	3.57	0.21
K20734*	3.75	0.21
K20743	3.87	0.21
K19830	3.91	0.21
K19831	4.01	0.21
K20721	4.06	0.22
Clouseau	4.11	0.21
K20221	4.13	0.21
Denali	4.15	0.21
K20715	4.18	0.21
Coho	4.23	0.21
K19610	4.25	0.21
Snowdon	4.26	0.21
K20742	4.27	0.21
K20239	4.33	0.21
K20217	4.39	0.21
K20212	4.47	0.21
K20732	4.54	0.21

Table 2.12 (cont'd)

K20744	4.74	0.21
Red Cedar	5.06	0.21
LSD	0.59	-

Table 2.13: Least squares means, least significant difference, and standard error by line for root rot rating of the Natural infection trial 2021 analysis. Asterisk (*) indicates lines that are not significantly different from the most resistant line.

Line	Lsmean	SE
K19832*	3.45	0.28
K20730*	3.70	0.28
K20743*	3.81	0.28
K21904*	3.88	0.28
K16136*	3.90	0.28
K20720*	3.90	0.28
K17704*	3.92	0.28
K20728*	3.93	0.28
K20734*	3.99	0.28
K17703*	4.01	0.28
K21909*	4.03	0.28
K21902*	4.04	0.28
K21913*	4.05	0.28
K19831*	4.07	0.28
K21901*	4.08	0.28
K20745*	4.08	0.28
K21906*	4.09	0.28
K20717*	4.10	0.28
Beluga*	4.14	0.28
K20235*	4.15	0.28
K20239*	4.15	0.28
I90013*	4.15	0.28

Table 2.13 (cont'd)

K20721*	4.16	0.28
Denali*	4.17	0.28
K19830*	4.17	0.28
Coho*	4.17	0.28
K20217	4.21	0.28
K17702	4.23	0.28
K20744	4.23	0.28
K21905	4.27	0.28
K19817	4.27	0.28
K19610	4.30	0.28
K20715	4.38	0.28
Snowdon	4.41	0.28
Clouseau	4.44	0.28
K20210	4.45	0.28
K21908	4.46	0.28
K20712	4.47	0.28
Red Hawk	4.49	0.28
K21912	4.50	0.28
K21907	4.51	0.28
Montcalm	4.51	0.28
Red Cedar	4.53	0.28
K20212	4.55	0.28
K20732	4.56	0.28
K19120	4.57	0.28

Table 2.13 (cont'd)

K20749	4.57	0.28
K19608	4.59	0.28
K20742	4.64	0.28
K21910	4.65	0.28
K20221	4.65	0.28
ND Whitetail	4.75	0.28
K18312	4.76	0.28
K21903	4.82	0.28
K21911	4.90	0.28
K20234	5.41	0.28
LSD	0.75	-

Table 2.14: Least squares means, least significant difference, and standard error by line for root rot rating of the Natural infection trial 2022 analysis. Asterisk (*) indicates lines that are not significantly different from the most resistant line.

Line	Lsmean	SE
K19817*	2.47	0.35
K19832*	2.89	0.35
K20745*	2.95	0.35
K20717*	3.02	0.36
K20730*	3.44	0.35
K20734	3.51	0.35
K20221	3.62	0.35
K19830	3.66	0.35
Clouseau	3.80	0.35
K20742	3.88	0.35
K19831	3.94	0.35
K20721	3.94	0.37
K20743	3.94	0.36
K20715	3.99	0.35
Snowdon	4.12	0.35
Denali	4.14	0.35
K19610	4.20	0.35
Coho	4.28	0.35
K20212	4.39	0.35
K20239	4.51	0.35
K20732	4.52	0.35
K20217	4.56	0.35

Table 2.14 (cont'd)

K20744	5.25	0.35
Red Cedar	5.58	0.35
LSD	0.97	-

Table 2.15: Least squares means, least significant difference, and standard error by line for root rot rating of the Middle-American *Rhizoctonia solani* trial combined analysis. Asterisk (*) indicates lines that are not significantly different from the most resistant line.

Line	Lsmean	SE
B19344*	3.31	1.47
N20395*	3.44	1.47
N19246*	3.50	1.47
Spectre*	3.63	1.47
N20404*	3.69	1.47
N19226*	4.38	1.47
Merlin*	4.75	1.47
B19309*	5.12	1.47
Ruby*	5.75	1.47
P19103*	6.56	1.47
Blizzard	7.13	1.47
N20388	7.19	1.47
B20591	7.31	1.47
HMS Bounty	7.38	1.47
Valiant	7.50	1.47
Zorro	7.63	1.47
Armada	8.19	1.47
Nimbus	8.19	1.47
G19613	8.25	1.47
P19713	8.63	1.47
R20627	8.69	1.47
Caldera	8.75	1.47

Table 2.15 (cont'd)

R20667	9.44	1.47
R20659	9.56	1.47
Zenith	9.69	1.47
B20547	9.75	1.47
Medalist	9.88	1.47
Black Bear	10.94	1.47
Black Beard	11.31	1.47
Black Tails	11.38	1.47
Viper	12.25	1.47
LSD	3.55	-

Table 2.16: Least squares means, least significant difference, and standard error by line for root rot rating of the Middle-American *Rhizoctonia solani* trial 2021 analysis. Asterisk (*) indicates lines that are not significantly different from the most resistant line.

Line	Lsmean	SE
N19226*	2.00	1.66
N20404*	3.12	1.66
G19611*	3.37	1.66
P19103*	3.62	1.66
N20395*	3.87	1.66
B18504*	4.00	1.66
B19344*	4.12	1.66
N20388*	4.37	1.66
N19246*	4.75	1.66
G19613*	5.00	1.66
P19707*	5.12	1.66
Blizzard*	5.25	1.66
N19285*	5.50	1.66
Spectre	6.00	1.66
S08418	6.25	1.66
B20602	6.37	1.66
R20627	6.37	1.66
Armada	6.40	1.66
B19309	6.50	1.66
B19332	6.75	1.66
B19330	7.12	1.66
R20683	7.25	1.66

Table 2.16 (cont'd)

B20591	7.37	1.66
Caldera	7.50	1.66
Merlin	7.50	1.66
N19239	7.62	1.66
S18904	7.75	1.66
Valiant	7.75	1.66
HMS Bounty	8.00	1.66
B20597	8.12	1.66
R20652	8.25	1.66
Ruby	8.25	1.66
R20659	8.37	1.66
Nimbus	8.62	1.66
B20547	8.75	1.66
Zorro	8.75	1.66
P16901	9.12	1.66
B20549	9.25	1.66
P19713	9.25	1.66
R12844	9.87	1.66
R20667	9.87	1.66
Black Tails	10.25	1.66
Black Bear	10.87	1.66
Black Beard	11.87	1.66
Zenith	11.87	1.66
R20639	12.00	1.66

Table 2.16 (cont'd)

Medalist	12.25	1.66
Viper	13.50	1.66
LSD	3.96	-

Table 2.17: Least squares means, least significant difference, and standard error by line for root rot rating of the Middle-American *Rhizoctonia solani* trial 2022 analysis. Asterisk (*) indicates lines that are not significantly different from the most resistant line.

Line	Lsmean	SE
Spectre*	1.25	2.54
G21811*	1.75	2.54
Merlin*	2.00	2.54
N19246*	2.25	2.54
B19344*	2.5	2.54
N20395*	3.00	2.54
Ruby*	3.25	2.54
B19309*	3.75	2.54
N18103*	4.25	2.54
N20404*	4.25	2.54
N21511*	5.25	2.54
B20599*	5.50	2.54
Liberty*	5.75	2.54
N21525*	6.25	2.54
Zorro*	6.50	2.54
HMS Bounty*	6.75	2.54
N19226*	6.75	2.54
Charro*	7.00	2.54
B20591*	7.25	2.54
Valiant*	7.25	2.54
Coral	7.50	2.54

Table 2.17 (cont'd)

Medalist	7.50	2.54
Zenith	7.50	2.54
Nimbus	7.75	2.54
P19713	8.00	2.54
Eiger	8.25	2.54
Cayenne	8.50	2.54
Rosetta	8.75	2.54
Blizzard	9.00	2.54
R20667	9.00	2.54
B21714	9.50	2.54
P19103	9.50	2.54
Adams	9.75	2.54
Armada	10.00	2.54
Caldera	10.00	2.54
N20388	10.00	2.54
B20547	10.75	2.54
Black Beard	10.75	2.54
R20659	10.75	2.54
B21710	11.00	2.54
Black Bear	11.00	2.54
R20627	11.00	2.54
Viper	11.00	2.54
G19613	11.50	2.54
R20669	12.25	2.54

Table 2.17 (cont'd)

Black Tails	12.50	2.54
B21708	14.75	2.54
B20536	16.25	2.54
LSD	6.22	-

Table 2.18: Correlation of least squares means for root rot rating between trials.

Pearson's Correlation (r)	ADP and GH	KDB and GH	KDB and Natural
Combined	0.20	0.65*	0.22
2021	0.04	0.37	0.10
2022	0.51	0.71**	0.24

Values marked with *, **, and *** were significant at the 0.1, 0.05, and 0.01 level, respectively.

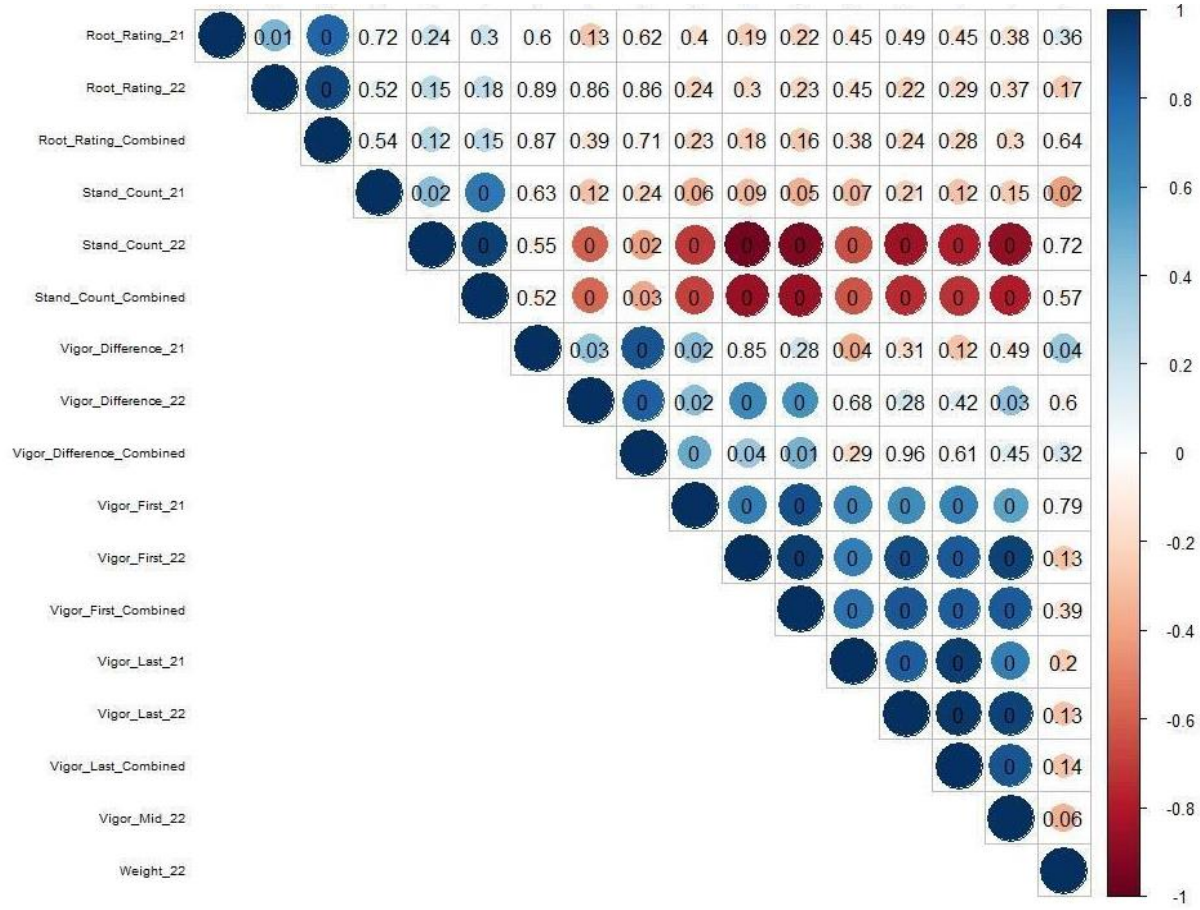

Figure 2.16: Correlation of root rot rating, stand count, vigor, and weight for the KDB trial. The scale on the right indicates correlation where the values represent the significance of correlation.

Table 2.19: Correlation of root rot rating, stand count, vigor, and weight for the KDB trial.

	Stand Count	First Vigor Measurement	Vigor Difference Measurement	Last Vigor Measurement	2022 Fresh Weight (g)
Correlation with Root Rot Rating	0.28	-0.27	-0.23	-0.17	-0.15

Values marked with *, **, and *** were significant at the 0.1, 0.05, and 0.01 level, respectively.

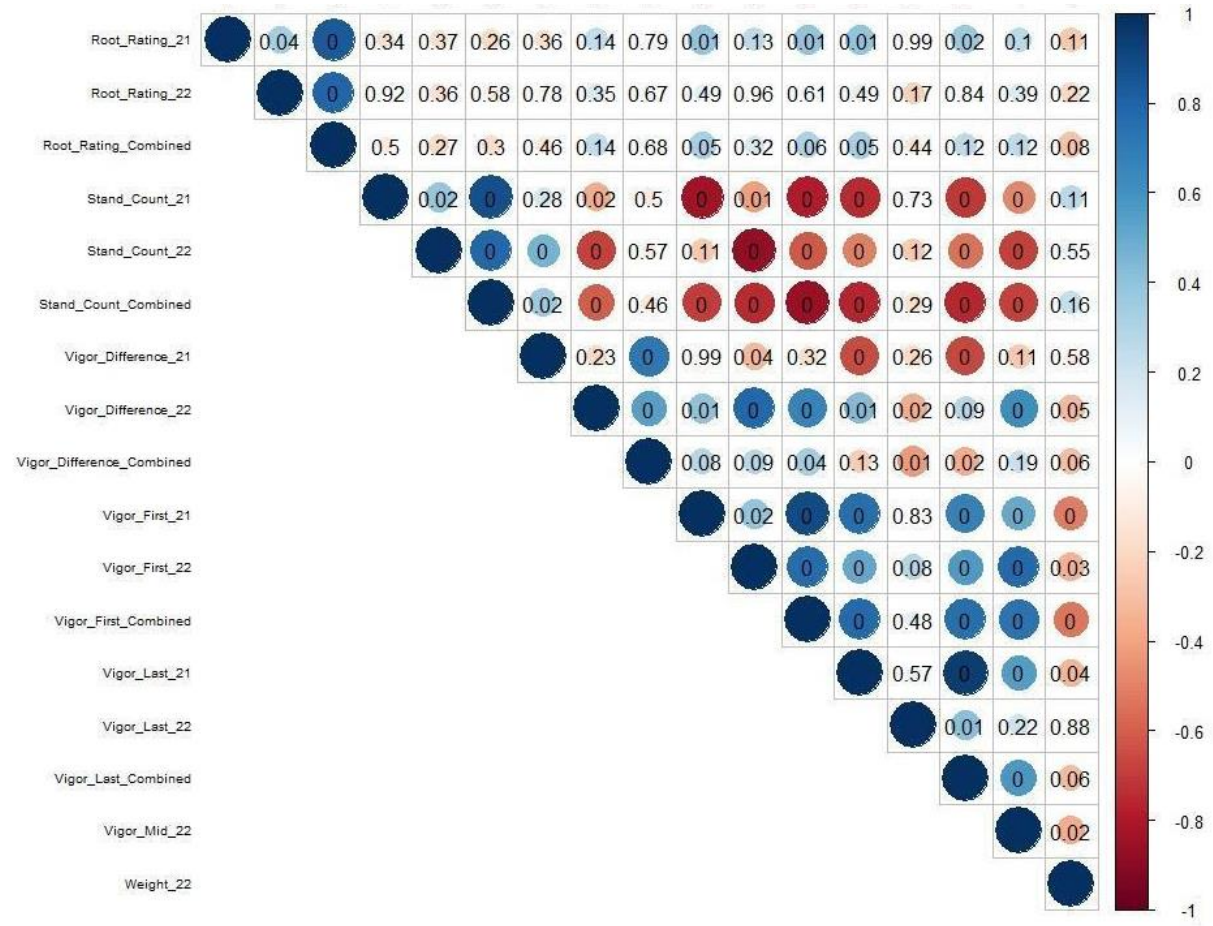

Figure 2.17: Correlation of root rot rating, stand count, vigor, and weight for the ADP trial. The scale on the right indicates correlation where the values represent the significance of correlation.

Table 2.20: Correlation of root rot rating, stand count, vigor, and weight for the ADP trial.

	Stand Count	First Vigor Measurement	Vigor Difference Measurement	Last Vigor Measurement	2022 Fresh Weight (g)
Correlation with Root Rot Rating	-0.17	0.3*	0.07	0.25	-0.29*

Values marked with *, **, and *** were significant at the 0.1, 0.05, and 0.01 level, respectively.

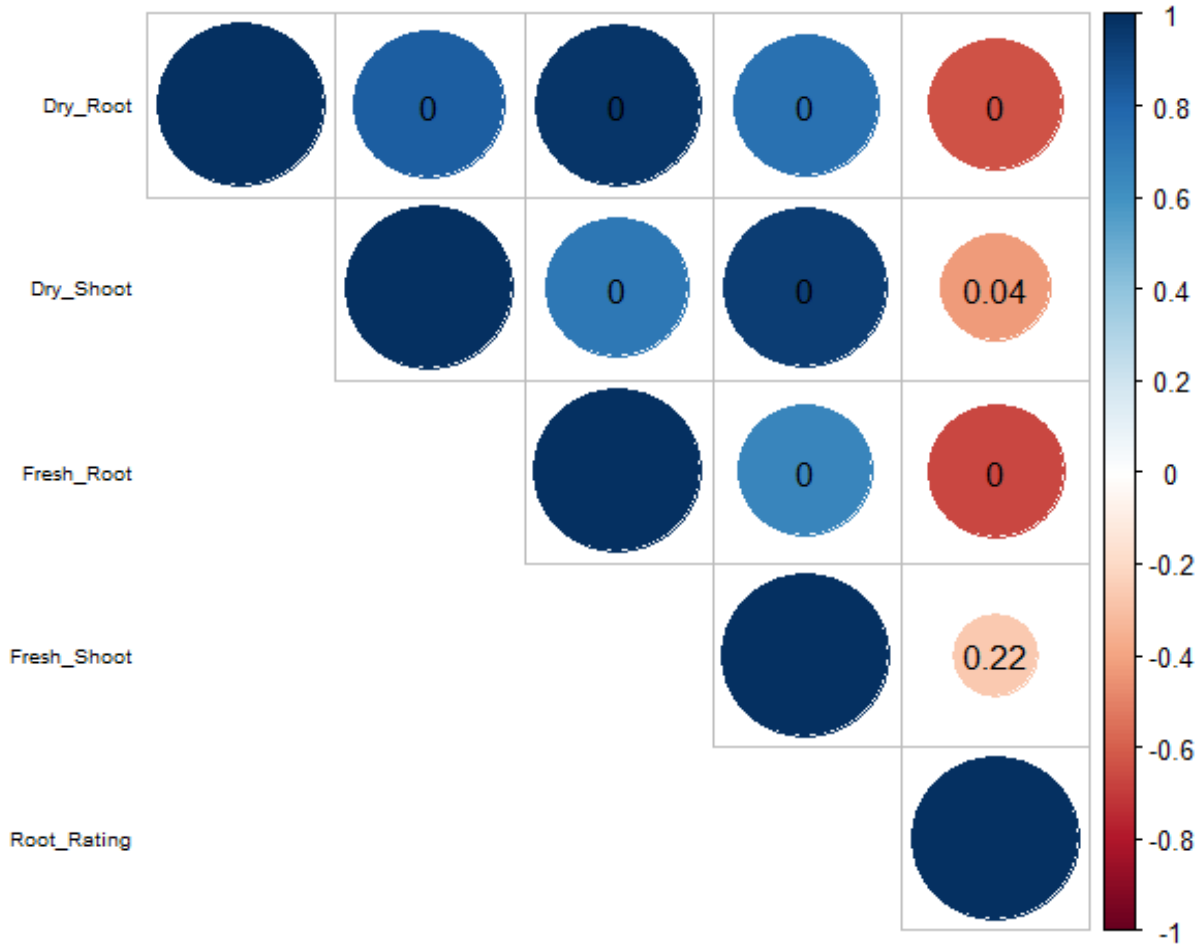

Figure 2.18: Correlation of root rot rating, root and shoot weights for the GH trial. The scale on the right indicates correlation where the values represent the significance of correlation.

Table 2.21: Correlation of root rot rating, root and shoot weight for the GH infection trial.

	Fresh Root Weight (g)	Fresh Shoot Weight (g)	Dry Root Weight (g)	Dry Shoot Weight (g)
Correlation with Root Rot Rating	-0.67***	-0.27(P=0.22)	-0.64***	-0.43**

Values marked with *, **, and *** were significant at the 0.1, 0.05, and 0.01 level, respectively.

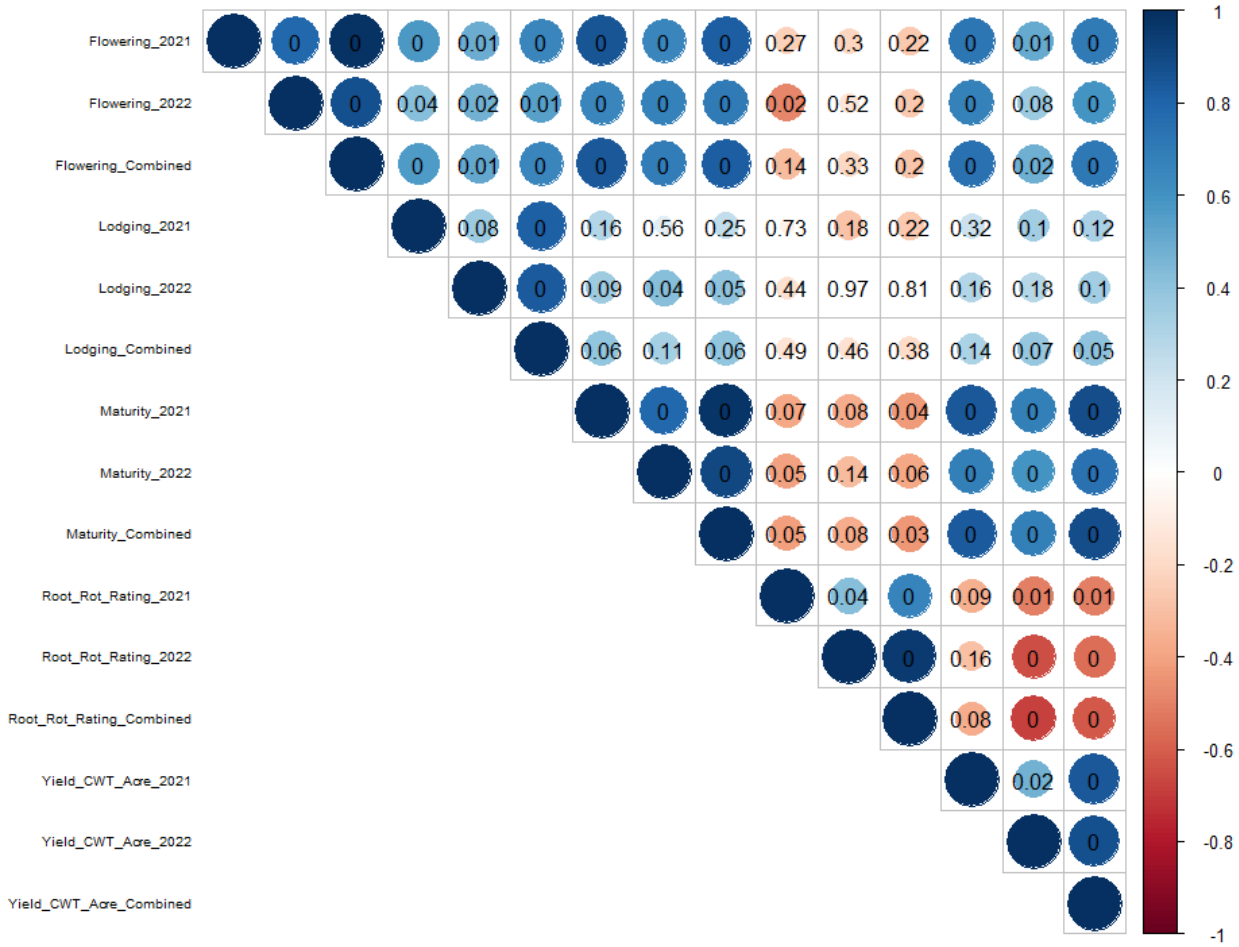

Figure 2.19: Correlation of root rot rating, yield, flowering date, lodging score, and maturity date for the Natural infection trial. The scale on the right indicates correlation where the values represent the significance of correlation.

Table 2.22: Correlation of root rot rating, yield, flowering date, lodging score, and maturity date for the Natural infection trial.

	Yield (CWT Acre)	Flowering Date	Lodging Score	Maturity Date
Correlation with Root Rot Rating	-0.62***	-0.27	-0.19	-0.43***

Values marked with *, **, and *** were significant at the 0.1, 0.05, and 0.01 level, respectively.

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