Aportes al control de roya estriada de trigo: variabilidad del patógeno y resistencia en el hospedero

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Resumen

La roya estriada del trigo, causada por *Puccinia striiformis* f. sp. *tritici* (*Pst*), ha experimentado una expansión global desde el año 2000, con la aparición de razas agresivas que provocaron epidemias en regiones donde antes no era importante, como Uruguay. Desde 2017, estas razas han causado epidemias severas, en Argentina y Uruguay. La resistencia genética es considerada la estrategia de manejo más sostenible por su menor impacto económico y ambiental al reducir el uso de fungicidas. Este proyecto abordó dos áreas clave: la variabilidad del patógeno y la base genética de la resistencia. El análisis de muestras de *Pst* colectadas en Uruguay entre 2017 y 2021 permitió asignarlas a tres grupos genéticos descritos: *PstS7*, *PstS10* y *PstS13*, de los cuales *PstS13* fue el más prevalente. Dentro de *PstS13* se identificaron razas con nuevos perfiles de virulencia sobre genes anteriormente efectivos, lo que indica evolución local del patógeno. Se identificaron ocho regiones genómicas (QTL) asociadas con resistencia parcial (RP) a campo. Una de estas QTL, localizada en el cromosoma 5BS, podría ser novedosa. Las restantes requieren validación para determinar si coinciden con regiones ya reportadas. Todas las QTL detectadas confieren RP con efecto aditivo, útiles para programas de mejoramiento. Los modelos de predicción genómica mostraron precisiones de predicción promedio de 0,7, lo que destaca su potencial para acelerar la selección de líneas resistentes. En conjunto, estos resultados aportan conocimiento clave sobre la evolución del patógeno en Uruguay y brindan herramientas genéticas valiosas para el desarrollo de variedades con resistencia duradera, adaptadas a los sistemas productivos locales.

Palabras clave: *Puccinia striiformis,* mapeo asociativo, predicción genómica, roya amarilla, caracterización genética

Contributions to the control of stripe rust of wheat: pathogen variability and host resistance

Summary

Wheat yellow rust, caused by *Puccinia striiformis* f. sp. *tritici* (*Pst*), has undergone global expansion since 2000, with the emergence of aggressive races that have caused epidemics in regions where it was previously unimportant, such as Uruguay. Since 2017, these races have caused severe epidemics in Argentina and Uruguay. Genetic resistance is considered the most sustainable management strategy due to its lower economic and environmental impact by reducing fungicide use. This project addressed two key areas: pathogen variability and the genetic basis of resistance. Analysis of *Pst* samples collected in Uruguay between 2017 and 2021 allowed their assignment to three described genetic groups: *PstS7*, *PstS10* and *PstS13*, with *PstS13* being the most prevalent. Within *PstS13*, races with new virulence profiles to previously effective genes were identified, indicating local evolution of the pathogen. Eight genomic regions (QTL) associated with adult plant resistance (APR) in the field were identified. One of these QTL, located on chromosome 5BS, may be novel. The remaining ones require validation to determine whether they correspond to already reported regions. All detected QTLs confer APR with additive effects, making them useful for breeding programs. Genomic prediction models achieved an average prediction ability of 0.7, highlighting their potential for accelerating the selection of resistant lines. Together, these results provide key knowledge about pathogen evolution in Uruguay and offer valuable genetic tools for developing varieties with durable resistance, adapted to local production systems.

Keywords: *Puccinia striiformis*, genome-wide association study (GWAS), genomic prediction (GP), genotypic characterization, yellow (stripe) rust

1. Introducción

1.1. El trigo dentro de los principales cultivos base de la alimentación humana

El trigo (*Triticum aestivum* L.), es junto al arroz y al maíz, uno de los tres cultivos base más importantes de la alimentación humana, no solo como fuente de energía, sino también por su aporte de componentes beneficiosos para la salud como proteínas, vitaminas y fibra (Shewry y Hey, 2015). En el 2019, 240 millones de hectáreas fueron destinadas en el mundo para su cultivo, con una producción de casi 900 millones de toneladas (Food and Agriculture Organization [FAO], 2019). En Uruguay, el trigo es el principal cultivo de invierno con 254.900 hectáreas sembradas y un rendimiento promedio de 4145 kg/ha entre las zafras del 2019-2020 al 2023-2024, lo que lo posiciona como segundo cultivo en área sembrada después de la soja (Dirección de Estadísticas Agropecuarias [DIEA], 2024).

1.2. Desafíos para la producción actual de trigo

En los últimos años, la producción mundial de trigo se ha visto amenazada por el surgimiento de nuevas razas de patógenos más agresivas (Hovmøller et al., 2008) y adaptadas a nuevos ambientes (Milus et al., 2006), entre las que destaca *Puccinia striiformis* Westend. f. sp. *tritici* Erikss. (*Pst*), el agente causal de la roya amarilla o roya estriada (RE) del trigo (Hovmøller et al., 2011; Muleta et al., 2017; Sørensen et al., 2014). Las pérdidas de rendimiento causadas por RE pueden ser muy elevadas (hasta 100 %) en cultivares susceptibles en condiciones extremas, cuando la enfermedad se presenta temprano y las condiciones climáticas son favorables para su desarrollo (Chen y Kang, 2017; Chen, 2005; Roelfs et al., 1992). En Europa, las nuevas razas de *Pst* afectaron a la mayoría de los genotipos con pérdidas superiores al 50 % del rendimiento (Vergara-Diaz et al., 2015).

1.3. La roya estriada del trigo resurge y se posiciona como la principal enfermedad foliar del trigo

La RE, también conocida como *roya amarilla*, produce síntomas en forma de pústulas de color amarillento en orientación lineal (o estrías) sobre hojas y ocasionalmente sobre espigas. Al inicio de la infección, en estados tempranos de desarrollo del trigo, puede confundirse con roya de la hoja, ya que las pústulas no se disponen en estrías. La RE es favorecida por temperaturas relativamente bajas de 10-

15 ºC, por lo que puede presentarse temprano en el desarrollo del cultivo y causar pérdidas de rendimiento de grano totales en cultivares susceptibles cuando las condiciones climáticas son favorables para su desarrollo (Roelfs et al., 1992). La población del patógeno está compuesta por razas que tienen distintos perfiles de avirulencia/virulencia sobre materiales de trigo. En regiones donde la enfermedad es endémica, la aparición e incremento en frecuencia de nuevas razas virulentas de *Pst* tiene como consecuencia que cultivares inicialmente resistentes muestren niveles crecientes de infección, lo que determina una corta duración de la resistencia (Stubbs, 1985).

La RE fue observada y descrita por primera vez en Argentina y Uruguay en 1929 (Rudolf y Job, 1931). Durante 1929 y 1930 causó epidemias generalizadas y muy severas en la mayor parte de la región del Cono Sur, con pérdidas de rendimiento extremadamente altas (Boerger, 1934; Vallega, 1938). Desde su primera detección hasta el año 2016, la RE se presentó de forma esporádica, aunque raramente alcanzó niveles epidémicos en Argentina y Uruguay (Germán et al., 2007, 2018; Germán y Caffarel, 1999).

A partir del año 2000, en poco tiempo ocurrió en el mundo una dispersión muy rápida de razas similares de *Pst* (Hovmøller et al., 2008). Estas razas son más agresivas que las razas conocidas en Europa y Estados Unidos de América (EUA) hasta el año 2000, pudiendo producir hasta dos a tres veces más esporas por día, y han causado epidemias en regiones donde la RE no era una enfermedad importante. Las razas presentes en la región sur y central de EUA hasta Canadá a partir del año 2000 tienen mayor adaptación a temperaturas de 18-20 ºC (Milus et al., 2006).

A partir de 2017 la RE ha causado epidemias generalizadas en Argentina y Uruguay (Germán et al., 2018, 2021; Silva et al., 2023), y es hoy la enfermedad foliar mas prevalente. El cultivar más susceptible a RE incluido en los ensayos de la Evaluación Nacional de Cultivares (ENC) INIA/INASE, afectado solo por esta enfermedad, presentó pérdidas de rendimiento de grano entre 71 % y 82 % en dos ensayos instalados en La Estanzuela y uno en Dolores (Instituto Nacional de Investigación Agropecuaria [INIA] e Intituto Nacional de Semillas [INASE], 2018). Se detectaron cambios en el comportamiento de varios cultivares respecto a su caracterización anterior, probablemente asociados a la presencia de la(s) nueva(s) raza(s) del patógeno. En 2020 más del 50 % del área de trigo fue sembrada con cultivares de comportamiento susceptible o moderadamente susceptible frente a RE (Dirección de Estadísticas Agropecuarias [DIEA], 2020; Germán et al., 2021).

1.4. Roya estriada, población del patógeno

Pst es un hongo biotrófico, lo que significa que solo puede sobrevivir en tejido vivo del hospedero. Se propaga principalmente a través de urediniosporas (esporas asexuales) transportadas por el viento, capaces de recorrer distancias de hasta 2000 km (Roelfs et al., 1992).

El inóculo inicial necesario para el desarrollo de la enfermedad puede ser de origen local (endógeno), proveniente de plantas voluntarias que sobreviven durante el verano, o bien ser transportado por el viento y depositado por la lluvia (exógeno). Las condiciones óptimas para el establecimiento de la roya estriada incluyen temperaturas frescas (8-15 °C), la presencia de agua libre en el follaje (mínimo seis horas), la cual puede provenir del rocío o de lluvias ligeras, y días preferentemente secos y soleados. Estas condiciones favorecen la aparición temprana de la enfermedad en el ciclo del cultivo (Roelf et al., 1992).

La población de *Pst* está compuesta por razas con distintos perfiles de avirulencia/virulencia sobre materiales de trigo. En la patología de royas de cereales, el término *raza* se define por el patrón de interacciones compatibles e incompatibles entre hospedero y patógeno. La interpretación genética de estos datos depende de cuán bien se hayan identificado los genes de resistencia (*R-genes*) presentes en las líneas diferenciales del hospedero, así como de la resolución de nuevas especificidades de resistencia mediante la exposición de estas líneas a un amplio espectro de aislados del patógeno, procedentes de diversas regiones geográficas y con trayectorias evolutivas diferentes (Hovmøller et al., 2017).

El fenotipo del patógeno se describe como *virulento* cuando ocurre una interacción compatible (tipo de infección alto) con las líneas diferenciales del hospedero y como *avirulento* cuando la interacción es incompatible (tipo de infección bajo) (Hovmøller et al., 2017). En regiones donde la enfermedad es endémica, la aparición y el incremento en la frecuencia de nuevas razas de *Pst* pueden hacer que los cultivares inicialmente resistentes se vuelvan susceptibles y presenten niveles crecientes de infección (Stubbs, 1985).

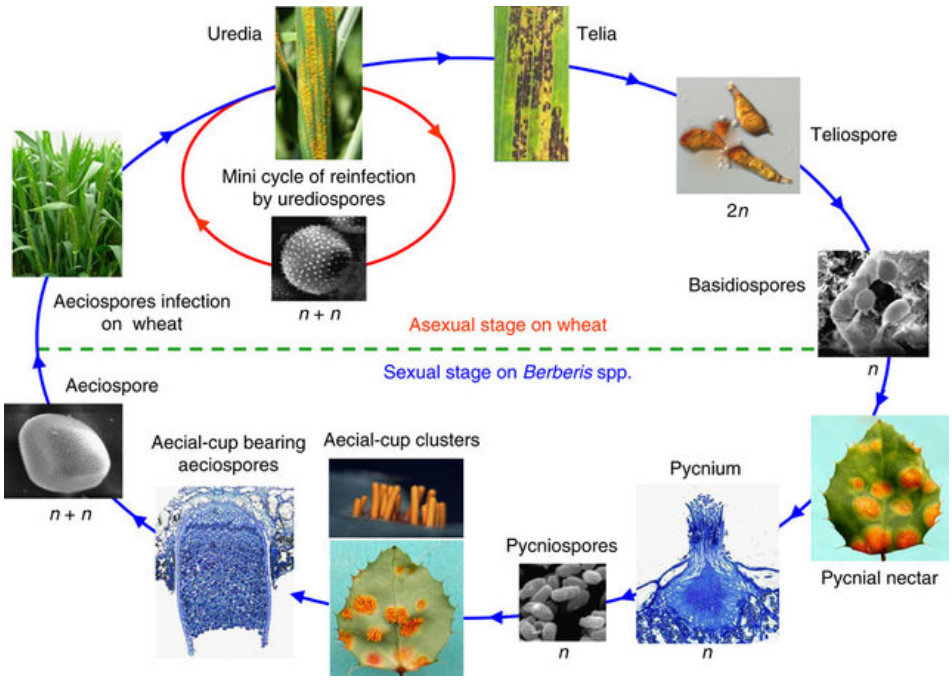
En el estudio de la diversidad genética de *Pst*, un linaje o grupo genético se define como un conjunto de aislados con origen evolutivo común, que comparten características genéticas detectables mediante herramientas moleculares como microsatélites o SNPs. Estos linajes pueden identificarse a través de análisis de estructura poblacional y se

asocian frecuentemente con regiones geográficas, perfiles de virulencia o eventos de introducción recientes (Ali et al., 2014; Hubbard et al., 2015; Rodríguez-Algaba et al., 2016).

En la mayoría de las regiones del mundo, el ciclo biológico de *Pst* (figura 1) consiste en generaciones continuas de urediniosporas sobre su principal hospedero primario, el trigo, (aunque también tiene la capacidad de infectar hospederos secundarios como centeno y triticale), lo que determina una estructura poblacional predominantemente clonal de la población del patógeno (Ali et al., 2014; Hovmøller et al., 2011; Wellings y McIntosh, 1990). El ciclo sexual (producción de aeciosporas sobre el hospedante alterno *Berberis vulgaris* demostrado por Jin et al. (2010) es raro en la mayoría de las regiones productoras de trigo, pero ocurre en el centro de origen de *Pst*, ubicado en la región del Himalaya y áreas cercanas. En esas condiciones el patógeno muestra una estructura poblacional recombinante y una alta diversidad donde se generan nuevas combinaciones de genes de virulencia y agresividad en el patógeno. En Uruguay y la región no se ha documentado la presencia del ciclo sexual. En su lugar, *Pst* ha adoptado una estrategia de reproducción asexual que le permite mantener los genes de virulencia en bloques y generar nuevas combinaciones mediante mutaciones genéticas.

Figura 1

Ciclo de vida de *Puccinia striiformis* f. sp. *tritici* (*Pst*).



Nota. Tomado de Zheng et al. (2013).

En estudios realizados en Dinamarca de 36 muestras de *Pst* recolectadas durante 2017 en Argentina, se identificaron tres razas pertenecientes a distintos linajes o grupos genéticos (*PstS13* en treinta aislamientos, *PstS14* en cinco aislamientos, *PstS7* o raza warrior en un aislamiento) que coincidieron con razas que causaron epidemias recientes en Europa y norte de África (Hovmøller et al., 2018). En 2018 se detectó el grupo genético *PstS13* en veintiocho muestras de Argentina y dieciocho de Chile y el *PstS7* en tres muestras de Argentina. A partir de 2017, en la región del Cono Sur han predominado ampliamente las razas pertenecientes a los linajes *Pst13* (Hovmøller et al., 2020, 2021).

1.5. Perspectivas para las epidemias de roya estriada en la región

La primera detección de RE en Uruguay en 2018 fue el 16 de agosto, un mes antes del primer registro en 2017; provocó el desarrollo de una epidemia más severa y daños potenciales mayores, situación que se mantuvo en las zafras 2019, 2020 y 2021 con inicios de las epidemias en los últimos días de julio. La aparición temprana de RE en Argentina y Uruguay indica que la sobrevivencia del patógeno durante la estación crítica del verano ocurre en las proximidades o en el área de producción del cultivo, lo que puede estar asociado a la presencia de las nuevas razas. Por este motivo, la enfermedad probablemente tendrá frecuencia de aparición y severidad mayores que antes de 2017. Dado que en las últimas décadas la RE era una enfermedad de importancia marginal en nuestro país, la única medida utilizada en el Programa de Mejoramiento Genético de Trigo (PMGT) de INIA cuando se presentaban infecciones, generalmente tardías, era eliminar las plantas o líneas con alta susceptibilidad, pero no se utilizaban fuentes de resistencia específicas en cruzamientos ni se seleccionaba por resistencia en el germoplasma de mejoramiento. A partir de 2017 se dispone de información fenotípica del comportamiento frente a RE del germoplasma utilizado por el PMGT, pero no se han realizado estudios sobre la base genética de resistencia a RE, por lo que partimos de una situación en la que disponemos de poca información genotípica sobre esta característica.

1.6. Resistencia genética como la mejor estrategia de control para la roya estriada

La RE fue la primera enfermedad en la que se demostró que la resistencia del genotipo hospedero se heredaba de manera mendeliana (Biffen, 1905). La resistencia a RE puede clasificarse en dos grandes categorías: resistencia de plántula o de todas las etapas (del inglés ASR, *all-stage resistance*) y resistencia parcial (RP). La ASR se expresa a lo largo de todo el ciclo del cultivo y está asociada principalmente a genes de efecto

mayor, dominantes, de herencia cualitativa, los cuales siguen la relación gen por gen descrita por Flor (1955): cada gen de resistencia en el hospedero interactúa con un gen de avirulencia complementario en el patógeno. Cuando el hospedero posee el gen resistencia, y el patógeno, el gen complementario de avirulencia, la interacción hospedero-patógeno no es compatible. En los otros casos, cuando el hospedero no posee el gen de resistencia y cuando el patógeno posee el gen complementario de virulencia, la interacción es compatible. Un genotipo de trigo con múltiples genes de resistencia es susceptible si el patógeno combina virulencia sobre todos los genes presentes en el hospedero (Person, 1959).

La ASR, caracterizada por una respuesta de hipersensibilidad (Ayliffe et al., 2008), es típicamente específica para la(s) raza(s) del patógeno, y ha demostrado ser poco durable, ya que nuevas razas virulentas pueden superar su eficacia en poco tiempo.

En contraste, la RP se expresa en estadios posplántula y está controlada por genes menores de efecto aditivo, con herencia cuantitativa. Esta resistencia no detiene completamente la infección, pero reduce el progreso de la enfermedad mediante un desarrollo lento del patógeno (enroyamiento lento), caracterizado por períodos de latencia prolongados, menor número y tamaño de uredinias y baja producción de esporas. La RP es generalmente no específica para una determinada raza y se considera más durable, ya que no se ha registrado adaptación del patógeno a este tipo de resistencia. Para alcanzar niveles cercanos a la inmunidad, se requiere la acumulación de múltiples genes RP (4 a 6) (Bhavani et al., 2011; R. Liu et al., 2020; Singh et al., 2000).

Hasta la fecha, se han catalogado 81 genes de resistencia a RE (*Yr*), distribuidos en dieciséis de los veintiún cromosomas del trigo (MASWheat, 2025; McIntosh et al., 2018; Tong et al., 2024), muchos de ellos provenientes de especies emparentadas. La mayoría corresponden a genes ASR, que expresan hipersensibilidad y confieren resistencia eficaz pero temporal, por ser genes específicos a determinada raza. La combinación de múltiples genes ASR puede extender la durabilidad, ya que la aparición de razas virulentas requeriría mutaciones simultáneas en varios loci del patógeno.

Por otro lado, los genes RP no solo proporcionan resistencia duradera a RE, sino que algunos también confieren resistencia pleiotrópica a otras enfermedades foliares importantes, como roya del tallo (*Sr*), roya de la hoja (*Lr*) y oídio (*Pm*). Ejemplos bien conocidos de estos genes pleiotrópicos incluyen *Yr18*/*Lr34*/*Sr57*/*Pm38*, *Yr29*/*Lr46*/*Sr58*/*Pm39*, *Yr30*/*Lr27*/*Sr2* y *Yr46*/*Lr67*/*Sr55*/*Pm46* (Dyck et al., 1966; Hiebert et al., 2010; Mago et al., 2011; Singh et al., 1998). Su acumulación en los

cultivares de trigo representa una estrategia eficaz y sostenible para el control de RE y otras enfermedades.

En el caso de RP, los fenotipos asociados son difíciles de identificar y este tipo de resistencia es enmascarada por la presencia de genes ASR efectivos, por lo que el uso de marcadores moleculares puede acelerar su selección durante el proceso de mejoramiento. Se han identificado marcadores moleculares ligados a genes específicos de resistencia a las royas del trigo, los cuales pueden ser utilizados para la implementación de selección asistida en los programas de mejoramiento. En el caso de RE, se cuenta con marcadores moleculares para varios de los genes ASR y para los genes de RP (MASWheat, 2025).

En general, la RP a RE está asociada a RP a roya de la hoja (Singh, 1992), por lo que se presume que el uso creciente de RP durable a roya de la hoja también incrementará el nivel de RP a RE (Germán et al., 2007). Sin embargo, no se conoce la contribución relativa de los genes de RP a RE en nuestra región.

1.7. Principales herramientas para el desarrollo de variedades de trigo resistentes

El uso de marcadores moleculares ha demostrado ser valioso en el mejoramiento por resistencia a enfermedades (Bernardo, 2008), al permitir una mayor eficiencia en el proceso de mejoramiento, ya que evita la redundancia de utilizar materiales portadores de los mismos genes y facilita la identificación de nuevas fuentes para utilizar para ampliar la diversidad genética.

Las herramientas biotecnológicas permiten el estudio de la resistencia basada en genes que determinan ASR y RP mediante diferentes estrategias de análisis genético molecular. El mapeo de regiones genómicas asociadas a caracteres de variación cuantitativa o análisis de locus cuantitativo (QTL), basado en polimorfismos entre los padres de un cruzamiento, es una herramienta de análisis estadístico que vincula información fenotípica y genotípica para explicar la base genética de la variación en rasgos complejos. El análisis de QTL basado en polimorfismos en poblaciones biparentales ha sido ampliamente utilizado para el estudio genético de la resistencia a las royas del trigo (Prins et al., 2011; Rosewarne et al., 2008; Singh y Herrera-Foessel, 2013).

La técnica de mapeo asociativo (GWAS, del inglés *genome-wide association study*) es una estrategia de análisis que se basa en el desequilibrio de ligamiento presente en una población y estudia la asociación entre marcadores moleculares y características fenotípicas de interés. Una asociación estadísticamente significativa indica que el marcador está en desequilibrio de ligamiento con una región del genoma involucrada en

la expresión del fenotipo de interés (QTL, del inglés *quantitative trait loci*); el desequilibrio puede deberse a la próximidad fisica entre marcador y QTL (Schmid y Bennewitz, 2017).

La identificación de QTL mediante GWAS requiere de una población de genotipos diversos fenotipados para la variable de interés, información de marcadores moleculares y el uso de herramientas estadísticas, como modelos mixtos, que permiten considerar la estructura genética de la población para eliminar falsos positivos o asociaciones significativas no debidas a la existencia de un ligamiento físico entre marcadores y QTL (Pritchard et al., 2000; Zhu y Yu, 2009). GWAS se ha utilizado con éxito en trigo (Crossa et al., 2007; Maccaferri et al., 2015), que es una especie de genoma complejo. El GWAS tiene poder limitado para detectar variantes alélicas raras, presentes en baja proporción (Brachi et al., 2011; Wallace et al., 2014; Zuk et al., 2014) o loci con múltiples variantes alélicas (Zhang et al., 2012). A pesar de estas limitaciones, GWAS ha sido utilizado exitosamente para mapeo de QTL asociados a resistencia a RE (Maccaferri et al., 2015; Rosewarne et al., 2013; F. -P. Yuan et al., 2018). Mediante la utilización de ambas metodologías (mapeo en poblaciones biparentales y GWAS), durante los últimos dieciocho años se identificaron más de 160 QTL en 49 regiones distribuidas en los veintiún cromosomas de trigo.

La dificultad de detectar variantes presentes en baja proporción en una determinada población se debe a la necesidad de controlar la tasa de falsos positivos, al eliminar asociaciones marcador y QTL que no se deben a un ligamiento físico entre ambos (Brachi et al., 2010; Wallace et al., 2014; Zuk et al., 2014). Originalmente, la identificación de QTL se realizaba con el objetivo de obtener marcadores para selección asistida por marcadores (SAM) (Dekkers, 2004). La SAM comprende la selección de líneas por la presencia de marcadores asociados a QTL de efecto mayor sobre el fenotipo; no se utilizan marcadores que no están significativamente asociados a una característica (Crossa et al., 2017). Los intentos de mejorar caracteres de herencia cuantitativa mediante el uso de SAM han tenido escaso éxito debido a la dificultad de encontrar el mismo QTL en múltiples entornos (debido a QTL por interacciones ambientales) o en diferentes trasfondos genéticos (Bernardo, 2016). El uso de modelos de predicción que utilizan la información del genoma completo generalmente tiene mayor poder para capturar loci de efectos pequeños que SAM (Heffner et al., 2009), más aún en el mejoramiento para características complejas que son controladas por muchos genes de efecto menor (Bernardo, 2008; Cerrudo et al., 2018; Lorenz et al., 2011; Mayor y Bernardo, 2009).

La predicción genómica (PG) se plantea como un método complementario a SAM en el uso de la información genética en programas de mejoramiento. La PG es posible dada la abundancia de polimorfismos de un solo nucleótido (SNP: *single nucleotide polymorphisms*) en el genoma (Mrode, 2014) y la drástica disminución en el costo de genotipado en los últimos años (Crossa et al., 2017), que alcanza un costo menor que el de fenotipado en algunos programas de mejoramiento (Bernardo, 2008). La PG es una estrategia de selección de individuos basada en modelos que utilizan la información de todos los marcadores moleculares de genoma y datos fenotípicos para la selección de individuos candidatos para ser seleccionados con base en sus valores genéticos estimados (Bernardo, 2016; CIMMYT [International Maize and Wheat Improvement Center], 2018; Crossa et al., 2016; Mrode, 2014; Schmid y Bennewitz, 2017).

Para realizar PG, una población que ha sido genotipada y fenotipada, denominada *población de entrenamiento*, se usa para entrenar o calibrar un modelo estadístico con el que luego se predicen los valores genéticos de los individuos de la población de predicción, que no han sido fenotipados, pero sí genotipados (Bassi et al., 2016). Previo a la aplicación de selección genómica en un programa de mejoramiento, es necesario evaluar la habilidad predictiva de los modelos de predicción genómica mediante estrategias de validación cruzada en una población genotipada y fenotipada para la variable que se quiere predecir (Crossa et al., 2017). Se predice que la ganancia genética anual al utilizar PG podría ser dos o tres veces mayor que para un programa de selección fenotípica convencional (Haile et al., 2020), debido a que se incrementa la ganancia genética por unidad de tiempo, al acortar la duración y el número de ciclos de mejoramiento e incrementa la precisión de las evaluaciones a campo. Se espera que la PG permita una reducción en el costo y tiempo de desarrollo de cultivares (Crossa et al., 2017; Jannink et al., 2010). La PG en el mejoramiento genético para resistencia a enfermedades en cultivos ha sido utilizada en diversos trabajos en los últimos años (Poland y Rutkoski, 2016), en particular para caracteres de herencia cuantitativa, siendo el patosistema de las royas del trigo uno de los más estudiados (Daetwyler et al., 2014; Ornella et al., 2017; Rutkoski et al., 2014, 2015, 2016). La PG para resistencia a royas en trigo permitiría reducir el largo de los ciclos de selección en programas de mejoramiento y contribuir a piramidar genes de APR (Rutkoski et al., 2011).

1.8. Mejoramiento genético por resistencia a royas en Uruguay

En el PMGT del INIA se está intentando incorporar resistencia durable a royas con el objetivo de incrementar la vida útil de las variedades. Hay muy escasa información sobre la base genética de resistencia a RE en el germoplasma del PMGT de INIA, basada en la información sobre los genes que confieren también resistencia a roya de la hoja. Para diseñar una estrategia de mejoramiento por resistencia, es fundamental conocer la identidad de los genes o regiones genómicas que confieren resistencia en los materiales utilizados, lo que permite ser más eficiente en el proceso de mejoramiento y evita la redundancia de utilizar materiales portadores de los mismos genes y facilitar la identificación de nuevas fuentes a utilizar para ampliar la diversidad genética, aspecto relevante para el caso de patógenos altamente variables como *Pst*.

El gen *Yr18*/*Lr34* y otros genes que confieren RP están ampliamente distribuidos en el germoplasma de Centro Internacional de Mejoramiento de Maíz y Trigo (CIMMYT) (Singh, Huerta-Espino et al., 2011) que es utilizado en el PMGT de INIA. Se ha confirmado la presencia de *Yr18* en cultivares liberados por PMGT de INIA (Germán y Kolmer, 2012, 2014) y líneas avanzadas del programa (Paula Silva, comunicación personal, 20 de mayo de 2024).

1.9. Problema a abordar

La ocurrencia de epidemias asociadas a la presencia de nuevas razas agresivas de *Pst* en la región, la posible sobrevivencia durante el verano del patógeno en zonas dentro o cercanas al área del cultivo y la constatación de infecciones que provocaron pérdidas importantes en materiales susceptibles indican con alta probabilidad que la RE continuará teniendo gran importancia económica, se presentará con frecuencia y causará daños de magnitud cuando no sea controlada con fungicidas.

La disponibilidad de cultivares resistentes es la medida de control más sustentable. Se debe comenzar a trabajar en este tema lo antes posible, intentar abarcar una amplia gama de recursos genéticos y obtener la mayor información posible para desarrollar germoplasma adaptado resistente. El proceso de mejoramiento genético insume varios años desde que se realiza un cruzamiento hasta liberar cultivares, por lo que la disponibilidad de cultivares resistentes con alto potencial de rendimiento y la calidad requerida por el mercado será a mediano plazo. El objetivo general del proyecto es

contribuir a la sustentabilidad de la producción nacional de trigo al aportar materiales resistentes, información sobre la base de resistencia a RE y la variabilidad del patógeno.

1.10. Hipótesis

Las epidemias recientes de RE en Uruguay están asociadas a la introducción de razas de *Pst* provenientes de otros continentes, que han migrado y se han establecido en la región.

El germoplasma nacional de trigo contiene regiones genómicas asociadas a la resistencia a RE. El apilado (o priamidización) de múltiples regiones de resistencia en una misma línea permitirá alcanzar niveles elevados de resistencia, lo que las convierte en recursos valiosos para el desarrollo de cultivares resistentes en el marco del PMGT.

El comportamiento fenotípico de líneas de trigo frente a RE podría ser predicho mediante el uso de modelos de predicción basados en información genómica. La incorporación de información genómica adicional, como marcadores asociados a QTL de resistencia a RE mejorarán la precisión de los modelos de predicción genómica.

1.11. Objetivo general

Contribuir al incremento y la sustentabilidad de la producción de trigo en Uruguay a través del desarrollo de materiales resistentes a roya esriada.

1.12. Objetivos específicos

Identificar las razas de *Pst* presentes en Uruguay y nuevas razas que puedan aparecer durante la ejecución del proyecto mediante fenotipos de avirulencia/virulencia e instrumentar la asignación de los aislamientos a linajes de referencia internacional mediante genotipado con marcadores microsatélites.

Identificar regiones genómicas asociadas con la resistencia a RE en un panel de materiales diversos de trigo a través de GWAS y evaluar la precisión de modelos de predicción genómica para la resistencia a RE en líneas de trigo.

Estos esfuerzos tienen el objetivo de aportar información y materiales para el control de RE de trigo con base en resistencia genética, considerada la mejor estrategia de control por su menor impacto económico y ambiental asociado a la reducción del uso de fungicidas.

1.13. Estructura de la tesis

El capítulo 1 contiene una introducción general que abarca desde la importancia y contexto de la producción de trigo hasta la situación y perspectivas de la RE en la región, que lleva a las hipótesis y objetivos de esta tesis de doctorado.

El capítulo 2 desarrolla los objetivos específicos vinculados al estudio de la diversidad del patógeno *Pst*, cuyos resultados están publicados en Riella, V., Rodríguez-Algaba, J., García, R., Pereira, F., Silva, P., Hovmøller, M.S. y Germán, S. (2024). New races with wider virulence indicate rapid evolution of *Puccinia striiformis* f. sp. *tritici* in the Southern Cone of America. *Plant Disease*, *108*(8), 2454-2461 (https://doi.org/10.1094/PDIS-02-24-0320-RE).

El capítulo 3 trata sobre la identificación de regiones genómicas asociadas a la resistencia a RE y la implementación de modelos de PG y sus resultados se encuentran publicados en Riella, V., Lado, B., Condón, F., Pritsch, C., Quincke, M., Kavanová, M., García, R., Pereira, F., Pérez, N., Castro, A., Gutiérrez, L., Germán, S. y Silva, P. (2025). Wheat yellow rust in Uruguay: understanding the genetic resistance in a panel of breeding and commercial germplasm. *Theoretical and Applied Genetics*, *138*, 145 (https://doi.org/10.1007/s00122-025-04937-5).

El capítulo 4 presenta resultados y discusión general de todos los estudios realizados.

En el capítulo 5 se presentan las conclusiones generales del trabajo.

2. Nuevas razas con mayor espectro de virulencia indican una rápida evolución de *Puccinia striiformis* f. sp. *tritici* en el Cono Sur de América

2.1. Resumen

La roya amarilla o roya estriada del trigo, causada por *Puccinia striiformis* f. sp. *tritici* (*Pst*), es una de las enfermedades más devastadoras del trigo en el mundo. Las poblaciones de *Pst* están compuestas por múltiples grupos genéticos, cada uno portador de una o más razas caracterizadas por diferentes combinaciones de genes de avirulencia/virulencia sobre genotipos específicos de trigo. Desde las graves epidemias de 2017, la roya amarilla se ha convertido en la enfermedad foliar del trigo de mayor importancia económica en Uruguay. Se caracterizó fenotípicamente un conjunto de 124 aislados de *Pst* recolectados en campos de trigo en Uruguay entre 2017 y 2021, y 27 de esos aislados fueron posteriormente investigados en profundidad mediante análisis adicionales de genotipado molecular y fenotipado de razas. Se identificaron tres grupos genéticos: *PstS7*, *PstS10* y *PstS13*, de los cuales *PstS13* el más prevalente. Se detectaron dos razas previamente reportadas en Europa, Warrior (*PstS7*) y Benchmark (*PstS10*), en cuatro y dos aislados, respectivamente. Una tercera raza conocida como Triticale2015 (*PstS13*), detectada por primera vez en Europa en 2015 y en Argentina en 2017, fue identificada en ocho aislados. Se detectó virulencia adicional sobre *Yr3*, *Yr17*, *Yr25*, *Yr27* o *Yr32* en tres nuevas variantes dentro de *PstS13*. La identificación de estas tres nuevas razas, que no han sido reportadas fuera de Sudamérica, proporciona evidencia sólida de la evolución local de la virulencia en *Pst* durante los recientes años de epidemia.

Palabras clave: roya amarilla (estriada) de trigo, fenotipado de razas, caracterización genética, trigo

New Races with Wider Virulence Indicate Rapid Evolution of *Puccinia striiformis* f. sp. *tritici* in the Southern Cone of America

2.2. Abstract

Wheat yellow or stripe rust, caused by *Puccinia striiformis* f. sp. *tritici* (*Pst*), is one of the most devastating diseases of wheat worldwide. *Pst* populations are composed of multiple genetic groups, each carrying one or more races characterized by different combinations of avirulence/virulence genes on specific wheat genotypes. Since the severe epidemics in 2017, yellow rust has become the most economically important wheat foliar disease in Uruguay. A set of 124 *Pst* isolates collected from wheat fields in Uruguay between 2017 and 2021 were characterized phenotypically and 27 of those isolates were subsequently investigated in-depth by additional molecular genotyping and race phenotyping analyses. Three genetic groups were identified, i.e., *PstS7*, *PstS10* and *PstS13*, being *PstS13* the most prevalent. Two races previously reported in Europe, Warrior (*PstS7*) and Benchmark (*Pst*S10), were detected in four and two isolates, respectively. A third race known as Triticale2015 (*PstS13*), first detected in Europe in 2015 and in Argentina in 2017, was detected in eight isolates. Additional virulence to *Yr3, Yr17*, *Yr25*, *Yr27* or *Yr32* was detected in three new race variants within *PstS13*. The identification of these three new races, which have not been reported outside South America, provides strong evidence of the local evolution of virulence in *Pst* during the recent epidemic years.

Keywords: Yellow (stripe) rust, race typing, genotypic characterization, wheat

2.3. Introduction

Wheat yellow (stripe) rust, caused by *Puccinia striiformis* f. sp. *tritici* (*Pst*), is one of the most devastating diseases of wheat worldwide (Chen et al. 2014; Wellings 2011; Stubbs 1985; Beddow et al. 2015). *Pst* is favored by relatively low temperatures of 10-15ºC and may cause very significant grain yield losses in susceptible cultivars (Chen 2005; Carmona et al. 2019; Roelfs et al. 1992). Historically, *Pst* has mainly been a problem in cool climates, however, since 2000, the pathogen has gained increased tolerance to higher temperatures and become an increasing problem in areas normally considered too warm for *Pst* establishment (Wellings 2007; Milus et al. 2009). Moreover, *Pst* epidemics originating from distant geographical areas have been reported, either as an incursion to new regions where it was previously absent or as a re-emergence of new

races with increased aggressiveness (Bahri et al. 2009; Hovmøller et al. 2023b). As a consequence, *Pst* epidemics have been an increasing problem threatening global wheat production (Boshoff et al. 2002; Ali et al. 2014; Hovmøller et al. 2016).

Pst was detected for the first time in Uruguay and Argentina in 1929 (Rudolf and Job 1931). During 1929 and 1930, *Pst* caused widespread epidemics through most of the Southern Cone region, causing extremely high yield losses (Boerger 1934; Vallega 1938). From its first detection and until 2016, *Pst* occurred sporadically, rarely reaching epidemic levels in Uruguay (Germán and Caffarel 1999; Germán et al. 2007, 2018). Since 2017, *Pst* has caused generalized epidemics in Uruguay and Argentina (Germán et al. 2018, 2021; Carmona and Sautua 2018; Campos 2020). *Pst* is currently the most economically important wheat foliar disease, requiring the highest number of fungicide applications relative to other prevalent diseases. This is probably due to the earlier onset of the disease during the growing season and because more than 50% of the wheat area has been deployed with susceptible or moderately susceptible cultivars (Silva et al., 2023). To control *Pst*, farmers typically spray two fungicide applications each growing season to obtain adequate disease control. The most susceptible wheat cultivars to *Pst* included in the National Cultivar Evaluation trials in Uruguay, had grain yield losses of up to 82% in 2017 comparing trials with and without fungicide application (Germán et al. 2018). Additionally, in Argentina, *Pst* races have overcome the resistance of most of the locally adapted cultivars (Carmona et al. 2019).

Mutations and subsequent selection are considered the main driving forces that generates new races with virulence to the deployed host resistance genes (de Vallavieille-Pope et al. 2012; Hovmøller and Justesen 2007). Sexual recombination is another mechanism that may generate variability in *Pst*, which has been reported under experimental conditions involving the alternate host *Berberis spp.* (Rodriguez-Algaba et al. 2014, 2020), however, rarely reported under natural conditions. As these alternate hosts are not present in Uruguay, sexual recombination is thought to have no impact as a source of new variability locally, leading to the assumption of a strongly clonal *Pst* population in Uruguay as reported for other regions (Ding et al. 2021; Hovmøller et al. 2002).

Races of *Pst* are characterized phenotypically through their avirulence/virulence pattern to *Yr* resistance genes. Additionally, the *Pst* genetic diversity can be studied using microsatellite molecular markers to assign the different *Pst* isolates to different genetic groups (Bai et al. 2021; Sharma-Poudyal et al. 2020; Walter et al. 2016; Ali et al. 2017),

allowing a better understanding of the evolutionary and dispersal dynamics of the pathogen (Thach et al. 2016; Ali et al. 2014; Ding et al. 2021).

Recent *Pst* epidemics worldwide and the spread of epidemics to new areas, where the disease was previously not relevant, make it urgent to understand the evolution of *Pst* races, their spread and establishment. Knowledge of the evolution of the pathogen virulence is a key factor in determining the best strategy to breed locally adapted cultivars with effective and durable resistance to *Pst*. In the present study, we examined the population structure of *Pst* in Uruguay based on samples collected from wheat fields between 2017 and 2021. The isolates were assigned to races to study the diversity for virulence. Subsets of isolates were genotyped and assigned to internationally defined genetic groups, which allowed interpretation of the results in a wider geographical and evolutionary context. The results have strong implications for prevention and control of rust diseases in wheat.

2.4. Materials and methods

A collection of 124 *Pst* isolates maintained at INIA-La Estanzuela (Colonia, Uruguay) was recovered from samples collected from spring bread wheat (*Triticum aestivum*) at different locations in the major wheat production area in Uruguay between 2017 and 2021. A set of 27 representative isolates were selected from the 124 *Pst* isolates based on the avirulence/virulence phenotypes observed at the preliminary race phenotyping (Table 1), sampled cultivars (when available), location and year (Supplementary material 1). From the 27 samples, 4 were collected in 2017, 5 in 2018, 2 in 2019, 11 in 2020 and 5 in 2021 (Supplementary material 1).

Table 1. Number of isolates with different virulence phenotypes for the 124 samples collected in Uruguay during 2017-2021, and number of isolates selected for in depth phenotypic analysis.

Virulence phenotype	2017	2018	2019	2020	2021	Total	Selected for in depth analysis
1,2,3,4,NA,6,7,-,9,-,-,17,NA,25,-,32,Sp,AvS,Amb					22	**22**	1
1,NA,NA,NA,-,-,7,-,9,-,-,17,-,NA,-,NA,Sp,AvS,NA		5		4		**9**	4
1,2,3,4,NA,6,7,-,9,-,-,17,NA,25,-,32,Sp,AvS,-					4	**4**	0
-,NA,NA,NA,-,6,7,8,9,-,-,-,-,NA,-,NA,-,AvS,NA	7					**7**	4
-,2,-,-,NA,6,7,8,9,-,-,-,NA,-,-,-,-,AvS,-					3	**3**	1
-,NA,NA,NA,-,NA,7,NA,9,-,-,17,-,-,-,-,-,AvS,NA		3	3			**6**	4
-,NA,NA,NA,-,NA,7,NA,9,-,-,17,-,NA,-,NA,-,AvS,NA		2	2			**4**	2
-,NA,NA,NA,-,6,7,8,9,-,-,17,-,NA,-,32,-,AvS,NA				12		**12**	7
-,2,-,-,NA,6,7,8,9,-,-,17,NA,-,-,32,-,AvS,-					10	**10**	1
-,2,-,-,NA,6,7,8,9,-,-,17,NA,-,27,32,-,AvS,-					41	**41**	2
-,2,3,-,NA,6,7,8,9,-,-,17,NA,25,-,32,-,AvS,-					6	**6**	1

Note. Figures and abbreviations indicate virulence to yellow rust resistance genes *Yr1, Yr2, Yr3, Yr4, Yr5, Yr6, Yr7, Yr8, Yr9, Yr10, Yr15, Yr17, Yr24, Yr25, Yr27, Yr32*, Spalding Prolific (*Sp*), Avocet S (*AvS*) and Ambition (*Amb*), respectively. The hyphen "-" indicates avirulence. "NA" indicates that the Yr-gene was not included in the differential set.

2.4.1. Sample recovery and race typing

The original samples, preserved at 5ºC, were used to inoculate seedlings of susceptible wheat cultivar Morocco. A reduced differential set representing 13 R-genes (*Yr1, Yr5, Yr6, Yr7, Yr8, Yr9, Yr10, Yr15, Yr17, Yr24, Yr27, Sp, AvS*) was used for preliminary race phenotyping of the 38 Pst isolates collected between 2017 and 2020. Additional 86 samples collected in 2021 were race phenotyped using an extended differential set representing 17 R-genes (*Yr1, Yr2, Yr3, Yr4, Yr6, Yr7, Yr8, Yr9, Yr10, Yr15, Yr17, Yr25, Yr27, Yr32, Sp, AvS, Amb*). Based on these data (Table 1), 27 isolates (Supplementary material 1) were selected for in depth molecular genotyping and virulence phenotyping.

For the 27 selected isolates, infected leaves were collected separately and sent to the Global Rust Reference Center (GRRC) in Denmark following their standard practice recommendations for sample shipment. For isolate testing at the GRRC, the protocol described in Hovmøller et al. (2017) was used. Leaf segments were placed in Petri dishes with moist filter paper and incubated between 24h to 48h to promote spore production. Seedlings of wheat cultivars Morocco and Morocco/*Lr*19 were used for multiplication of spores. Seedlings were grown in pots, 12–15 plants per pot, and treated with 5ml of 0.5%

Antergon MH180 (Nordisk Alkali, Randers, Denmark) to regulate plant growth and enhance spore production. Leaves with newly emerged urediniospores were gently rubbed on the wheat seedlings. Inoculated seedlings were misted with water and incubated in a dew chamber at 10–12ºC in darkness for 24h, then transferred to spore-proof greenhouse cabins at 17ºC during the day/12ºC during the night with a 16h photoperiod of natural light and supplemental sodium light (100µmol/s/m) and 8h darkness, relative humidity of 70–80%. Pots were covered with cellophane bags (Helmut Schmidt Ver-packungsfolien GmbH, Königswinter, Germany) prior to sporulation to prevent cross-contamination among isolates. Spores were harvested by shaking the plants inside the cellophane bag, and then transferred to cryovials. These were then dried in a desiccator for approximately three days and preserved at -80ºC until further use.

The 27 isolates were phenotyped using a set of 24 wheat differential lines (Table 2). A supplementary test was carried out including additional lines for further confirmation of specific race variants. Previously characterized reference isolates of the genetic groups detected for the Uruguayan isolates were included as controls. Ten to twelve seeds of each wheat genotype were sown in 7×7×8cm pots with a 1:1 Pindstrup Substrate peat mix containing slow-release plant nutrients (Pindstrup Mose-brug A/S, Ryomgaard Denmark). Differential sets with one pot of each wheat genotype were placed in a tray and grown in spore-proof cabins in the greenhouse at 17ºC day/12ºC night temperature regime and inoculated approximately 12 days after sowing when the second leaf was half unfolded. Urediniospores were retrieved from -80ºC and used for inoculation after heat shock treatment in a water bath at 40–42ºC for 2min. Approximately 25mg of spores were suspended in 3ml engineered fluid 3M™ Novec™ 7100, (3M, St. Paul, MN, USA) and gently mixed. Wheat seedlings were spray inoculated using an airbrush spray gun (standard class, Revell GmbH, Bünde, Germany) in a laboratory fume hood. Seedlings were subsequently sprayed with mist water, incubated in a dew chamber, and transferred to the greenhouse under the same conditions described above. Infection type (IT) was scored on individual plants/leaves after 15–17 days. The first and the second leaves were scored separately using a 0–9 scale (McNeal et al. 1971), where scores between 7 and 9 indicated compatibility (virulence) and scores equal to or below 6 indicated incompatibility (avirulence).

Table 2. Wheat differential lines used for race typing of *Puccinia striiformis* f. sp. *tritici* isolates.

Differential line	Yellow rust resistance genes (*Yr*)	GRRC Standard set	Supplementary test
Chinese 166	*1*	x	
Kalyansona	*2, +*	x	x
Vilmorin 23	*3, +*	x	x
Hybrid 46	*4, +*	x	
Suwon	*Su*	x	
Heines Kolben	*6, +*	x	
Avocet *Yr6*	*6, AvS*	x	
Lee	*7, +*	x	
Avocet *Yr8*	*8*	x	
Avocet *Yr9*	*9, AvS*	x	
Moro	*10*	x	
Cortez	*15*	x	
VPM1	*17, +*	x	x
Avocet *Yr17*	*17, AvS*	x	x
TP 981	*25, +*	x	x
Spaldings Prolific	*Sp, 25, +*	x	x
Opata	*27,18, +*	x	x
Avocet *Yr32*	*32, AvS*	x	x
Avocet S	*AvS*	x	x
Strubes Dickkopf	*Sd, 25, +*		x
Heines VII	*2, 25, +*		x
Carstens V	*32, 25, +*		x
Avocet *Yr27*	*27, AvS*		x
Heines Peko	*2, 6, 25, +*		x
Nord Desprez	*3*		x
Ambition	*Amb*	x	
Benchmark	unknown	x	
Kalmar	unknown	x	
Nemo	unknown	x	

2.4.2. Genotypic characterization of isolates

Genomic DNA was extracted from leaf-infected segments according to manufacturer instructions for the Sbeadex® Mini Plant Kit (LGC Genomics, Germany) with an automated KingFisher™ Magnetic Particle Processor (Thermo Fisher Scientific, United States). The genotyping of the 27 isolates was based on 19 Simple Sequence Repeat (SSR) markers according to Rodriguez-Algaba et al. (2017). PCR size fractionation was performed on Applied Biosystems™ 3,730 DNA analyzer (Thermo Fisher Scientific) using the service at KIGene, Karolinska University Hospital, Stockholm, Sweden. The amplicons were visualized in GeneMarker® (Softgenetics), and allele sizes were manually scored using the GeneScan™ 600 Liz® Size Standard (Thermo

Fisher Scientific). This allowed a genetic grouping based on genotypic similarities, i.e., minor (or no) allele differentiation among individuals within a group, and major differences between groups, following the principles of Ali et al. (2017). Allele sizes detected for individual isolates and associated genetic groups are presented in Supplementary material 2.

2.5. Results

2.5.1. Preliminary race typing

The preliminary race typing results for the 124 samples are presented in Table 1. This showed 11 distinct virulence phenotypes with different degrees of differentiation regarding avirulence/virulence towards 10 resistance genes. The first three virulence phenotypes shared virulence to *Yr1*, *Yr7*, *Yr9* and *Yr17*, but differed on their compatibility on cultivar *Ambition*. A second group (rows 4 to 11 in Table 1), shared avirulence to *Yr1* and virulence to *Yr2*, *Yr6*, *Yr7*, *Yr8*, *Yr9*, and *AvS*, but differed on their avirulence/virulence to *Yr3*, *Yr17*, *Yr27*, *Yr25* and *Yr32*. Based on these results, 27 isolates representing virulence diversity, host cultivars, locations and years were selected to in depth genotyping and additional virulence testing.

2.5.2. *Pst* genetic groups

The genetic profiles for the 27 samples analyzed with the 19 SSRs were compared with reference genetic profiles belonging to genetic groups reported worldwide (Hovmøller et al. 2016; Ali et al. 2017). The 27 isolates were assigned to one of the previously reported genetic groups if their genetic profiles for the set of 19 SSRs matched the reference profile. Three different *Pst* genetic groups were detected in Uruguay (Supplementary material 2). *PstS13* was the most prevalent group with 78% of the isolates analyzed, followed by *PstS7* with 15% and *PstS10* with 8% (Table 3). The *PstS13* group was identified in samples collected from all sampling years, *PstS7* was found for the first time in 2018 and then in 2020 and 2021, and *PstS10* was only detected in 2020.

2.5.3. Race typing

The virulence phenotype of the 27 isolates and their corresponding genetic group are presented in Table 3. The primary results of the phenotyped isolates are presented in Supplementary material 3. Four isolates with virulence on *Yr1, Yr2, Yr3, Yr4, Yr6, Yr7, Yr9, Yr17, Yr25, Yr32, Sp, AvS,* and *Amb,* shared virulence phenotype with a reference

isolate of the "Warrior" race (*PstS7*). Two isolates were assigned to the "Benchmark" race (*PstS10*), which is characterized by avirulence to European cultivar Ambition and virulence to the European cultivar Benchmark. Four additional closely related virulence phenotypes were detected among the isolates belonging to *PstS13* (Table 4). Within *PstS13* group, a race virulent on *Yr2, Yr6, Yr7, Yr8, Yr9* and *AvS* (Figure 1-a) was detected in 2017 and 2018, which corresponded to the original Triticale2015 race. The supplementary virulence test allowed the verification of three new race variants (Table 4). One variant, first detected in 2019, had gained virulence on *Yr17* and *Yr32*; a second variant, collected in 2020, had additional virulence on *Yr27* and a third variant, collected in 2021, was virulent on *Yr3*, *Yr17*, *Yr25* and *Yr32,* in addition to the virulence detected in the original Triticale2015 race.

Table 3. Number of isolates of different genetic groups and virulence phenotypes for samples collected in Uruguay during 2017-2021

Genetic group	Race		Virulence phenotype	2017	2018	2019	2020	2021
PstS7	Warrior		*1,2,3,4,6,7,-,9,-,-,17,25,-,32,Sp,AvS,Amb*		1		2	1
PstS10	Benchmark		*1,2,3,4,6,7,-,9,-,-,17,25,-,32,Sp,AvS,-*				2	
PstS13	Triticale2015	**a**	*-,2,-,-,6,7,8,9,-,-,-,-,-,-,-,AvS,-*	4	4			
		b	*-,2,-,-,6,7,8,9,-,-,17,-,-,32,-,AvS,-*			2	6	3
		c	*-,2,-,-,6,7,8,9,-,-,17,-,27,32,-,AvS,-*				1	
		d	*-,2,3,-,6,7,8,9,-,-,17,25,-,32,-,AvS,-*					1

Note. Virulence phenotype numbers indicate virulence to yellow rust resistance genes: *Yr1, Yr2, Yr3, Yr4, Yr6, Yr7, Yr8, Yr9, Yr10, Yr15, Yr17, Yr25, Yr27, Yr32*, Spalding Prolific (*Sp*), Avocet S (*AvS*) and Ambition (*Amb*), respectively. The hyphen "-" indicates avirulence. Letters **a-d** indicate different virulence phenotypes within genetic group *PstS13*. All isolates belonging to genetic group *PstS10* were virulent on differential line Benchmark.

Table 4. Infection types on *Pst* differential lines of *Pst13* variants tested in Supplementary test.

Variety =>	Avocet *S*	Strubes Dickkopf	Spaldings Prolific	Kalyansona	Heines Peko	Heines VII	Nord Desprez	Vilmorin 23	VPM1	TP 981	Avocet *Yr17*	Carstens V	Avocet *Yr32*	Avocet *Yr27*	Opata	Race
Yr-genes =>	*AvS*	*Sd,25*	*Sp,25,+*	*2,+*	*2,6,25,+*	*2,25,+*	*3,+*	*3,+*	*17,+*	*25,+*	*AvS,17*	*25,32,+*	*AvS,32*	*AvS,27*	*18,27,+*	
DK69	7	1	5,6,7	7	2	1	2	1	0,1	2	0	0,1	0,2,3	0	2,3,4	Triticale 2015 a
UY147	7	1	5,6	7	2	1	2	1	1	2,3,4	0	0,1	2,3	0	1,2,3	Triticale 2015 a
UY150	7	1	5,6,7	7	2	1	2	1	1	2,3,4	0,1,2	0,1,2	2,3	0,1	0,1,2	Triticale 2015 a
UY152	7	1	5,6,7	7	2	1	2	1	0,1	2,3,4	0,1,2	0,1,2	2,3	0,1	0,1,2	Triticale 2015 a
UY158	7	1	5,6,7	7	2	1	2	1	0,1	2	7	0,1	7	3,4	4,5	Triticale 2015 b
UY168	7	1	5,6,7	7	2	1	2	1	0,1	2	6,7	0,1,2	7	7	7	Triticale 2015 c
UY623	7	2,3	6,7	7	7	5,6	7	5,6	2,3	7	7	6,7	7	4,5,6	4,5	Triticale 2015 d

Note. The first and the second leaves were scored using a 0–9 scale (McNeal et al. 1971), scores between 7 and 9 indicate compatibility (virulence), and scores equal to or below 6 indicate incompatibility (avirulence). Letters a-d indicate different virulence phenotypes within genetic group *PstS13*.

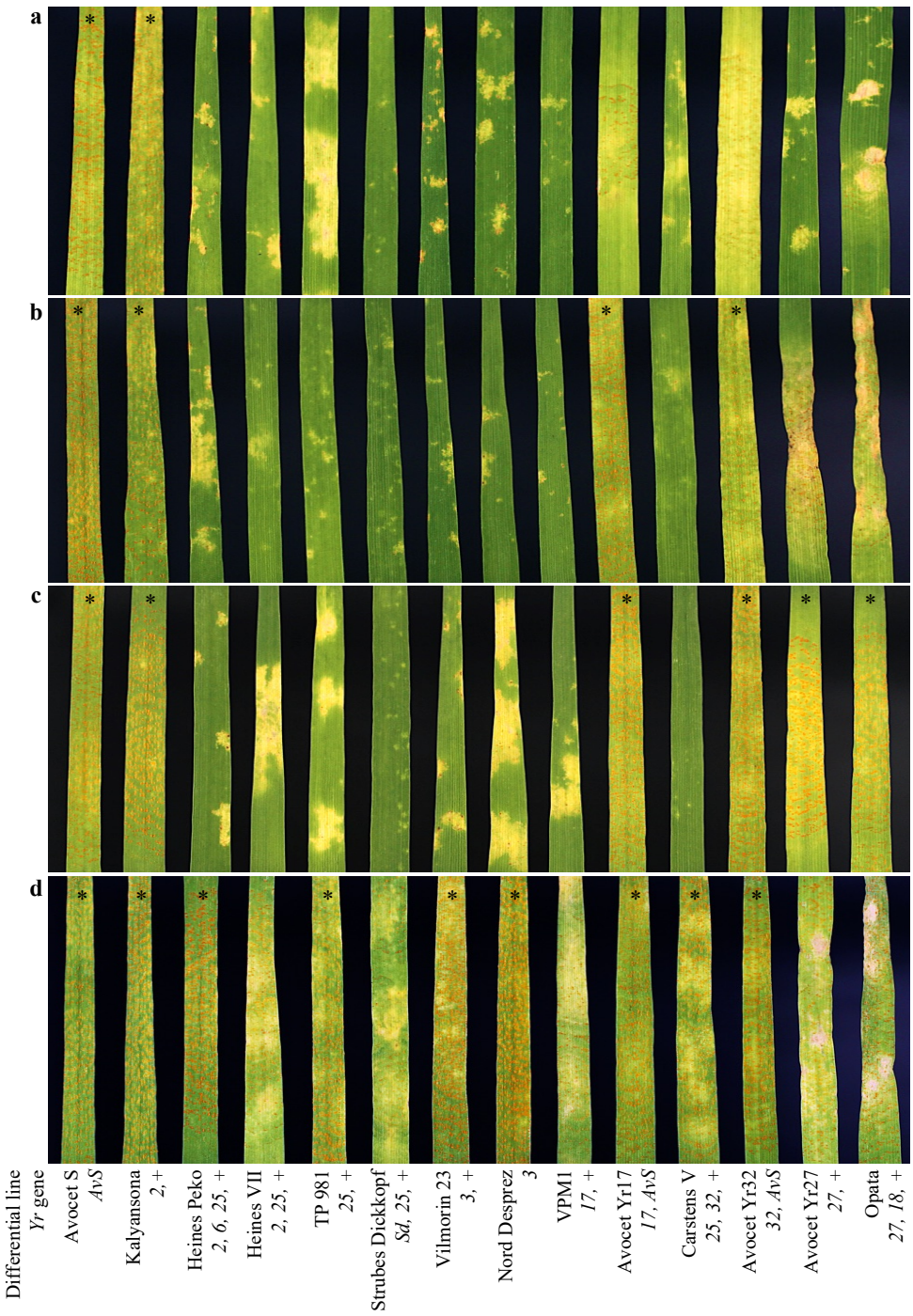


Figure 1. *Puccinia striiformis* f. sp. *tritici* infections on differential lines for the four different races (**a-d**) within Triticale2015 race (*PstS13* genetic group). Original Triticale2015 race **(a)**, races with additional virulence to *Yr17* and *Yr27* (**b**)*;* to *Yr17*, *Yr27* and *Yr32* (**c**)*,* and *Yr3*, *Yr17*, *Yr25* and *Yr32* (**d**), respectively. *: Compatible interaction (infection type 7-9)

2.6. Discussion

The Uruguayan collection of *Pst* isolates composed of 124 samples collected between 2017 and 2021 was preliminarily analyzed for virulence phenotype characterization. Based on the preliminary phenotypic results, a set of 27 isolates was analyzed in depth genetically and phenotypically. The set was considered representative of a local *Pst* population based on sampled cultivar, collection year, location and preliminary race typing. The virulence and genotypic profiles allowed assignment to races within *PstS7*, *PstS10* and *PstS13* genetic groups, some of which were previously reported as responsible for severe epidemics in the last decade (Beddow et al. 2015; Sørensen et al. 2014; Hovmøller et al. 2016; Ali et al. 2017). Races previously described in Europe within each genetic group, were detected in this study, i.e., Warrior (*PstS7*), Benchmark (*PstS10*) and Triticale2015 (*PstS13*). Triticale2015, first detected in Europe in 2015 (Hovmøller et al. 2018) and in Argentina in 2017 (Carmona et al. 2019), was found in several of the *Pst* isolates. Additionally, three new race variants within the *PstS13* genetic group were also identified. These results provide strong evidence not only of migration and establishment of the disease in regions where *Pst* was not previously widespread but also revealed the emergence of new race variants with wider virulence indicating the existence of regional evolution of *Pst.*

The appearance in the Southern Cone of America of *Pst* genetic groups previously reported in Europe may be associated with human activities (Zadoks 1961; Stubbs 1985; Brown and Hovmøller 2002; Wellings 2007; Ali et al. 2014) and/or long distance wind dispersal (Zadoks 1961; Brown and Hovmøller 2002). Argentina and Uruguay are located in the same rust epidemiological zone (Rajaram and Campos 1974), where there are no geographical barriers for urediniospores dispersal, which likely explains the almost simultaneous development of severe epidemics in both countries (Campos et al. 2016; Carmona et al. 2019; Germán et al. 2018). More recently *Pst* was detected in Paraguay for the first time (Fernández-Gamarra et al. 2023) and in Brazil. Before 2017, *Pst* did not cause severe epidemics in the epidemiological zone East of the Andes as the pathogen survived during the summer distant from the wheat crop regions (Germán et al. 2007). Since 2017, a relevant epidemiological change associated with the presence of *Pst* in Uruguay is the likely increased oversummering capacity of the pathogen. In fact, oversummering of the pathogen has been observed in the Argentinian wheat crop area,

which allows primary inoculum to infect crops earlier and thus cause severe epidemics not only in Argentina but also in Uruguay (Germán et al. 2018; Silva et al. 2023).

Among the three genetic groups detected in this study, *PstS7* and *PstS10* had the lowest prevalence. Since 2011 and 2012, both genetic groups have been associated with important epidemics with substantial economic losses in various parts of the world (Hovmøller et al. 2016; Hubbard et al. 2015). In Europe, new races from *PstS7* and *PstS10* genetic groups have been detected since 2011 and have replaced the previous pathogen population (Sørensen et al. 2014; Ali et al. 2017). So far, only one race has been described within the *PstS7* genetic group (Warrior race), which has spread from Europe to North Africa (Hovmøller et al. 2016) and South America (Hovmøller et al. 2023a). The genetic group *PstS10* has been the most prevalent genetic group in Europe from 2013 to 2022. One race has been prevalent within this genetic group, but some variants virulent to widely grown cultivars have been described (Hovmøller et al. 2020). In 2018, it was also detected in Australia (Ding et al. 2021; Park et al. 2020). Up to now, it has not yet been possible to differentiate the new race variants within *PstS10* by molecular techniques nor by standard wheat differential lines (Hovmøller et al. 2022). In terms of virulence, the races within *PstS10* are similar to the Warrior race (*PstS7*), except for their avirulence/virulence to the European wheat cultivars Warrior and Ambition (Hovmøller et al. 2020).

In this study, *PstS13* was the most prevalent genetic group. *PstS13* was first reported in Europe in 2015, mainly affecting triticale and durum wheat (Hovmøller et al. 2018). A single race within this genetic group is still prevalent in Europe although local variants have been observed (Hovmøller et al. 2018). The same genetic group caused severe epidemics on durum and bread wheat in Italy in 2017 (Hovmøller et al. 2018). A new *Yr10* virulent variant was detected in Poland in 2019 and in Germany in 2020 (Hovmøller et al. 2021). In Australia, *PstS13* was detected in 2018 and has become one of the most widespread genetic groups (Ding et al. 2021; Park et al. 2020). In South America, *PstS13* was reported as the prevalent genetic group in Argentina in 2017 and 2018 (Carmona et al. 2019; Hovmøller et al. 2018, 2019). Samples collected in Chile during 2018 were also assigned to this genetic group (Hovmøller et al. 2019). All samples from Argentina and Chile showed the same virulence phenotype as the original *PstS13* race reported in Denmark (Hovmøller et al. 2019, 2020). More recently, *PstS13* spread to Paraguay (Fernández-Gamarra et al. 2023). Since *PstS13* has been the most prevalent genetic group in South America accounting for severe *Pst* epidemics in the region, thus

new virulence variants could be expected based on previously reported high mutation rates in yellow/stripe rust (Hovmøller and Justesen 2007).

Here we report new race variants in South America with additional virulence within the *PstS13* genetic group. The emergence of new *Pst* races within the same genetic group is probably due to mutation and subsequent selection of races with virulence to the deployed host resistance genes as suggested by de Vallavieille-Pope et al. (2012) and Hovmøller and Justesen (2007). Through this mechanism, *Pst* isolates may acquire the ability to avoid recognition by resistance genes in host plants resulting in step-wise mutations (Hovmøller et al. 2002). Similar patterns have been observed in Australia, where the sexual cycle of *Pst* does not occur, and new races usually differ from existing races by virulence on a single resistance gene (Wellings and McIntosh 1990; Park 2015; Ding et al. 2021). The detection of new races in higher frequencies may be related to the presence of the corresponding resistance genes in wheat cultivars prevalent locally. The presence of new races with wider virulence was not associated to a dramatic change on field susceptibility of any of the widely grown cultivars. However, during 2017-2021 some cultivars changed their level of susceptibility from very low to intermediate (cv. Génesis 6.87, cv. Génesis 4.33 and cv. SYN 211) or from intermediate to high (cv. Algarrobo) (Instituto Nacional de Investigación Agropecuaria [INIA], Instituto Nacional de Semillas [INASE], 2023). The observed changes in the level of susceptibility is probably due to the presence of the newly detected *PstS13* race variants.

One possible pathway to explain the observed pathogen evolution in Uruguay starts with the original *PstS13* detected in samples from 2017 and 2018 (*PstS13*a, Table 3). *PstS13*b, detected in 2019, gained virulence on *Yr17* and *Yr32*. More recently, two new races emerged, *PstS13*c (year 2020), which gained virulence on *Yr27*, and the *PstS13*d (year 2021), which gained virulence on *Yr3* and *Yr25* in addition to the virulence pattern observed for *PstS13*b. The new *PstS13*d variant, virulent on both *Yr3* and *Yr25* could be the result of two subsequent single-set mutations. This hypothesis implies the existence of a race virulent on *Yr3* or *Yr25*, but it has not been detected in local surveys so far, which may be due to the small sample size or because it might have not been present in Uruguay. Another possible scenario of the appearance of *PstS13*d virulent on *Yr3* could be that this race variant was already present in the *PstS13* races, but not detectable by the differential lines used up to date. The detection of *PstS13d* with virulence to *Yr3* and *Yr25* allows a better interpretation of the genes that might be present in some of the differential lines. Differential lines Vilmorin 23 and Nord Desprez, both

carrying *Yr3* (Chen et al. 1996; Chen and Line 1993), resulted in a compatible interaction when tested with the *Yr25*-virulent race, which suggests that these differentials might also carry *Yr25*. The possible presence of *Yr25* in some differential lines, i.e., Carstens V, Spaldings Prolific and Strubes Dickkopf, was previously suggested (Calonnec and Johnson 1998; Eriksen et al. 2004; Calonnec et al. 2002). Following the assumption that differential lines carrying *Yr3* may also carry *Yr25*, indicate that *Yr3* is probably ineffective in previously reported *PstS13* races, which has been masked by a low IT conferred by the *Avr25/Yr25* phenotype. If the two *Yr3* differentials also carry *Yr25*, we cannot securely confirm virulence/avirulence for *Yr3* using the current differential lines, except for mutant isolates with virulence on *Yr25*. Interpretation of virulence entirely depends on knowledge about R-genes (known or unknown) in the differential lines, and many of the differential lines currently used could also contain other uncharacterized R-genes (Johnson 1992). In this context, the use of multiple differential lines representing each *Yr*-gene could be recommended for a more precise race typing analysis.

In this study, we were able to detect three widely spread *Pst* genetic groups. However, additional *Pst* genetic groups could have been undetected. For example, *PstS14* reported in Argentina in 2017 (Carmona et al. 2019), was not detected in Uruguay despite the geographical proximity of both countries. *PstS14* was first detected in Northern Africa and Europe in 2016, causing severe epidemics on bread wheat in Morocco in 2017 (Hovmøller et al. 2018). Future surveys, with a larger number of samples collected at a regional scale, could complement this work and provide further insights into the understanding of the current distribution of genetic groups in the Southern Cone of South America.

2.7. Conclusions

The hypothesis of initial migration of *Pst* into Southern Cone of America from distant pathogen sources confirmed the current epidemic outbreaks observed in the region. The identification of *Pst* races in Uruguay that were not previously described provides strong evidence of recent evolution of virulence of *Pst* in Uruguay and possibly Argentina, where the most severe epidemics occurred. The emergence of new races of *Pst* with wider virulence has implications for the management of the disease and plant breeding for disease resistance in Uruguay and neighboring countries. In the short term, local management of ongoing epidemics on susceptible wheat varieties is limited to fungicide spraying. However, the deployment of resistant wheat cultivars appears as the

most environmentally friendly strategy without additional cost for producers. The development of resistant cultivars implies permanent monitoring of the *Pst* races to generate cultivars with effective resistance to races present in a specific epidemiological zone. The occurrence of long-distance dispersal of races migrating from distant continents emphasizes the relevance of worldwide coordination of survey efforts. The new race variants reported in this work might migrate to other areas as well and potentially cause important yield losses in regions where cultivars that possess the overcome *Pst* resistance genes are deployed.

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2.9. Supplementary material

- **Supplementary material 1**. Information of the 124 samples collected from spring-bread wheat in Uruguay during 2017-2021.

Collection year	Site	Region	Latitude	Longitude	Altitude	Variety	ID in set of 27
2017	Farmer Field	Soriano	-33.596958	-57.697885	105		UY147
2017	Farmer Field	Colonia	-33.887676	-58.365218	28		UY148
2017	Farmer Field	Rio Negro	-33.077091	-58.054353	25		UY149
2017	Farmer Field	Florida					
2017	Trial	Colonia	-34.336435	-57.712592	72		UY150
2017	Farmer Field	Florida					
2017	Farmer Field	San José					
2018	Trial	Rio Negro	-32.68305	-57.65959	83	Ceibo	
2018	Trial	Rio Negro	-32.687306	-57.65625	70		UY151
2018	Trial	Rio Negro	-32.68305	-57.65959	83	Fuste	
2018	Trial	Colonia	-34.336435	-57.712592	72		UY152
2018	Trial	Colonia	-34.33888	-57.72702	80	DM 1601 T	
2018	Trial	Colonia	-34.33888	-57.72702	80	Lalbahadur	
2018	Trial	Colonia	-34.33888	-57.72702	80	LE 2359	
2018	Trial	Colonia	-34.336435	-57.712592	72	LE2320 x Onix x Nogal	UY153
2018	Trial	Colonia	-34.336435	-57.712592	72	LE 2359	UY154
2018	Trial	Colonia	-34.336435	-57.712592	72	LE2320 x Onix x Nogal	UY155
2019	Trial	Colonia	-34.33888	-57.72702	80		
2019	Trial	Colonia	-34.33888	-57.72702	80		
2019	Trial	Colonia	-34.33888	-57.72702	80		
2019	Trial	Colonia	-32.679278	-57.648778	79	Fuste	UY156
2019	Trial	Colonia	-34.336435	-57.712592	72	Fuste	UY157
2020	Trial	Rio Negro	-32.683049	-57.659589	87		UY158
2020	Trial	Colonia	-34.33888	-57.72702	80		
2020	Trial	Colonia	-34.336435	-57.712592	72		UY159
2020	Trial	Colonia	-34.33888	-57.72702	80		
2020	Trial	Colonia	-34.336435	-57.712592	72	Thatcher Lr19	UY160
2020	Trial	Colonia	-34.33888	-57.72702	80		
2020	Trial	Colonia	-34.336435	-57.712592	72		UY161
2020	Trial	Colonia	-34.336435	-57.712592	72		UY162
2020	Trial	Colonia	-34.336435	-57.712592	72	Génesis687	UY163
2020	Trial	Colonia	-34.336435	-57.712592	72		UY164
2020	Trial	Colonia	-34.336435	-57.712592	72		UY165
2020	Trial	Colonia	-34.336435	-57.712592	72		UY166
2020	Trial	Colonia	-34.33888	-57.72702	80		
2020	Trial	Colonia	-34.336435	-57.712592	72	Klein Mercurio	UY167
2020	Trial	Colonia	-34.336435	-57.712592	72	Klein Mercurio	UY168
2020	Trial	Colonia	-34.33888	-57.72702	80		
2021	Trial	Paysandú	-32.3766	-58.05849	47	Fuste	
2021	Trial	Paysandú	-32.3766	-58.05849	47	DM Catalpa	
2021	Farmer Field	Paysandú	-32.46941	-57.9316	40		
2021	Trial	Rio Negro	-32.68305	-57.65959	87	Ceibo	
2021	Farmer Field	Rio Negro	-32.99775	-57.98839	74		

2021	Farmer Field	Rio Negro	-32.99775	-57.98839	74	
2021	Trial	Rio Negro	-32.68305	-57.65959	87	Genesis638
2021	Trial	Rio Negro	-32.68305	-57.65959	87	Sauce
2021	Farmer Field	Rio Negro	-32.71959	-57.73703	70	
2021	Trial	Rio Negro	-32.68283	-57.65931	87	Lenox
2021	Farmer Field	Rio Negro	-32.86106	-57.98792	42	
2021	Trial	Rio Negro	-32.68305	-57.65959	87	Aca 2622
2021	Trial	Rio Negro	-32.68305	-57.65959	87	
2021	Trial	Rio Negro	-32.68305	-57.65959	87	
2021	Farmer Field	Colonia	-34.09457	-58.19048	26	Audaz
2021	Farmer Field	Colonia	-33.83092	-58.02348	35	
2021	Farmer Field	Colonia	-33,632,370	-58,172,900	28	Ñandubay
2021	Farmer Field	Colonia	-33,696,170	-58.11001	25	
2021	Farmer Field	Colonia	-33.52697	-58.13083	52	
2021	Trial	Colonia	-34.33644	-57.71259	72	Algarrobo
2021	Trial	Colonia	-34.33644	-57.71259	72	Ceibo
2021	Trial	Colonia	-34.33644	-57.71259	72	Fuste
2021	Trial	Colonia	-34.33644	-57.71259	72	Kleinprometeo
2021	Trial	Colonia	-34.33644	-57.71259	72	Kleinmercurio
2021	Trial	Colonia	-34.33644	-57.71259	72	Genesis4.33
2021	Trial	Colonia	-34.33644	-57.71259	72	Kleinnutria
2021	Trial	Colonia	-34.33644	-57.71259	72	Aca908-zeus
2021	Trial	Colonia	-34.33644	-57.71259	72	Buckcharrua
2021	Trial	Colonia	-34.33644	-57.71259	72	Kleinseleniocl
2021	Trial	Colonia	-34.33644	-57.71259	72	Kleincastor
2021	Trial	Colonia	-34.33644	-57.71259	72	Lgarlask
2021	Trial	Colonia	-34.33644	-57.71259	72	Tbioaudaz
2021	Farmer Field	Soriano	-33.678	-57.56776	131	
2021	Farmer Field	Soriano	-33.30554	-58.05686	54	
2021	Trial	Colonia	-34.33644	-57.71259	72	Sy110
2021	Trial	Colonia	-34.33644	-57.71259	72	Nandubay
2021	Trial	Colonia	-34.33644	-57.71259	72	Genesis5.28
2021	Trial	Rio Negro	-32.68305	-57.65959	83	
2021	Trial	Rio Negro	-32.68305	-57.65959	83	LG_Arlask
2021	Trial	Rio Negro	-32.68305	-57.65959	83	LG_Arlask
2021	Trial	Rio Negro	-32.68305	-57.65959	83	LE_2375
2021	Trial	Rio Negro	-32.68305	-57.65959	83	LE_2466
2021	Farmer Field	Colonia	-34.34632	-57.37861	23	
2021	Farmer Field	Colonia	-34.21795	-56.8822	104	
2021	Farmer Field	Colonia	-33.886	-57.42167	170	
2021	Farmer Field	Colonia	-33.92661	-57.71542	125	
2021	Farmer Field	Colonia	-33.91192	-57.77362	117	Ceibo
2021	Farmer Field	Soriano	-33.84344	-57.99096	82	
2021	Farmer Field	Soriano	-33.84344	-57.99096	82	
2021	Farmer Field	Soriano	-33.84344	-57.99096	82	
2021	Farmer Field	Colonia	-33.79974	-58.10938	70	
2021	Farmer Field	Colonia	-33.86845	-58.30331	47	

2021	Farmer Field	Soriano	-33.70729	-58.33858	26	Berretin	
2021	Farmer Field	Colonia	-34.06558	-58.16997	49		
2021	Farmer Field	Colonia	-32.48164	-58.0044	33	SY_300	
2021	Trial	Paysandú	-32.3766	-58.05849	47	Baguette_11	
2021	Trial	Paysandú	-32.3766	-58.05849	47	Tijereta	
2021	Farmer Field	Rio Negro	-32.7617	-57.5812	73		
2021	Farmer Field	Rio Negro	-32.82484	-57.52861	51		
2021	Farmer Field	Flores	-33.60808	-56.99622	135		
2021	Farmer Field	Rio Negro	-33.06819	-57.24065	59		
2021	Farmer Field	Soriano	-33.85025	-57.33375	125		
2021	Farmer Field	Colonia	-33.89771	-57.41535	161		
2021	Farmer Field	Colonia	-34.05325	-58.21442	10		
2021	Farmer Field	Colonia	-34.3379	-57.75722	37		
2021	Trial	Rio Negro	-32.68305	-57.65959	87		
2021	Trial	Rio Negro	-32.68305	-57.65959	87	LE2466	
2021	Trial	Rio Negro	-32.68305	-57.65959	87		
2021	Trial	Rio Negro	-32.68305	-57.65959	87		
2021	Trial	Rio Negro	-32.68305	-57.65959	87		
2021	Trial	Colonia	-34.33644	-57.71259	72	Nandubay	
2021	Trial	Colonia	-34.33644	-57.71259	72		
2021	Trial	Colonia	-34.33888	-57.72702	80	Baguette620	
2021	Trial	Colonia	-34.33888	-57.72702	80	LG_Arlask	
2021	Trial	Colonia	-34.33888	-57.72702	80	Ñandubay	
2021	Trial	Colonia	-34.33888	-57.72702	80	Genesis5.28	
2021	Trial	Colonia	-34.33888	-57.72702	80	Ceibo	
2021	Trial	Colonia	-34,339,500	-57.69539	65	Morocco	
2021	Trial	Colonia	-34,339,500	-57.69539	65	Morocco	UY169
2021	Trial	Colonia	-34,339,500	-57.69539	65	LE2407	UY170
2021	Trial	Colonia	-34,339,500	-57.69539	65	LE2145	UY171
2021	Trial	Colonia	-34,339,500	-57.69539	65	LE2418	UY172
2021	Trial	Colonia	-34,339,500	-57.69539	65	LE2469	UY173

- **Supplementary material 2**. Allele sizes for each SSRs and genetic group assignment of samples collected in Uruguay during 2017-2021.

	Multiplex-1																				Multiplex-2																Single		Single		
Flourescence =>	6FAM		6FAM		VIC		VIC		NED		NED		PET		PET		6FAM		6FAM		6FAM		6FAM		VIC		VIC		NED		NED		PET		PET		PET		NED		
Motif size =>	3bp		3bp		3bp		3bp		3bp		2bp		2bp		3bp		3bp		3bp		3bp		2bp		3bp		2bp		2bp		2bp		2bp		3bp		2bp		2bp		**Genetic**
Isolate ID	NRJO4		NRJO24		NRJN12		NRJN8		NRJN13		NRJN3		NRJN11		NRJN6		NRJO21		NRJN10		NRJO18		NWU-6		NRJO20		NRJN4		NRJN9		NRJN5		NWU-12		NRJO27		NRJN2		**group**
UY155	199	199	284	293	196	196	307	316	147	150	336	336	176	182	315	318	170	170	224	224	331	331	210	210	284	287	255	255	332	332	226	226	332	332	230	242	nd	nd	PstS7
UY163	199	199	284	293	196	196	307	316	147	150	336	336	176	182	315	318	170	170	224	224	331	331	210	210	284	287	255	255	332	332	226	226	332	332	nd	nd	169	169	PstS7
UY165	199	199	284	293	196	196	307	316	147	150	336	336	176	182	315	318	170	170	224	224	331	331	210	210	284	287	255	255	332	332	226	226	332	332	242	242	nd	nd	PstS7
UY173	199	199	284	293	196	196	307	316	147	150	336	336	176	182	315	318	170	170	224	224	331	331	210	210	284	287	255	255	332	332	226	226	332	332	230	230	169	169	PstS7
UY161	199	202	275	284	196	196	307	307	147	150	336	336	176	176	315	318	170	170	224	224	331	337	210	210	284	287	255	255	332	334	226	230	323	332	232	232	183	183	PstS10
UY166	199	202	275	284	196	196	307	307	147	150	336	336	176	176	315	318	170	170	224	224	331	337	210	210	284	287	255	255	332	334	226	230	323	332	232	232	183	183	PstS10
UY147	199	205	272	284	196	196	304	307	150	150	336	336	172	176	315	315	170	176	224	224	331	337	210	210	287	287	255	257	332	332	226	228	323	332	230	230	169	169	PstS13
UY148	199	205	272	284	196	196	304	307	150	150	336	336	172	176	315	315	170	176	224	224	331	337	210	210	287	287	255	257	332	332	226	228	323	332	230	230	169	169	PstS13
UY149	199	205	272	284	196	196	304	307	150	150	336	336	172	176	315	315	170	176	224	224	331	337	210	210	287	287	255	257	332	332	226	228	323	332	nd	nd	169	169	PstS13
UY150	199	205	272	284	196	196	304	307	150	150	336	336	172	176	315	315	170	176	224	224	331	337	210	210	287	287	255	257	332	332	226	228	323	332	230	230	169	169	PstS13
UY151	199	205	272	284	196	196	304	307	150	150	336	336	172	176	315	315	170	176	224	224	331	337	210	210	287	287	255	257	332	332	226	228	323	332	230	230	169	169	PstS13
UY152	199	205	272	284	196	196	304	307	150	150	336	336	172	176	315	315	170	176	224	224	331	337	210	210	287	287	255	257	332	332	226	228	323	332	230	230	169	169	PstS13
UY153	199	205	272	284	196	196	304	307	150	150	336	336	172	176	315	315	170	176	224	224	331	337	210	210	287	287	255	257	332	332	226	228	323	332	230	230	169	169	PstS13
UY154	199	205	272	284	196	196	304	307	150	150	336	336	172	176	315	315	170	176	224	224	331	337	210	210	287	287	255	257	332	332	226	228	323	332	230	230	169	169	PstS13
UY156	199	205	272	284	196	196	304	307	150	150	336	336	172	176	315	315	170	176	224	224	331	337	210	210	287	287	255	257	332	332	226	228	323	332	230	230	169	169	PstS13
UY157	199	205	272	284	196	196	304	307	150	150	336	336	172	176	315	315	170	176	224	224	331	337	210	210	287	287	255	257	332	332	226	228	323	332	230	230	169	169	PstS13
UY158	199	205	272	284	196	196	304	307	150	150	336	336	172	176	315	315	170	176	224	224	331	337	210	210	287	287	255	257	332	332	226	228	323	332	230	230	169	169	PstS13
UY159	199	205	272	284	196	196	304	307	150	150	336	336	172	176	315	315	170	176	224	224	331	337	210	210	287	287	255	257	332	332	226	228	323	332	230	230	169	169	PstS13
UY160	199	205	272	284	196	196	304	307	150	150	336	336	172	176	315	315	170	176	224	224	331	337	210	210	287	287	255	257	332	332	226	228	323	332	230	230	169	169	PstS13
UY167	199	205	272	284	196	196	304	307	150	150	336	336	172	176	315	315	170	176	224	224	331	337	210	210	287	287	255	257	332	332	226	228	323	332	230	230	169	169	PstS13
UY168	199	205	272	284	196	196	304	307	150	150	336	336	172	176	315	315	170	176	224	224	331	337	210	210	287	287	255	257	332	332	226	228	323	332	230	230	169	169	PstS13
UY170	199	205	272	284	196	196	304	307	150	150	336	336	172	176	315	315	170	176	224	224	331	337	210	210	287	287	255	257	332	332	226	228	323	332	230	230	169	169	PstS13
UY171	199	205	272	284	196	196	304	307	150	150	336	336	172	176	315	315	170	176	224	224	331	337	210	210	287	287	255	257	332	332	226	228	323	332	230	230	169	169	PstS13
UY172	199	205	272	284	196	196	304	307	150	150	336	336	172	176	315	315	170	176	224	224	331	337	210	210	287	287	255	257	332	332	226	228	323	332	230	230	169	169	PstS13
UY162	199	205	272	284	196	196	304	307	150	150	336	336	172	176	315	315	170	176	224	224	331	337	210	210	287	287	255	257	332	332	226	228	323	332	230	230	169	169	PstS13
UY164	199	205	272	284	196	196	304	307	150	150	336	336	172	176	315	315	170	176	224	224	331	337	210	210	287	287	255	257	332	332	226	228	323	332	230	230	169	169	PstS13
UY169	199	205	272	284	196	196	304	307	150	150	336	336	172	176	315	315	170	176	224	224	331	337	210	210	287	287	255	257	332	332	226	228	323	332	230	230	169	169	PstS13

- **Supplementary material 3**. Infection types on *Pst* differential lines of *Pst* isolates collected in Uruguay during 2017-2021.

Variety =>	Chinese 166	Kalyansona	Vilmorin 23	Hybrid 46	Suwon	Heines Kolben	Avocet *Yr6*	Lee	Avocet *Yr8*	Avocet *Yr9*	Moro	Cortez	VPM1	Avocet *Yr17*	TP 981	Opata	Avocet *Yr32*	Spaldings Prolific	Avocet *S*	Ambition	Benchmark	Kalmar	KWS Zyatt	Nemo	Virulence phenotype	Race
Yr-genes =>	*1*	*2,+*	*3,+*	*4,+*	*Su*	*6,+*	*AvS,6*	*7,+*	*AvS,8*	*AvS,9*	*10*	*15*	*17,+*	*AvS,17*	*25,+*	*18,27,+*	*AvS,32*	*Sp,25,+*	*AvS*	*Amb*						
UY155	8	8	6,7	5,6,7	8	8	8	8	1	8	1	1	7	7	7	3,4	7	7	8	7	7	4,5,6	7	3,4,5	1,2,3,4,6,7,-,9,-,-,17,25,-,32,Sp,AvS,Amb	Warrior
UY163	8	8	6,7	5,6	8	8	7	8	1	8	1	1	7,8	8	7	3,4	8	7	8	8	6,7	3,4,5	7	4,5,6	1,2,3,4,6,7,-,9,-,-,17,25,-,32,Sp,AvS,Amb	Warrior
UY165	8	7	6,7	5,6	8	8	8	8	0	8	1	1	7	7	8	3	7	7	8	8	8	4,5,6	7	4,5,6	1,2,3,4,6,7,-,9,-,-,17,25,-,32,Sp,AvS,Amb	Warrior
UY173	7	7	7	5,6	8	8	8	8	1	8	1	1	7	7	7	3,4	8	7	8	7	7	5,6,7	7	4,5	1,2,3,4,6,7,-,9,-,-,17,25,-,32,Sp,AvS,Amb	Warrior
UY161	8	8	8	5,6	8	8	8	8	2,3	8	2,3	1	8	8	7	nd	7	6,7	8	3,4	7	3,4,5	6,7	2,3	1,2,3,4,6,7,-,9,-,-,17,25,-,32,Sp,AvS,-	Benchmark
UY166	7	8	6,7	5	7	7	7	7	1	8	1	1	6,7	7	7	3,4	7	7	7	nd	7	4,5,6,7	6,7	3,4,5	1,2,3,4,6,7,-,9,-,-,17,25,-,32,Sp,AvS,-	Benchmark
UY147	1	8	1	4,5	8	8	7	8	8	8	2,3	0,1	1	2,3	3,4,3	4	2,3	5,6,7	8	3,4	0,1	0,1	1	1,2	-,2,-,-,6,7,8,9,-,-,-,-,-,-,-,AvS,-	Triticale2015
UY148	1	7	1	2,3,4	7	8	7	8	8	7	2,3	1,2	1,2	2,3	3,4	4,5,6	2,3	5,6,7	7	4,5	1	0,1	1	2	-,2,-,-,6,7,8,9,-,-,-,-,-,-,-,AvS,-	Triticale2015
UY149	1	8	1,2	3,4	8	8	7	8	8	8	2,3	1	1	2,3	3,4	5,6	3,4	5,6,7	7	4,5,6	0,1	0,1	0	1,2	-,2,-,-,6,7,8,9,-,-,-,-,-,-,-,AvS,-	Triticale2015
UY150	1	8	1,2	3,4	7	8	8	8	8	8	2	1,2	1	2,3	3,4,5	4	3,4	5,6,7	8	4,5	2	1	1	1	-,2,-,-,6,7,8,9,-,-,-,-,-,-,-,AvS,-	Triticale2015
UY151	1	8	1	3,4	8	8	7	8	8	7,8	2,3	1	1,2	2,3	3	3,4,5	4	6,7	7	5,6	0,1	0,1	1	1,2	-,2,-,-,6,7,8,9,-,-,-,-,-,-,-,AvS,-	Triticale2015
UY152	1	8	1,2	4	8	8	8	8	8	8	3	1	1,2	1,2	3,4	4	2,3,4	5,6,7	8	5,6	1	1	1	1,2	-,2,-,-,6,7,8,9,-,-,-,-,-,-,-,AvS,-	Triticale2015
UY153	1	8	1	3,4,5	8	8	8	8	8	8	2,3	0,1	1	1,2	3	3,4,5	4,5	5,6,7	8	5,6,7	1	1	1,2	1,2	-,2,-,-,6,7,8,9,-,-,-,-,-,-,-,AvS,-	Triticale2015
UY154	1	8	2,3	3,4	8	8	8	8	8	8	2,3	1,2	1,2	1,2	3,4	3,4,5	4	5,6,7	8	4,5,6	1	0,1	1,2	1	-,2,-,-,6,7,8,9,-,-,-,-,-,-,-,AvS,-	Triticale2015
UY156	1	8	2	3,4	7,8	8	8	8	8	8	2	1	1,2	8	3,4	5,6	8	5,6,7	8	4,5	1	1	1,2	1,2	-,2,-,-,6,7,8,9,-,-,17,-,-,32,-,AvS,-	Triticale2015
UY157	1	8	1	3,4	8	8	8	8	8	8	3,4,5	1	1,2	7	3,4	4,5	7	5,6,7	8	5,6	1	1	1	1,2	-,2,-,-,6,7,8,9,-,-,17,-,-,32,-,AvS,-	Triticale2015
UY158	1	8	2	4,5	7	8	8	8	8	8	2	2	2	7,8	2	4,5	8	5,6,7	8	5,6	1	1	nd	1	-,2,-,-,6,7,8,9,-,-,17,-,-,32,-,AvS,-	Triticale2015
UY159	1	8	1,2	3,4	7	8	8	8	8	8	2	1,2	1,2,3	7,8	4,5	6	8	5,6,7	8	5,6	1	1	1	1,2	-,2,-,-,6,7,8,9,-,-,17,-,-,32,-,AvS,-	Triticale2015
UY160	1	8	1	3,4	7	7	8	8	7	8	2	1,2	1	8	5	6	8	5,6,7	8	5,6	1,2	1	1,2	1,2	-,2,-,-,6,7,8,9,-,-,17,-,-,32,-,AvS,-	Triticale2015
UY162	1	7	1	4,5	7	8	7	8	8	8	2,3	1	1,2	7	3,4	5	7	5,6,7	8	4,5,6	0,1	0,1	0,1	1,2	-,2,-,-,6,7,8,9,-,-,17,-,-,32,-,AvS,-	Triticale2015
UY164	1	8	1	3,4	8	8	8	8	8	8	2,3	1	1,2	7	3,4	6	8	5,6,7	8	4,5	1	0,1	1	1,2	-,2,-,-,6,7,8,9,-,-,17,-,-,32,-,AvS,-	Triticale2015
UY167	1	8	1,2	3,4	7,8	8	8	8	8	8	2,3	2	1,2	8	3,4	5	8	5,6,7	8	5,6	0,1	0,1	1	1,2	-,2,-,-,6,7,8,9,-,-,17,-,-,32,-,AvS,-	Triticale2015
UY170	1	8	1	3,4	7	7	7	7	7	7	2,3	1	1	7	2,3	6	7	5,6,7	7	4,5	1	0,1	1	2	-,2,-,-,6,7,8,9,-,-,17,-,-,32,-,AvS,-	Triticale2015
UY171	1	8	1,2	4	8	8	8	8	8	8	2,3,4	1	1	7	3	5	7	5,6,7	7	5,6	0,1	0,1	1	1,2	-,2,-,-,6,7,8,9,-,-,17,-,-,32,-,AvS,-	Triticale2015
UY172	1	8	1	2,3,4	8	8	8	8	8	8	2,3	1,2	1,2	7	2,3	5,6	7	5,6,7	7	4,5	0,1	0,1	1	1	-,2,-,-,6,7,8,9,-,-,17,-,-,32,-,AvS,-	Triticale2015
UY168	1	8	1,2	2,3,4	7,8	7	8	8	8	8	3	1	1,2	7	3,4	7	7,8	6	8	5,6	1	0,1	1	1,2	-,2,-,-,6,7,8,9,-,-,17,-,27,32,-,AvS,-	Triticale2015

Note. Race typing was performed using a 0–9 infection type scale (McNeal et al. 1971), scores between 7 and 9 indicate compatibility (virulence), and scores equal to or below 6 indicate incompatibility (avirulence)

3. Roya estriada de trigo en Uruguay: entendiendo la base genética de la resistencia en un panel de líneas de mejoramiento y comericales

3.1. Resumen

La roya estriada del trigo (RE), causada por *Puccinia striiformis* f. sp. *tritici* (*Pst*), es una de las enfermedades más devastadoras que afectan al trigo en el mundo. Desde el año 2000, la RE se ha expandido hacia regiones donde anteriormente no se consideraba una enfermedad económicamente importante. La utilización de cultivares resistentes a RE sigue siendo la estrategia de control más efectiva y sostenible. En este estudio se conformó un panel de mapeo diverso con el objetivo de i) identificar regiones genómicas asociadas con la resistencia a RE mediante estudios de asociación de genoma completo (GWAS) y ii) evaluar la precisión de modelos de predicción genómica (PG) para la resistencia a RE. El panel, compuesto por 366 líneas de trigo que incluyen germoplasma del INIA-Uruguay y de otros programas de mejoramiento, fue fenotipado bajo inoculaciones artificiales a campo durante 2021 y 2022, y en etapa de plántula con las mismas dos razas de *Pst* empleadas para las inoculaciones de campo. El GWAS identificó ocho regiones genómicas asociadas con resistencia en campo, localizadas en los cromosomas 1B, 2B (tres regiones), 5B (dos regiones), 5D y 7B, que explicaron entre un 4,9 % y un 21,2 % de la variabilidad fenotípica. Ninguna de estas regiones se asoció con resistencia en plántula frente a la raza triticale2015b, la raza más virulenta y ampliamente distribuida, lo que indica que los QTL confieren resistencia en planta adulta. Además, estas regiones no correspondieron con genes *Yr* previamente mapeados. Se identificaron dos QTL en los cromosomas 2D y 3A asociados con resistencia en plántula a la raza triticale2015a, aunque no contribuyeron a la resistencia en planta adulta. Los modelos PG lograron una capacidad de predicción promedio de 0,64, lo que destaca su potencial para acelerar la selección de líneas resistentes. Estos hallazgos proporcionan información valiosa sobre la base genética de la resistencia a RE y ofrecen herramientas sólidas para fortalecer los esfuerzos de mejoramiento genético para la resistencia a RE en trigo.

Palabras clave: *Puccinia striiformis*, mapeo asociativo, resistencia genética cuantitativa, predicción genómica

Wheat yellow rust in Uruguay: understanding the genetic resistance in a panel of breeding and commercial germplasm

3.2. Key message

Eight QTL conferring additive APR to YR were identified in wheat germplasm using GWAS. The high accuracy of GP models supports the feasibility of accelerating breeding for YR resistance.

3.3. Abstract

Wheat yellow rust (YR), caused by *Puccinia striiformis* f. sp. *tritici* (*Pst*), is among the most devastating diseases affecting wheat worldwide. Since 2000, YR has expanded into regions where it was previously not considered an economically important disease. The deployment of YR-resistant cultivars remains the most effective and sustainable control strategy. We assembled a diverse mapping panel (i) to identify genomic regions associated with YR resistance using genome-wide association studies (GWAS), and (ii) assess the prediction accuracy of genomic prediction (GP) models for YR resistance. The panel of 366 wheat lines, including germplasm from INIA-Uruguay and other breeding programs, was phenotyped under artificial field inoculations in 2021 and 2022, and at the seedling stage using the same two *Pst* races used for field inoculations. GWAS identified eight genomic regions associated with field resistance, located on chromosomes 1B, 2B (three regions), 5B (two regions), 5D, 7B, explaining 4.9 to 21.2% of the phenotypic variability. None of these regions were identified with seedling resistance to race *Triticale2015b*, the most widely virulent race, indicating that they conferred adult-plant resistance. Moreover, these regions did not correspond to previously mapped *Yr* genes. Two QTL on 2D and 3A were identified at the seedling stage to race *Triticale2015a* but did not contribute to field resistance. GP models achieved an average prediction ability of 0.64, highlighting their potential for accelerating the selection of resistant lines. These findings provide valuable insights into the genetic basis of YR and offer robust tools for enhancing YR resistance breeding efforts in wheat.

Keywords: *Puccinia striiformis*, genome-wide association study (GWAS), quantitative disease resistance, genomic prediction (GP).

3.4. Introduction

Wheat (*Triticum aestivum* L.) ranks among the top three staple crops globally, alongside rice and maize, serving as a critical source of nutrition not only for its caloric contribution but also for its protein, vitamin, and fiber content, which are essential for human health (Bao & Malunga, 2022). In recent years, global wheat production has been increasingly threatened by the emergence of new, more aggressive pathogen strains (Hovmøller et al. 2008) adapted to diverse environments (Milus et al., 2006). Of particular concern is *Puccinia striiformis* Westend. f. sp. *tritici* (*Pst*), the causal agent of wheat yellow rust (YR) (Hovmøller et al., 2011; Sørensen et al., 2014). Historically, *Pst* has posed a significant challenge primarily in cooler climates; however, since 2000, the pathogen has shown increased aggressiveness (Hovmøller et al., 2008) and tolerance to higher temperatures, leading to its spread in regions previously considered too warm for its establishment (Milus et al., 2009; Wellings, 2007). Additionally, distant geographic areas have reported YR epidemics, either as new incursions in previously unaffected regions or as re-emergences of novel, more widely virulent strains (Bahri et al., 2009; Hovmøller et al., 2023, Riella et al., 2024). As a result, YR has become one of the most severe and damaging diseases affecting common wheat globally, with potential yield losses reaching up to 100% under high disease pressure (Ali et al., 2014).

Genetic resistance to rust diseases is generally categorized into two types: all-stage resistance (ASR), also known as seedling resistance, which is expressed throughout the plant's lifecycle, and adult-plant resistance (APR) (X. Chen, 2013). ASR is typically qualitative, conferred by one or a few major genes with largely dominant effects which follows a “gene-for-gene” relationship between host and pathogen (Flor, 1955), in which each host gene provides resistance against pathogen races that carry the complementary avirulence gene. This type of resistance is race-specific, mediated by hypersensitive responses (Ayliffe et al., 2008), but generally provides short-lived effectiveness, as extensive use over large areas selects for new,

virulent *Pst* races. Conversely, APR is effective in post-seedling stages, involves minor additive genes and confers partial resistance or "slow rusting" resistance, characterized by prolonged latent periods, fewer and smaller pustules, and reduced spore production (Bhavani et al., 2011; Singh et al., 2000; Singh, Hodson, et al., 2011). APR is generally race non-specific and considered durable; the accumulation of three to five minor APR genes can confer near-complete immunity (Singh et al., 2000). At least 87 YR resistance genes have been reported to date, but less than 30% confer APR (McIntosh, 2024). The limited number of reported APR genes, coupled with their effects often being influenced by environmental factors and genetic background (D. Liu et al., 2022; Silva et al., 2015; C. Yuan et al., 2020), highlights the need to identify genomic regions associated with YR APR resistance in locally adapted materials. The discovery of new genomic regions is essential for making better use of the genetic diversity available and improving YR resistance effectivity and durability.

The identification of molecular markers associated with YR resistance is a promising approach to accelerate the development of resistant cultivars by identifying and pyramiding resistance genes within the same genotype. Among molecular markers, single nucleotide polymorphisms (SNPs) have gained widespread use due to their abundance across the genome and the significant reduction in genotyping costs in recent years (Crossa et al., 2017). One of the most utilized strategies for identifying the genetic basis of resistance to diseases is the genome-wide association study (GWAS). GWAS leverages linkage disequilibrium within a population to investigate associations between molecular markers and phenotypic traits. A statistically significant association suggests that the marker is linked to a genomic region contributing to the trait of interest, known as a quantitative trait locus (QTL) (Pritchard et al., 2000; C. Zhu & Yu, 2009). GWAS has been successfully applied in wheat, identifying over 160 QTL across 49 regions on 21 chromosomes associated with YR resistance (Maccaferri et al., 2015; Rosewarne et al., 2013; F.-P. Yuan et al., 2018). While GWAS enables fine-scale genome mapping using genetically diverse populations with extensive recombination histories, it also faces limitations, such as reduced power to detect rare allelic variants and the need to control for false positives, where marker-QTL associations are not due to physical linkage (Brachi et al., 2010;

Wallace et al., 2014; Zuk et al., 2014). Moreover, the identification of QTL is often influenced by genotype-by-environment interactions, emphasizing the importance of detecting QTL that remain stable across different environments to ensure their utility in breeding programs (Gutiérrez et al., 2015).

Genomic prediction (GP) models using whole-genome data generally have higher power to capture small-effect loci compared to marker-assisted selection (MAS) (Heffner et al., 2009), particularly for complex traits controlled by many minor genes (Bernardo, 2008; Cerrudo et al., 2018; Lorenz et al., 2011; Mayor & Bernardo, 2009). GP leverages all genome-wide markers and phenotypic data to estimate genetic values and select candidates based on predicted genetic merit (Bernardo, 2016; Crossa et al., 2017; Mrode, 2014; Schmid & Bennewitz, 2017). GP requires a training population that has been genotyped and phenotyped to calibrate a model, which can then predict genetic values of a selection population based solely on genotypic information (Bassi et al., 2016). GP is expected to reduce the time and cost required for cultivar development since annual genetic gains using GP are predicted to be two to three times higher than those achieved through conventional phenotypic selection due to shortened breeding cycles and increased selection accuracy (Crossa et al., 2017; Jannink et al., 2010). GP for disease resistance in crops has been applied in numerous studies (Poland & Rutkoski, 2016), particularly for quantitatively inherited traits, with wheat rusts being among the most studied systems (Daetwyler et al., 2014; Muleta et al., 2017; Ornella et al., 2017; Rutkoski et al., 2014, 2015, 2016). GP in wheat rust resistance breeding could accelerate selection cycles and help pyramiding APR genes (Rutkoski et al., 2011).

3.4.1. Objective

With the aim of contributing to the sustainability of wheat production through the development of YR resistant cultivars, this study focuses on (i) to identify genomic regions associated with YR resistance in a diverse wheat germplasm panel through GWAS, and (ii) to assess the prediction accuracy of GP models for YR resistance in wheat lines.

3.5. Materials and methods

3.5.1 Plant material

The GWAS and GP panel consisted of 366 diverse spring bread wheat genotypes, representing the most currently and historically important wheat cultivars and advanced breeding lines of Uruguay. The panel includes 172 lines from the INIA Resistant Germplasm Development Program (INIA-RGDP), developed to introgress APR to leaf rust, primarily from CIMMYT germplasm, and to address other prevalent diseases in Uruguay prior to 2017, 117 lines from the INIA-Wheat Breeding program (INIA-WBP), including advanced and elite lines as well as released varieties, representing a century of wheat breeding in the country, 73 cultivars from other breeding programs sown in Uruguay, and four check lines, selected for their diversity in maturity date and susceptibility to YR (**Table S1**). For most of the lines present in the panel there was not previous YR phenotypic information since the disease was not present prior to 2017. Cultivar Morocco was used as a susceptible check in field and seedling trials but was not included in the GWAS and GP panel.

3.5.2. Phenotypic trait evaluation

3.5.2.1. Field yellow rust and heading date phenotyping

Field experiments were conducted at INIA La Estanzuela Experimental Station, (latitude 34.3°S, longitude 57.7°W, elevation 70 masl), Colonia, Uruguay, during two consecutive crop seasons (2021 and 2022). Sowing dates were May 14th 2021 and May 4th 2022. The experimental design consisted of an alpha lattice resolvable incomplete block design with three replications. Plots consisted of single 1 m long rows 0.30 m apart. Spreader rows of a mixture of susceptible cultivars (Morocco, Avocet S, Fuste, Algarrobo, Ceibo, and Onix) were sown perpendicular to all plots to ensure the presence and even distribution of the disease. Artificial inoculations were performed on the spreader rows with a mixture of the two most prevalent races in previous years (*Triticale2015a* and *b*), both races belonging to the *PstS13* genetic group (Riella et al., 2024). Three and six inoculations with a suspension of inoculum in lightweight mineral oil Soltrol 170 (Phillips Petroleum Co., Borger,

TX) were performed in 2021 and 2022 respectively, between July 20 and August 20. In 2021 the experiment was rainfed, meanwhile, in 2022, due to dry weather conditions, the trial was irrigated using a sprinkler system. Days to heading for each plot were calculated as the days from seedling emergence to heading date. Heading date was recorded based on the crop ontology trait CO_321:0000840 as the date when 50% of the head emerged in 50% of the plot (https://cropontology.org).

First disease assessment took place when the susceptible check Morocco displayed a disease severity (DS) of at least 50% and continued for six times at 7-12 days intervals. For each evaluation, DS was visually scored as the percentage of infected tissue (0 – 100%). The six DS obtained for each plot were combined in a single value as the area under the disease progress curve (AUDPC) according to following formula:

$$AUDPC = \sum_{i=1}^{N_i-1} \frac{(y_i + y_{i+1})}{2}(t_{i+1} - t_i)$$

where AUDPC for each plot is given by y_i rust DS at the time of recording t_i, y_{i+1} rust infection rate at the time of recording t_{i+1}, N the number of records to assess the DS from 1 to 6.

3.5.2.2. Statistical analyses for field yellow rust phenotyping

The phenotypic data were analyzed using R software (R Core Team, 2024). To evaluate quality of each trial and accurately estimate the phenotypic means, the AUDPC data was analyzed independently for each year (2021 and 2022) fitting a linear mixed model with the lme function of the lme4 package (Bates et al., 2015). The statical model, followed the experimental design and including days to heading as a covariate, was:

$$y_{ijk} = \mu + \tau_i + \beta_j + \gamma_{k(j)} + \lambda(x_{ijk} - \bar{x}) + \varepsilon_{ijk} \quad \textbf{[1]}$$

where y_{ijk} represents the AUDPC (response variable) measured on the i-th wheat line in the j-th complete replicate, in the k-th incomplete block, μ the overall mean, τ_i the relative effect of the i-th wheat line, β_j the effect of the j-th complete replicate, and $\gamma_{k(j)}$ the random effect of the k-th block nested in the j-th complete replicate which is assumed to be random with normal distribution centered on zero and with constant

variance (σ_γ^2), $\lambda(x_{ijk} - \bar{x})$ is a covariate term for days to heading correction, where x_{ijk} is days to heading, $\bar{x}$ the mean for days to heading, and λ the regression coefficient associated with the covariate. Given that the panel consists of highly diverse lines with important variability in maturity time, days to heading was included as a covariate in the model. This adjustment aimed to minimize potential noise that could lead to the identification of regions associated with phenology rather than YR resistance. This model assumes that the errors (ε_{ijk}) are independent random variables, normally distributed with zero mean and constant variance (σ_ε^2), and that there is no interaction between blocks and treatments (Di Rienzo et al., 2009).

To select the best model for estimating phenotypic means while incorporating information from the experimental design, spatial effects, and covariate (days to heading), the fit of the baseline model (model **[1]**) was compared to alternative models. These alternatives included models that incorporated the spatial position of the plot in the field as an additional factor (e.g., column effects). They also considered models that assumed different variance-covariance structures for the experimental errors, such as Gaussian, spherical, and exponential models, to account for potential correlations among the experimental units. Model comparisons were conducted separately for each year using fitting criteria such as the Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC), as well as performance indicators, including the correlation between observed and predicted values and broad-sense heritability (H^2) estimates. Broad-sense heritability was calculated for each trial, the variance components (σ_g^2 genotypic variance and σ_{error}^2 error variance) were estimated from equations **[1]** and **[2]** with genotypes as random effects, using equation $H^2 = \sigma_g^2/(\sigma_g^2 + \sigma_{error}^2/rep)$. The error variance was corrected for the number of replicates. Due to the strong correlation between environments and similar accuracy between the best models each year, the adjusted means (Best Linear Unbiased Estimators, BLUEs) for AUDPC for each genotype were obtained and estimated by fitting a combined model using data from both years. This model is similar to model **[1]** except that it includes the year effect (α_l) as follow:

$$y_{ijkl} = \mu + \tau_i + \alpha_l + \beta_{j(l)} + \gamma_{k(jl)} + \lambda(x_{ijkl} - \bar{x}) + \varepsilon_{ijkl} \qquad \textbf{[2]}$$

3.5.3. Seedling yellow rust phenotyping

Greenhouse trials were conducted to determine resistance at the seedling stage of the panel, which indicates the presence of ASR genes. The phenotyping was conducted during 2023 at INIA La Estanzuela Experimental Station, using the two *Pst* races used in field inoculations, *Triticale2015a* and *Triticale2015b* (Riella et al., 2024). Eight seeds of each genotype were sown in plastic trays with substrate (mixture of one third soil, one third vermiculite and one third seedbed substrate: Potting mix, Bioterra), 25 genotypes per tray, plus Morocco as the susceptible check. Fully expanded leaves (8–10 days after sowing) were inoculated by spraying urediniospores suspended in Soltrol 170, incubated at 10ºC in a dew chamber overnight and then kept in the greenhouse at 15–25 °C with supplemental lighting. Infection type (IT) was recorded for each genotype 15–20 days after inoculation based on a 0–9 scale (McNeal et al., 1971) (crop ontology CO_321:0000606), lines with IT values of 0-3 were considered resistant, 4-6 intermediate and 7-9 susceptible. Two complete replicates were used for each genotype and race. The IT adjusted means for each genotype were obtained by fitting a linear model with the lm function (R Core Team, 2024). The statistical model used was $y_{ij} = \mu_i + \varepsilon_{ij}$ where y_{ij} represents the IT value (response variable) measured on the i-th wheat line in the j-th replicate, μ the overall mean, this model assumes that the errors (ε_{ij}) are independent random variables, normally distributed with zero mean and constant variance (σ_ε^2). For each inoculation, a tray with the set of YR differential lines was included in order to corroborate the identity and purity of the race used.

3.5.4. Genotypic data

The genomic DNA of the 366 wheat lines was isolated from fresh leaves of 20-day old plants by the CTAB method (Saghai-Maroof et al., 1984). Genotyping was performed by Genotyping-by-sequencing (GBS) using an Illumina 150 bp paired-end sequencer at the University of Wisconsin-Madison DNA Sequencing Facility. Analysis of the genotypic data first involved SNP calling using the TASSEL GBSv2 pipeline (Glaubitz et al., 2014), and the cv Chinese Spring as the reference genome (IWGSC CS RefSeq v2.1) (T. Zhu et al., 2021). SNPs with >80% missing data and

SNPs with a minor allele frequency (MAF) less than 0.01 were also removed. Missing data were imputed with BEAGLE 5.4 (Browning et al., 2018). Data were transformed to numerical coding (0, 1, and 2 for homozygotes for the major allele, heterozygotes, and homozygotes for the minor allele, respectively) for analyses, obtaining the complete matrix with a total of 156,032 SNPs.

In addition, the presence of APR genes *Yr18* (*TCCIND*, Rasheed et al. 2016), *Yr29* (*SNP1G22,* Lagudah et al., pers. comm.), and *Yr46* (*csSNP856,* Forrest et al. 2014) within the wheat panel was verified based on competitive allele-specific PCR (KASP) assays. These assays were performed at INIA-Las Brujas lab following CIMMYT protocols (Dreisigacker et al., 2016).

3.5.5. Population structure and Linkage disequilibrium (LD)

The genetic structure of the population was studied using the Admixture program (Alexander et al., 2009) to determine the number of subpopulations (K). The ΔK was observed as the number of subpopulations increased (K = 0 to K = 20). Additionally, with the SNP matrix obtained in the previous step, Euclidean genetic distances were calculated between the panel lines, from which a principal co-ordinate analysis (PCoA) and a genetic distance plot were created using the R package ade4 (Dray & Dufour, 2007). The extent of linkage disequilibrium (LD) in this association panel was calculated according to (J. Zhang et al., 2018), based on pairwise squared LD correlation coefficients (r^2) for all intrachromosomal SNP loci. Nonlinear model, described by Remington et al. (2001), was fitted to study the relation between r^2 and physical distances. To fit the non-linear model nls function in R was used (R Core Team, 2024). The physical distance at which LD fell below the r^2 thresholds determined according to Zhang et al. (2018)was used to define the confidence intervals of the QTL detected in the GWAS analysis.

3.5.6. Genome-wide association analysis between phenotype and genotype

GWAS was performed to identify genomic regions associated with YR resistance, using a matrix of 156,032 SNPs and 366 genotypes. The R package GWASpoly (Rosyara et al., 2016) was used to conduct the GWAS. GWAS was performed using mixed model with best linear unbiased estimates (BLUE) for AUDPC

(from join analysis of both years) as response variable, SNP coded as 0,1,2 as fixed effect and random polygenic effect to control for population structure, commonly known as the K model (Yu et al., 2006). This method uses a covariance matrix, effectively treating all markers as random effects. However, as this can lead to "proximal contamination" (Listgarten et al., 2012), where markers tested as fixed effects are also included as random effects, reducing model performance. The leave-one-chromosome-out (LOCO) approach (Yang et al. 2014) was used to improve accuracy by calculating covariance matrices for each chromosome using markers from other chromosomes. The results were summarized with Manhattan plots to visualize associations between SNPs and traits, utilizing functions in GWASpoly. Population structure was controlled by incorporating a kinship matrix, as implemented in GWASpoly, to avoid spurious associations. Quantile–quantile (QQ) plots were used as a visual criterion for assessing the model fit of the GWAS. To minimize the risk of false positives, GWASpoly applies corrections such as the false discovery rate (FDR). In this study, a significance threshold of FDR=0.1 was used.

QTL identified in this study were named following Boden et al. (2023), using the prefix "Q" for QTL, "Yr" for yellow rust, and "uy" to indicate Uruguay, followed by a hyphen and the corresponding chromosome number and genome and the chromosome arm (short: S, long: L). When more than one QTL were identified on the same chromosome arm, an additional number was added after a decimal point (Boden et al., 2023). Additionally, the chromosome arm (short: S, long: L) was also specified. A region was considered a QTL if it contained at least two markers in LD above the threshold. All significantly associated markers within each region were considered to define haplotypes. The physical position of the first and last markers above the threshold was defined as the start and end of the QTL, respectively. The p-value, effect, and percentage of explained variance for each QTL were obtained by fitting a separate linear regression model for each QTL. The regression model includes BLUE of AUDPC as the response variable. It is regressed on a dichotomous variable where one (1) was assigned to the favorable more resistant haplotype (lower AUDPC) and two (2) to the more susceptible haplotype (higher AUDPC).

To determine whether the significant SNPs detected in this study were located in the same position as previously reported *Yr* genes and resistance QTL, the physical locations of the identified genomic regions were compared with positional data from the most updated database of wheat rust resistance genes and QTL currently available in the literature (McIntosh, 2024; Tong et al., 2024).

To determine the effect of accumulation of favorable YR QTL alleles on AUDPC, wheat lines were grouped according to their number of favorable QTL alleles. The AUDPC means for each group were compared using a Tukey multiple comparisons test ($P < 0.05$).

3.5.7. Genomic prediction (GP)

We assessed the predictive ability of seven genomic prediction models with different assumptions regarding marker effect distributions. The first model, additive best linear unbiased predictor (A-BLUP), used only the pedigree-based relationship matrix without including genetic marker data. The pedigree matrix was created using the prepPed and makeA functions of the nadiv package (Wolak, 2012) based on parental information from INIA-WBP, however, information was missing for 44 of the commercial lines present in the panel (12% of the total). Subsequently, other models incorporating genetic information in different ways and assuming different marker effect distributions were tested. The models compared included two mixed models: ridge-regression best linear unbiased predictor (RR-BLUP), which uses information from all markers, genomic best linear unbiased predictor (G-BLUP), which leverages genetic distance between lines for predictions, and four types of Bayesian models: Bayesian A (BA), Bayesian B (BB), Bayesian C (BC), and Bayesian Lasso (BL).

To conduct the comparisons, a 10-fold cross-validation with 100 iterations was performed. This validation strategy involves randomly dividing the panel lines (366 lines) into 10 groups (with 36 or 37 lines); nine groups were used to train the model, and predictions were made for the lines in the tenth group. This process was repeated for each of the 10 groups over 100 iterations. All analyses were conducted in R software, using the BGLR package (Pérez & De Los Campos, 2014).

Predictive ability was estimated as the Pearson's correlation between observed and predicted values in each iteration. The mean squared error (MSE) was calculated as the difference between observed and predicted values. Additionally, in a second phase, identified QTL from the previous GWAS were sequentially added to the genomic prediction model as fixed effects, ordered by the amount of variance they explained. The performance of these models was then compared with the model excluding these fixed effects.

3.6. Results

3.6.1. Phenotypic traits

Field trials in both years had uniform infection levels, with high infection levels in check lines. The AUDPC values for the check cultivar Morocco in 2021 ranged between 5075 and 5260 among reps, and in 2022, between 6125 and 6475. The panel of 366 wheat lines displayed a continuous distribution of YR AUDPC values over the two years (**Fig. S1**), ranging from 0 to 5491 (**Table 1**). The average AUDPC for INIA-RGDP lines was the lowest with value of 2624 followed by the cultivars from other breeding programs with 2908, while INIA-WBP lines presented an average AUDPC of 3433. The proportion of phenotypic variance attributed to genetic factors, as estimated by broad-sense heritability (H^2) was 0.98 (**Table S2**).

Seedling tests also showed high and uniform infection, with the check cultivar Morocco consistently exhibiting IT scores of 8 or 9. In seedling tests with race *Triticale2015a*, 30.6% of genotypes showed resistant reactions (IT = 0–3), 42.6% displayed intermediate reactions (IT = 4–6), and 26.8% were susceptible (IT = 7–9). For the more widely virulent race *Triticale2015b*, 18.3% of genotypes were resistant, 41% showed intermediate reactions, and 40.7% were susceptible (**Fig. S2**).

Table 1. Average (Mean), minimum (Min), maximum (Max), and standard deviation (SD) and heritability (H^2) of field yellow rust area under the disease progress curve (AUDPC) and seedling infection type (IT) of the 366 wheat lines of the genome-wide association study (GWAS) panel evaluated in field conditions during 2021-2022, and under greenhouse conditions in the seedling stage after inoculation with two locally prevalent *Pts* races.

Resistance type	Trait	Trial	Mean	Min	Max	SD	H^2
Field evaluation	AUDPC	2021	2949	0	4973	1293	0.98
		2022	3234	0	6468	1793	0.98
		Both years	2955	0	5491	1423	0.92
Seedling stage	IT	*Triticale2015a*	4.24	0	9	2.18	0.95
		Triticale2015b	5.17	0	9	2.08	0.98

The BLUEs for YR AUDPC values obtained from field trials were calculated for each year using the best-fitting statistical model. In addition, all models evaluated showed similar accuracy and fitness indicators (AIC, BIC, heritability and correlation between observed and predicted values). For 2021, the model base was selected, as it showed lower AIC and BIC values, minimal differences in heritability and correlation between observed and predicted values compared to the same model but including the column effect (**Table S2**), and a homogeneous and normal residual distribution (**Fig. S3**). In contrast, for 2022, the model including the random column effect provided a better fit, evidenced by lower AIC and BIC values, higher heritability and correlation between observed and predicted values (**Table S2**), and a more uniform residual distribution (**Fig. S3**). In both years, incorporating a spatial correlation structure for the residuals did not improve model fit (**Table S2**).

Pearson correlation analysis of the AUDPC BLUEs from the selected models for 2021 and 2022 revealed a strong correlation between years (r = 0.74), and high correlations among replicates within each year (**Fig. S1**). Based on these results, the data from both years were combined into a single dataset and AUDPC BLUEs were obtained using the model **[2]** which includes effects for experimental design, days to

heading as covariate and the year effect for the combined data from 2021 and 2022 (**Table S2**).

3.6.2. Genotypic data

SNP calling using TASSEL identified 237,282 SNPs for all lines. SNPs with >80% missing data (~38,000) and those with a minor allele frequency (MAF) <0.01 (~43,000) were removed. The final dataset included 366 wheat lines and 156,034 SNPs, with missing data imputed using BEAGLE. The detected SNPs provided good coverage of all chromosomes, with a low marker saturation in the D genome (**Fig. S4**). Heatmap with cluster analysis using the Euclidean distance matrix revealed no distinct groups (**Fig. S5 A**). Similarly, no clear clustering was observed in the PCoA, where the two principal components explained only 0.020 and 0.022 of the variances, respectively, with no relationship to YR AUDPC values or the panel lines' origins (**Fig. S5 B and C**). The admixture analysis also provided no significant evidence of population stratification (**Fig. S5 D**). Together, these results indicated that including subpopulation effects was unnecessary for subsequent analyses. Linkage disequilibrium (LD) analysis showed rapid LD decay along the chromosomes, with average r^2 values falling below 0.2 within 0.12 Mb.

3.6.3. Genome-Wide Association Study (GWAS)

Eight genomic regions associated with field YR resistance were identified (**Fig. 1**) using a false discovery rate (FDR) threshold of $-\log_{10}(p)$, $P < 0.1$ resulting in a value of 3.7. These regions were located on chromosomes 1BL, 2BL (three regions), 5B (one in 5BS and the other in 5BL), 5DL and 7BL (**Table 2**). The three regions identified on chromosome 2BL (as well as the two on chromosome 5B) were considered independent because their physical distance was larger than the 0.12 Mb distance estimated by the LD analysis. Regions with at least two markers above the significance threshold were classified as quantitative trait loci (QTL). When we analyzed all significantly associated markers within each QTL region together to define haplotypes, we found only two haplotypes in each QTL region.

The QTL explaining the highest proportion of the phenotypic variance was *QYr.uy-2BL.3* (21.24%), followed by *Qyr.uy-2BL.2* (12.1%), *Qyr.uy-5BS*, *Qyr.uy-*

5BL, *Qyr.uyt-5DL*, *Qyr.uyt-7DL*, *Qyr.uyt-2BL.1* and *Qyr.uy-1BL* explained progressively lower proportions of the phenotypic variance (**Table 2**). The effect of each QTL on AUDPC is illustrated by the boxplots in **Fig. 2**. The favorable QTL allele (associated with lower AUDPC values) was assigned the value "1", while the less favorable QTL was assigned the value "2". Furthermore, **Fig. 2** represents the distribution of the number of lines according to their YR AUDPC values for each allele of each QTL, based on the width of the surface surrounding each boxplot.

The proportion of lines carrying the favorable YR resistant QTL varied according to their origin (INIA-WBP, INIA-RGDP, and cultivars from other breeding programs). *Qyr.uy-5BL* was present in 84% of all lines, *Qyr.uy-1BL* in 80%, *Qyr.uy-5DL* in 75%, *Qyr.uy-7BL* in 61%, *Qyr.uy-5BS* in 36%, *Qyr.uy-2BL.1* in 35%, *Qyr.uy-2BL.3* in 27%, and *Qyr.uy-2BL.2* in 18%of the lines. *Qyr.uy-1BL*, *Qyr.uy-2BL.3*, *Qyr.uy-5BS*, *Qyr.uy-5BL*, and *Qyr.uy-7BL* were found in a higher percentage of lines from INIA-RGDP, in contrast, *Qyr.uy-2BL.1* and *Qyr.uy-2BL.2* were more frequent in cultivars from other breeding programs (**Table 3**).

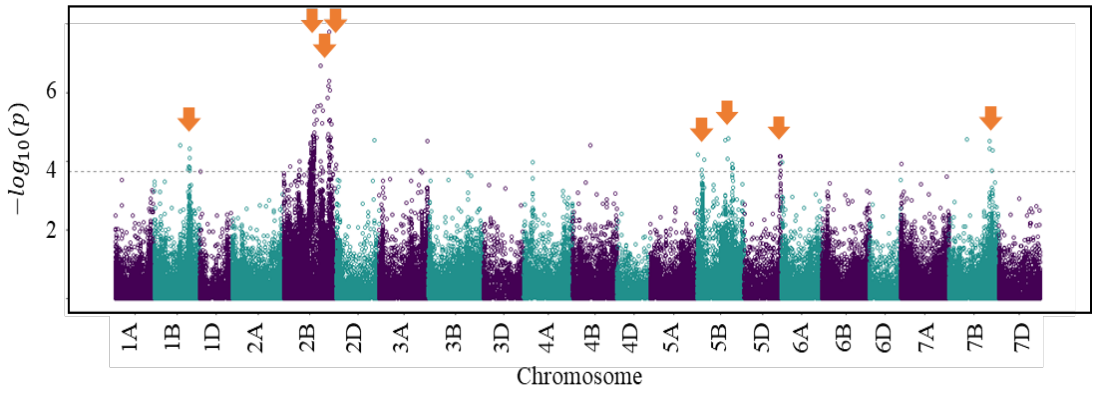


Figure 1. Manhattan plot for yellow rust (YR) resistance based on area under the disease progress curve (AUDPC) combined values from 2021 and 2022 field trials in 366 wheat lines of the panel. The horizontal dotted line indicates the genome-wide significance threshold

Table 2. Summary of significant Quantitative trait loci (QTL) associated with yellow rust (YR) resistance. The table includes QTL identified for field resistance based on area under the disease progress curve (AUDPC) combined values from 2021 and 2022, and seedling resistance based on infection types (IT) for *Pst* race *Triticale2015a* of 366 wheat lines of the panel.

Trait	QTL	Chr.[a]	Physical position of flanking markers (Mb)[b]	P value	Effect	PVE (%)[c]
Field AUDPC	*Qyr.uy-1BL*	1B	540.16 - 541.70	$2.7e^{-5}$	-848.82	4.9
	QYr.uy-2BL.1	2B	400.34 - 464.32	$5.5e^{-6}$	-706.7	5.7
	QYr.uy-2BL.2	2B	564.47 – 564.82	$9.1e^{-12}$	-1268.2	12.1
	QYr.uy-2BL.3	2B	690.94 – 709.24	$<2e^{-16}$	-1425.2	21.2
	QYr.uy-5BS	5B	69.74 – 74.92	$3.5e^{-10}$	-973.2	10.6
	QYr.uy-5BL	5B	537.95 – 538.48	$1.3e^{-8}$	-1156.8	8.6
	QYr.uy-5DL	5D	548.10 – 552.03	$2.1e^{-7}$	-898.4	7.3
	QYr.uy-7BL	7B	617.83 – 657.05	$1.5e^{-6}$	-777.2	6.6
Seedling IT *Triticale2015a*	*QYr.uy-2DS*	2D	15.34 - 18.30	$2.2e^{-16}$	-2.3	19.1
	QYr.uy-3AL	3A	488.45 - 490.15	$5.4e^{-8}$	-1.4	7.9

[a]Chromosome, [b]Physical positions (Mb) of flanking markers are based on the Chinese Spring reference IWGSC RefSeq v1.0; [c]PVE, phenotypic variance explained.

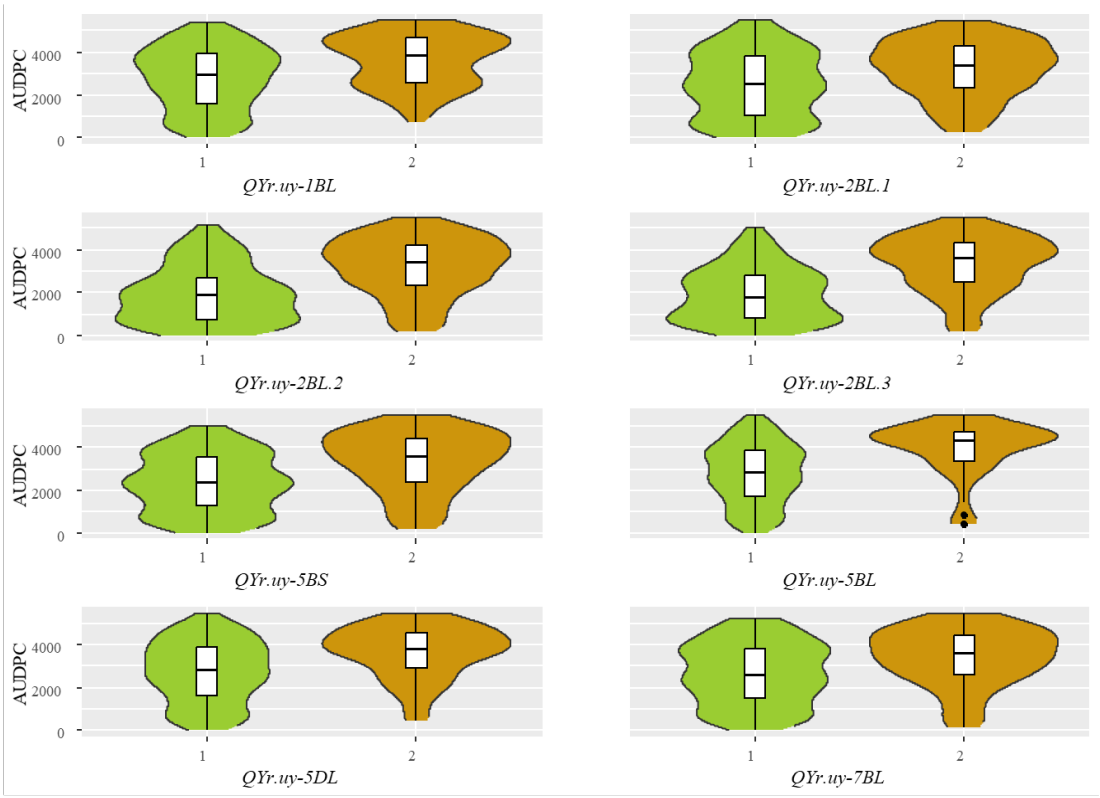
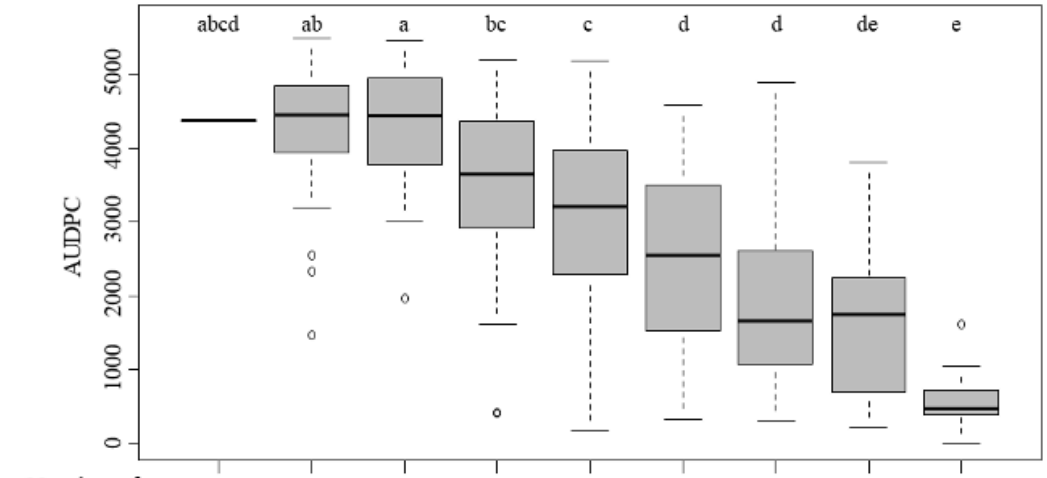


Figure 2. The effects of quantitative trait loci (QTL) identified for yellow rust (YR) resistance based on area under the disease progress curve (AUDPC) combined values from the 2021 and 2022 field trials. The AUDPC value for the 366 wheat lines of the GWAS panel are shown based on their haplotype for each QTL, with (1) being the more favorable resistant allele, and (2) being the more susceptible allele.

Table 3. Percentage of lines carrying the favorable allele for each quantitative trait loci (QTL) associated with area under the disease progress curve (AUDPC) in field trials and infection type (IT) in seedling tests for the *Triticale2015a* race, within each group of lines according to their origin and the full panel of 366 wheat lines.

Trait	QTL	Line origin			Full panel
		INIA-RGDP	INIA-WBP	Other breeding programs	
Field AUDPC	*QYr.uy-1BL*	84	80	68	80
	QYr.uy-2BL.1	19	33	77	35
	QYr.uy-2BL.2	14	12	38	18
	QYr.uy-2BL.3	30	21	27	27
	QYr.uy-5BS	44	23	38	36
	QYr.uy-5BL	89	82	77	84
	QYr.uy-5DL	78	80	62	75
	QYr.uy-7BL	67	54	62	61
Seedling IT	*QYr.uy-2DS*	20	25	14	20
Triticale2015a	*QYr.uy-3AL*	25	26	32	27

The lines were grouped into nine categories based on the number of favorable QTL alleles for YR AUDPC determined in the field trials, ranging from zero to eight favorable QTL. A pronounced in AUDPC values was observed as the number of favorable QTL increased (**Fig. 3**).



	Number of lines in each group								
INIA-RGDP	0	7	16	24	54	41	14	13	3
INIA-WBP	0	5	16	24	39	21	5	5	2
Other breeding programs	1	6	9	9	11	11	10	10	6
Total	1	18	41	57	104	73	29	28	11

Figure 3. Effect of the number of quantitative trait loci (QTL) associated with yellow rust (YR) resistance based on area under the disease progress curve (AUDPC) combined values from 2021 and 2022 field trials of 366 wheat lines. The number of lines into each group according to their origin is indicated below each boxplot. Different letters above the boxplots indicate significant differences ($P < 0.05$) in AUDPC between groups, as determined by Tukey′s test. Boxplots show the distribution of a dataset through five key summary statistics: minimum (lower whisker), first quartile (bottom of the box), median (line inside the box), third quartile (top of the box), and maximum (upper whisker). Points beyond the whiskers are values outside 1.5 times the interquartile range from the quartiles.

Two genomic regions associated with YR seedling resistance to race *Triticale2015a* were identified (**Fig. S6 A**). Haplotype analysis revealed two haplotypes for each identified region. The QTL on chromosome 2D explained 19.1% of the phenotypic variance, while another QTL on chromosome 3A accounted for 7.9% (**Table 2**). The favorable allele for *QYr.uy-2DS* was present in 20% of all lines, while the favorable allele for *QYr.uy-3AL* was found in 27%. The favorable allele of *QYr.uy-*

3AL was present in a higher proportion in INIA-WBP lines while the favorable allele of *QYr.uy-2DS* was present in a higher proportion of cultivars from other breeding programs (**Table 3**). GWAS for the more widely virulent race *Triticale2015b* did not identify any genomic regions significantly associated with YR resistance (**Fig. S6 B**). Through GWAS, we detected one mayor QTL for days to heading on chromosome 2D, which did not co-localized with any of the QTL associated with YR resistance in the field or seedling trials (**Fig. S6 C**). Additionally, the identified QTL for YR resistance did not coincide with the location of previously reported phenology-related genes (data not shown).

3.6.4. Genomic prediction (GP)

From the seven GP models evaluated, the A-BLUP model had the lowest prediction accuracy, with correlations between observed and predicted AUDPC values below 0.5, and the highest MSE (**Fig. S7 A**). The G-BLUP and RR-BLUP models performed similarly, achieving correlations between observed and predicted AUDPC values around 0.7 and a lower MSE compared to other models MSE (**Fig. S7 B**). Bayesian models (BA, BB, and BC) demonstrated comparable prediction accuracies as G-BLUP and RR-BLUP, with correlations near 0.7 and moderate MSE values. In contrast, the Bayesian LASSO (BL) model exhibited poorer performance, with accuracy closer to the A-BLUP model and higher MSE values. Overall, G-BLUP, RR-BLUP, and Bayesian (BA, BB and BC) models showed the most robust predictive performance.

The BL model performed worse than RR-BLUP, and the prediction ability of RR-BLUP model was nearly identical to that of the G-BLUP model. Therefore, the G-BLUP model was selected for further comparisons due to its simplicity and lower computational requirements. Subsequently, we investigated whether incorporating fixed effects for the identified QTL could improve the GP accuracy. We compared the AUDPC predictions from the G-BLUP model without fixed QTL effects and with the sequential addition of fixed effects for the eight QTL, added in descending order of the explained variance. The inclusion of fixed effects in the model led to an improvement in prediction accuracy, with correlations between observed and

predicted values increasing from an average of 0.64 in the G-BLUP model without fixed effects to 0.69 in models that included GWAS-identified QTL as fixed effects. Notably, the inclusion of *QYr.uy-2BL.3* alone was sufficient to achieve this improvement, as no further gains were observed when additional QTL were incorporated as fixed effects (**Fig. S8 A**). Moreover, the inclusion of fixed effects also impacted the MSE, which was reduced by 10.4% when *QYr.uy-2BL.3* was included, compared to the G-BLUP model without it (**Fig. S8 B**).

3.7. Discussion

Recent outbreaks of YR in major wheat-producing regions worldwide (Bouvet et al., 2022) pose a significant threat to wheat production and global food security. Particularly in the Southern Cone of South America, recent epidemics (Campos, 2020; Campos et al., 2016; S. E. Germán et al., 2018; Riella et al., 2024; Silva et al., 2023) have been linked to the incursion of new *Pst* genetic groups and races characterized by increased aggressiveness and improved adaptation to diverse temperature ranges (Rajaram & Campos, 1974). Argentina and Uruguay are located in the same rust epidemiological zone (Rajaram & Campos, 1974) where there are no geographical barriers for urediniospores dispersal, which likely explains the almost simultaneous development of severe epidemics in both countries (Rudolf & Job, 1931). In Uruguay, *Pst* was first detected in 1929 (Rudolf & Job, 1931). It caused widespread epidemics and substantial yield losses across the Southern Cone region during 1929 and 1930 (Boerger, 1934; Vallega, 1938). From its initial detection until 2016, *Pst* outbreaks remained sporadic, rarely reaching epidemic levels in Uruguay (S. E. Germán et al., 2007, 2018; S. E. Germán & Caffarel, 1999). However, since 2017, wheat crops grown in Uruguay and Argentina have experienced widespread epidemics, likely due to an earlier onset of the disease during the growing season, the extensive planting of susceptible or moderately susceptible cultivars (covering more than 50% of the wheat-growing area) (Silva et al., 2023), and the emergence of novel races in the region (Riella et al., 2024).

YR is currently the wheat foliar disease, which requires the largest number of fungicide applications in Uruguay. Deploying resistant wheat cultivars is an

economically and environmentally sustainable strategy, significantly reducing the use of fungicides (X. Chen, 2013; X. M. Chen, 2005). Therefore, it is essential to identify diverse resistance sources effective under local conditions, which can be utilized in breeding programs to introgress and pyramid resistance genes into locally adapted germplasm. From the perspective of resistance breeding, the most relevant phenotype is that expressed in the field (APR). However, this must be complemented with the seedling phenotype (ASR) to determine which types of resistance genes are effective: ASR and/or APR. Additionally, early identification of promising lines or parental candidates for breeding crosses is crucial to accelerate the development of wheat lines with durable YR resistance.

Despite Uruguay's longstanding wheat breeding program at the National Institute of Agronomical Research (INIA), which has focused on developing resistance to major regional diseases such as leaf (Silva et al., 2015) and stem rusts (Baraibar et al., 2020), Fusarium head blight (Díaz de Ackermann and Pereyra, 2011a; Germán et al., 2018), Septoria tritici blotch (Díaz de Ackermann, 2011a; Germán et al., 2018) and tan spot (Díaz de Ackermann, 2011b; Germán et al., 2018), YR was historically considered a minor threat. Consequently, no breeding efforts specifically targeting YR resistance were implemented, leaving the genetic basis of resistance in the local germplasm largely unknown (Germán & Luizzi, 2018). In Uruguay, the *PstS13* genetic group has been reported as the most prevalent since the 2017 epidemics (Riella et al., 2024). Within *PstS13*, the two races used in this study for field inoculations, *Triticale2015(a)* and its locally discovered variant, *Triticale2015b*, with additional virulence to *Yr17* and *Yr32*, have been the most prevalent in recent years. The original *PstS13* race, *Triticale2015*, was first reported in Europe in 2015, primarily affecting triticale and durum wheat (Hovmøller et al., 2018). Since 2017, *PstS13* has been the predominant genetic group in Argentina (Carmona et al., 2019; Hovmøller et al., 2018, 2019), it was also detected in Chile (Hovmøller et al., 2019) and more recently in Paraguay (Fernández-Gamarra et al., 2023).

3.7.1. Phenotypic variation for wheat resistance to yellow rust

The panel of 366 wheat lines, including INIA germplasm and other commercial varieties widely used locally, was phenotyped in field trials over two consecutive years under artificial inoculations with the predominant *Pst* races. Additionally, seedling assays were performed with the same races used for field inoculations. Phenotypic data showed significant variation among lines for all evaluated traits. Seedling IT exhibited a bias toward susceptibility (**Fig. S2**), for the broader virulent race (*Triticale2015b*), conversely, field YR AUDPC showed a bias toward resistance, suggesting a low presence of effective ASR genes in the panel and the presence of APR genes, which are more effective in the adult plant stage. This is consistent with the panel composition, as many lines originate from crosses with sources of leaf rust APR (generally pleiotropic for YR APR), while no intentional introgressions of YR ASR genes have been performed in the WBP. Another evidence of absence of effective ASR genes in the panel is the lack of significant correlation between seedling IT and field AUDPC (r^2=0.46).

The high broad-sense heritability for AUDPC across 2021 (0.98), 2022 (0.98), and combined years (0.97), as an indicator of repeatability, coupled with strong correlations among replicates within each year (0.94–0.95) and between BLUEs AUDPC values across years (0.74), underscores the robustness of this phenotypic dataset for GWAS and GP analyses.

3.7.2. Population structure of the wheat panel

Accounting for population structure is critical in GWAS to minimize false-positive marker-trait associations (Pritchard et al., 2000; Yu et al., 2006; C. Zhu et al., 2008). In this study, population structure analyses revealed no strong genetic stratification among the 366 wheat lines that would require inclusion in subsequent GWAS analyses. Lines did not cluster based on origin (INIA-WBP, INIA-RGDP, or other breeding programs), likely reflecting the diverse germplasm and resistance sources used by INIA. Moreover, no clear association was observed between AUDPC values and line origin. Interestingly, lines from the INIA-RGDP, which were selected for APR to leaf rust, exhibited lower average YR AUDPC values than commercial

varieties or advanced INIA-WBP lines. This observation suggests the presence of potentially pleiotropic APR genes in these lines, providing a valuable genetic resource for breeding wheat lines with enhanced YR resistance. Exploiting these genetic resources could accelerate the development of cultivars with durable, broad disease spectrum resistance.

3.7.3. Genome-Wide Association Study (GWAS)

The high-quality phenotypic data collected from the GWAS panel of 366 wheat lines evaluated in both field and greenhouse trials, combined with a dense set of SNPs distributed across the genome (**Fig. S4**), provided a robust framework for identifying genomic regions associated with YR resistance. As no strong population structure was found, the K model was enough to control for spurious associations, as confirmed by quantile-quantile (QQ) plots (**Fig. S9**).

Eight genomic regions associated with YR resistance were identified in field trials, with stable expression as these were consistently detected across data from both years (data not shown). Pyramiding the identified QTL for YR resistance significantly reduced YR AUDPC (**Fig. 3**), aligning with findings from other studies (Franco et al., 2022; M. Lin et al., 2023; Maccaferri et al., 2015; Miedaner et al., 2024; Wang et al., 2024; X. Zhou et al., 2021), which also highlight additive effects improving YR and leaf rust resistance as the number of favorable QTL increases.

GWAS analyses for seedling resistance did not identify any of the regions detected in the field. The two regions associated with seedling resistance for one of the *Pst* races were ineffective to the other race. Although both races belong to the same genetic group, race *Triticale2015b* carries additional virulence to *Yr17* and *Yr32* (Riella et al., 2024), which are located in genomic regions distinct from the QTL detected in the field trials. These results confirm that the QTL identified in the field confer APR.

3.7.4. Analysis of identified genomic regions associated with yellow rust resistance

Eight genomic regions associated with YR resistance were identified: one on chromosome 1B, three on 2B separated by more than 126 Mb, two on 5B, one on 5D and one on 7B. Regions significantly associated with YR resistance identified in this

study were compared with previously mapped *Yr* genes and QTL using the most updated atlas available (McIntosh, 2024; Tong et al., 2024). The possibility that these associations were due to differences in heading date was excluded, since GWAS analyses using days to heading as the response variable revealed no overlap between the QTL associated with YR resistance and QTL associated to heading date (**Fig. S6 C**). Additionally, two QTL associated with seedling resistance to the *Pst* race *Triticale2015a* were identified on chromosomes 2D and 3A, but none were identified to race *Triticale2015b*, indicating that the resistance detected in the field was expressed after the seedling stage and is most possibly conferred by APR genes.

3.7.5. Adult-plant resistance QTL

3.7.5.1. Chromosome 1B

QYr.uy-1BL located on the long arm of chromosome 1B explained only 4.9% of the total variance, and had the lowest effect, reducing YR AUDPC by 848.8 (**Table 2**), which represents an average AUDPC reduction of 12% compared to the lines with the less favorable allele. This QTL was widely present in the lines (80%), predominantly in germplasm from INIA (**Table 3**). Chromosome 1B, is considered a hotspot for YR resistance, as at least eight *Yr* genes have been mapped on this chromosome, including *Yr9* (Lukaszewski, 2000), *Yr10* (W. Liu et al., 2014), *Yr15* (Klymiuk et al., 2018), *Yr24*/*Yr26* (McIntosh, 2024), *Yr29* (William et al., 2003), *Yr64* (P. Cheng et al., 2014), and *Yr65* (P. Cheng et al., 2014). Numerous other temporarily designated genes, such as *YrChk* (F. Liu et al., 2007), *YrExp1* (F. Lin & Chen, 2007), and *YrGn22* (Q. Li et al., 2016), are also located on 1B. However, all these genes have been mapped far from the *QYr.uy-1BL* region detected in our study. *Yr29*, a pleiotropic APR gene with a moderate effect, located on chromosome 1BL at 661.86 Mb (J. Li et al., 2020), is the closets among these *Yr* genes, but still lies more than 120 Mb away from *QYr.uy-1BL* region. Genotyping of the panel using a KASP marker for *Yr29* (**Table S1**) revealed that it was present in 85% of the lines. However, *Yr29* did not show a significant effect on YR AUDPC. This evidence indicates that *QYr.uy-1BL* is not *Yr29.* In addition, over a dozen studies have reported QTL for YR resistance on this chromosome (Alemu et al., 2021; Draz et al., 2021). *QYr.uy-1BL* might correspond

to the closest reported QTL located at approximately 8 Mb (Rosewarne et al. 2012) (**Table S3**).

3.7.5.2. Chromosome 2B

Three QTL were identified on the long arm of chromosome 2B. *QYr.uy-2BL.1* accounted for 5.7% of the total phenotypic variance, with an estimated effect of 706.7, corresponding to a 45.3% reduction in AUDPC relative to lines carrying the susceptible allele. The favorable allele of *QYr.uy-2BL.1* was present in 35% of the lines and was more frequently observed in germplasm from breeding programs other than INIA (**Table 3**). *QYr.uy-2BL.2* explained 12.1% of the variance, with an estimated effect of -1268.2, corresponding to a 49% reduction in AUDPC. The favorable allele was detected in 18% of the lines, and was more prevalent in non-INIA cultivars (**Table 3**). *QYr.uy-2BL.3* had the largest effect among all QTL detected in this study, reducing YR AUDPC by 1425.2 (61%) and explaining 21.2% of the total variance (**Table 2**). The favorable allele was present in 27% of the lines (**Table 3**).

Several *Yr* genes, including *Yr5*, *Yr7* (Marchal et al., 2018), *Yr41* (Luo et al., 2008), *Yr43* (Feng et al., 2015), *Yr44* (Sui et al., 2009), *Yr53* (Xu et al., 2013), and *Yr72* (Chhetri et al., 2023), along with numerous temporarily designated genes and QTL, have been mapped to the long arm of chromosome 2B. Several loci associated with APR have also been identified on this chromosome. *QYr.uy-2BL.1*, located between 400.34 and 464.32 Mb, overlaps with two previously reported QTL: *QYr.caas-2BS.1* (Bai et al., 2012) and *QYr.ifa-2BL* (Buerstmayr et al., 2014) (**Table S3**). *QYr.uy-2BL.2*, located between 564.47 and 564.82 Mb, lies approximately 40 Mb from *Yr53*, which has not been introgressed into INIA-WBP germplasm. Two nearby QTL, *QYr.nafu-2BL* (X. L. Zhou et al., 2015) and *QYrqn.nwafu-2BL* (Zeng et al., 2019) are located 8 Mb and 15 Mb away, respectively, and confer YR APR. Due to its low frequency in the panel *QYr.uy-2BL.*2 represents a QTL with potential from the breeding perspective.

The physical position of *QYr.uy-2BL.3* is close to ASR genes *Yr5*, *Yr7*, and *YrSP* which belong to a complex gene cluster (Marchal et al., 2018). *Yr7* and *YrSP* are ASR genes ineffective to the *Pst* races present in Uruguay (Riella et al., 2024) therefore

these genes are not *QYr.uy-2BL.3*. Only ASE gene *Yr5* remains effective in Uruguay. Moreover, GWAS analyses of seedling ITs using the same *Pst* races as those used in field inoculations did not identify associations near the *Yr5* locus. Among lines carrying the favorable allele for *QYr.uy-2BL.3*, both resistant and susceptible seedling responses were observed, whereas the expected IT for *Yr5* carriers is 0; to ; (McIntosh et al., 1995). In race identification tests, *Avocet Yr5* which carries *Yr5* as the sole gene consistently showed an IT of 0 or 1 for both races. Additionally, KASP marker analysis (Marchal et al., 2018) indicated the absence of *Yr5* in the tested lines, including those carrying *QYr.uy-2BL.3* (data not shown). This evidence indicates that *QYr.uy-2BL.3* is not *Yr5*, but rather a distinct QTL associated with YR APR. QTL reported in this region, includ *QYrsnb.nwafu-2BL* (Zeng et al., 2019) and *Qyr.gaas.2B.1* (B. Cheng et al., 2022), both located approximately 10 Mb from *QYr.uy-2BL.3* (**Table S3**). *QYr.uy-2BL.3* stands out as the most promising QTL for INIA-WBP due to its strong effect and relatively low frequency in the germplasm panel.

3.7.5.3. Chromosome 5B

Two QTL were identified on chromosome 5B: *QYr.uy-5BS* on the short arm and *QYr.uy-5BL* on the long arm. *QYr.uy-5BS* explained 10.6% of the phenotypic variance, had an estimated effect of 973.2, corresponding to a 13.9% reduction in AUDPC relative to lines carrying the susceptible allele. The favorable allele of *QYr.uy-5BS* was present in 36% of the lines and was more frequently observed in germplasm from the INIA-RGDP program (**Table 3**). *QYr.uy-5BL* reduced YR AUDPC by 1156.8 (16%) and accounted for 8.6% of the phenotypic variance (**Table 2**). It is present at high frequency (84%), with greater representation in INIA-RGDP (**Table 3**).

Two YR resistance genes have been previously reported on chromosome 5B: *Yr47* (Bansal et al., 2011) and *Yr19* (X. M. Chen et al., 1995). However, the genomic region of *Yr47* on the short arm of the chromosome is 64 Mb from *QYr.uy-5BS*. Therefore, *Yr47* is not any of the QTL reported in our study. *Yr19* is an ASR gene, whose physical position on chromosome 5B is not known (X. M. Chen et al., 1995). Since both *QYr.uy-5BS* and *QYr.uy-5BL* were detected only in our field trials but not at the seedling stage, they do not correspond *Yr19*.

The closest previously reported QTL to *QYr.uy-5BS* is *QYr.ufs-5B* (Agenbag et al., 2012) but is located more than 40 Mb away (**Table S3**). This strongly suggests that *QYr.uy-5BS* may represent a novel QTL which valuable for INIA-WBP and other breeding programs. Three QTL reported on chromosome 5BL, are located 10 Mb or less from *QYr.uy-5BL* (*QYr.YBZR-5BL*, Deng et al. 2022; *QYr.AYH-5BL*, Long et al. 2021; *QYrdr.wgp-5BL.2*, Hou et al. 2015). *QYr.uy-5BL* might be *QYrdr.wgp-5BL.2* which lies less than 1Mb from it.

3.7.5.4. Chromosome 5D

QYr.uy-5DL, located on the long arm of chromosome 5D, explained 7.3% of the phenotypic variance and reduced YR AUDPC by 898.4, representing a 14% average reduction compared to lines carrying the less favorable allele (**Table 2**). This QTL was detected in 75% of the lines, being been more frequently present in germplasm from INIA (**Table 3**). *Yr70* (Pasam et al., 2017), the only nominated gene located on this chromosome is over 200 Mb away. Two previously reported QTL, *QYrdr.wgp-5DL* (Hou et al., 2015) and *QYrbr.wpg-5D* (Case et al., 2014), have been identified on 5DL, at about 5 Mb from the region where *QYr.uy-5DL* is located (**Table S3**). It is present in high frequency in INIA germplasm, efforts should be made to maintain this QTL in the breeding germplasm.

Chromosome 7B

QYr.uy-7BL, located on the long arm of chromosome 7B, explained 6.6% of the phenotypic variance and reduced YR AUDPC by 777.2, corresponding to an average reduction of 12.7% compared to lines carrying the less favorable allele (**Table 2**). This QTL was present in 61% of the lines, with no major differences in frequency across germplasm origins (**Table 3**). *Yr67* (Bariana et al., 2022) has been reported approximately 40 Mb from the physical position of *QYr.uy-7BL*. QTL *QYr.hebau-7BL* (P. Zhang et al., 2019), *QYr.niab-7B* (Powell et al., 2013), and *QYr.cim-7BL* (Calvo-Salazar et al., 2015) colocalize with the region where *QYr.uy-7BL* is located (**Table S3**).

3.7.6. All-stage resistance QTL

3.7.6.1. *QYr.uy-2DS*

QYr.uy-2DS, identified for the *Triticale2015a* race, was the QTL with the largest effect at the seedling stage, explaining 19.1% of the phenotypic variance. Lines carrying the favorable allele for this QTL (20%) had an average IT of 2.4, representing a reduction of 2.3 IT units (49%) compared to the lines lacking it (**Table 2**). No *Yr* genes have been mapped to the region where *QYr.uy-2DS* is located, although the major QTL *Yrq1* (Cao et al., 2012) colocalize with *QYr.uy-2DS* and *QYr.hbau-2DS* (Gebrewahid et al., 2020) is only 3 Mb away (**Table S3**).

3.7.6.2. *QYr.uy-3AL*

QYr.uy-3AL was the QTL with the smallest effect in the seedling stage to race *Triticale2015a*, reducing IT in 1.4 (49%) and explaining 7.9% of the phenotypic variance (**Table 2**). *QYr.uy-3AL* was the most frequent seedling QTL within cultivars from breeding programs other than INIA (**Table 3**). *QYr.uy-3AL* is located on the long arm of chromosome 3A. *Yr76*, the only *Yr* gene previously mapped on chromosome 3A (Xiang et al., 2016), is located on the short arm, indicating that *QYr.uy-3AL* is distinct. Several QTL for YR resistance have also been reported on 3AL; among them, *QYr.nmbu.3A.2* (M. Lin et al., 2023) and *QYr.hbaas-3AL* (Jia et al., 2020), are the closest to *QYr.uy-3AL*, located approximately 7 and 12 Mb away, respectively (**Table S3**).

Both, *QYr.uy-2DS* and *QYr.uy-3AL*, are not effective to race *Triticale2015b* and were not detected in field tests, therefore their relevance for resistance breeding is limited.

3.7.7. Implication of identified QTL in the breeding program context

The identification of eight genomic regions associated with YR resistance in field trials and two regions in seedling assays highlights the value of exploring local genetic resources. Local breeding programs represent a valuable reservoir of genetic diversity adapted to local conditions and are key resources for breeding programs. While several ASR *Yr* genes remain effective to the current *Pst* population, the

variability of the pathogen requires the continuous exploration and introgression of new resistance sources to increase the genetic diversity. *PstS13*, the predominant genetic group of *Pst* in local conditions, is virulent to several widely deployed *Yr* genes (Tadesse et al. 2014; Hovmøller et al. 2016). Other races within the *PstS13* group identified locally, acquired virulence to *Yr3*, *Yr17*, *Yr25*, *Yr27*, and *Yr32* (Riella et al., 2024).

Among the eight QTL identified in field trials, only *QYr.uy-2BL.3* co-localized with previously reported ASR *Yr* gene cluster (*Yr5*, *Yr7*, *YrSP*), but it was demonstrated it was not any of these genes. *QYr.uy-5BS* appears to be a novel QTL. Other QTL identified in this study are located near (10 Mb or less) of previously reported QTL, therefore, further confirmatory studies are required to determine whether these QTL correspond to known loci or represent new, distinct QTL, e.g. using markers for QTL reported in the literature near those identified in this study as well as developing functional markers and validating QTL through biparental populations.

The QTL identified in field trials were not detected in seedling tests (**Fig. S6**), indicating that these correspond to APR genes. Their additive effects (**Fig. 3**) further support that these are likely race non-specific and durable, which is expected within INIA germplasm, where ASR genes have not been deliberately used. Many of the INIA-RGDP lines with low YR AUDPC are derived from crosses between locally adapted materials and sources of leaf rust APR, mostly from CIMMYT. These lines were selected for leaf rust resistance, suggesting a pleiotropic effect of the APR to both rusts. In that sense, it would be expected that APR genes such as *Yr18*, *Yr29*, and *Yr46*, frequently present in CIMMYT germplasm, or QTL for YR resistance found in this germplasm (Singh, 1992; Singh & Rajaram, 1992, 1993) should have been detected in the GWAS analysis. However, genotype-by-environment interactions involving minor APR genes might influence their expression, as previously reported for rust diseases (Lillemo and Singh 2011; Calvo-Salazar et al. 2015; Silva et al. 2015).

KASP marker results revealed that *Yr18* was present in 28.7% of the lines (**Table S1**) and was associated with a non-significant reduction in AUDPC (~307) which was not detected in the GWAS analysis. One possible explanation is the low

marker saturation of the D genome, particularly in the region where *Yr18* resides (**Fig. S4**), which reflects the overall lower polymorphism of this genome compared to the A and B genomes. To address this, GWAS was performed incorporating the KASP marker for *Yr18* into the SNP matrix. However, the marker still did not surpass the significance threshold in the updated models, suggesting that low marker density was not the cause of its non-detection. Therefore, this result might be explained by the relatively small effect of *Yr18* in reducing AUDPC, consistent with previous studies reporting partial resistance conferred by this gene (Wu et al., 2015; Zelba et al., 2024). Similarly, *Yr29* was not detected by GWAS even after including its KASP marker in the SNP matrix, despite showing a statistically significant AUDPC reduction (~349) (data not shown). Its high frequency in the panel (present in 85% of lines) likely reduced the statistical power to detect associations. Nevertheless, *Yr29* has been shown to have a stronger effect under Mexican field conditions (D. Liu et al., 2022), suggesting that its effectiveness may be influenced by the environment. In contrast, KASP marker results showed that *Yr46* was absent from the panel except for the check line Thatcher *Yr46* (**Table S1**). Notably, this gene had a marked effect on disease resistance: Thatcher showed an AUDPC of 4808, whereas Thatcher *Yr46* exhibited a much lower value (3315), highlighting the potential of *Yr46* for introgression into INIA-WBP germplasm. However, its very low frequency in the panel (<1%) prevented its detection in the GWAS, as it did not meet the MAF threshold used in this study. A marked decrease in AUDPC was observed as the number of favorable QTL per line increased (**Fig. 3**), indicating additive effects among the identified QTL. This highlights QTL pyramiding is a promising strategy for breeding wheat with higher levels of durable resistance. Clearly, two of the QTL (*QYr.uy-1BL* and *QYr.uy-5BL*) were already present at a high proportion in the INIA-WBP advanced germplasm and in cultivars of other origin. However, the other three QTL were present at a much lower proportion and pyramiding them with those QTL already present may contribute to the development of YR resistant cultivars. Notably, the eleven lines carrying the favorable alleles for all eight QTL showed final disease severity values below 20%. Among them, two pre-breeding lines from the INIA program, R15F57341 and R17F57132,

exhibited near-immunity levels at the adult plant stage, making them invaluable resources for resistance breeding and future research.

3.7.8. Genomic Prediction (GP)

Modern breeding programs, especially in a context where genotyping costs are increasingly affordable and accessible, require the optimization of strategies not only for selecting lines but also for efficiently identifying parents for crosses at an early stage. This is key to developing adapted and resistant cultivars in the shortest possible time. This study aimed to determine the predictive ability of different GP models using the genomic and phenotypic information of the panel lines. Additionally, it sought to demonstrate whether incorporating the presence of the QTL identified through GWAS for YR AUDPC as fixed effects could improve the GP models' predictive ability. Seven different GP models, which assume different distributions for marker effects, were evaluated. These models included A-BLUP model; RR-BLUP, which uses information from all markers; G-BLUP, which uses information about the genetic distance between lines to make predictions; and four types of Bayesian models: BA, BB, BC, and BL. The results of the comparison between the seven models showed no significant differences in performance between RR-BLUP and G-BLUP, with both models having correlations between observed and predicted AUDPC values close to 0.7, which is not suppressing given that the equivalence between these two models has been previously reported (Habier et al., 2007). No differences were observed with the Bayesian models BA, BB, or BC. In contrast, the BL model showed worse performance, with correlations between observed and predicted values around 0.5. BL results were similar to the A-BLUP model, which only uses the available pedigree relationships between the panel lines. Similar results, with minimal differences between prediction models for this disease, were reported by Tehseen et al. (2021) and Manickavelu et al. (2016). The G-BLUP and Bayesian models investigated in this study gave nearly identical prediction accuracies, despite assuming similar variances for all marker effects in the G-BLUP model, as reported by Tehseen et al. (2021). However, since no significant differences were observed between the regression-based G-BLUP and RR methods, and the Bayesian-based models, the assumption of marker

effects having equal variances proved to be effective for YR AUDPC. Therefore, the higher computational time required for the prior densities and shrinkage of Bayesian models may not be necessary. G-BLUP or RR models have also been reported to offer similar prediction accuracies as the BC and BL methods for YR and stem rust (Ornella et al. 2012), stem rust (Rutkoski et al., 2014), and Fusarium head blight in wheat (Rutkoski et al., 2012).

GP proved to be efficient in predicting the response to YR within the GP panel, with prediction accuracies of around 0.7 for the equivalent models RR-BLUP and G-BLUP. The inclusion of GWAS-identified QTL as fixed effects in the G-BLUP model led to an improvement in prediction accuracy. Notably, the inclusion of the QTL with the highest effect was sufficient to achieve this improvement, as no further gains were observed when additional QTL were incorporated as fixed effects (**Fig. S8 A**). Moreover, the inclusion of fixed effects also impacted the MSE, which was reduced by 10.4% when *QYr.uy-2BL.3* was included, compared to the G-BLUP model without it (**Fig. S9 B**), likely due to their high effect on the response variable. In simulation studies it was demonstrated that modeling a large-effect locus as a fixed effect was advantageous when the heritability of the trait exceeded 0.5 and the locus explained more than 25% of the genetic variance (Bernardo, 2014). Consequently, studies with real data have shown that G-BLUP models incorporating fixed-effect markers outperformed standard G-BLUP for traits where the fixed-effect markers explained a substantial proportion of the variation (Juliana et al. 2017). Similarly, Rutkoski et al. (2014) found that including fixed-effect markers in G-BLUP increased accuracy for quantitative APR to stem rust in wheat. This approach would maximize genetic gain only if GP was applied to the specific dataset used in their study. However, for new samples, outcomes from GP using G-BLUP alone could be just as favorable as those obtained by including fixed-effect linked markers.

3.8. Conclusions

The results of this study lay the foundation for understanding the genetic basis of the YR resistance present in a diverse wheat panel and can be directly applied to the development of new locally adapted cultivars with better YR resistance. We report

eight genomic regions associated with field resistance, none of these regions were identified at seedling stage to race *Triticale2015b*. All loci conferred quantitative APR and did not correspond to known *Yr* genes. *QYr.uy-5BS* is most likely a novel QTL. The positions of the other seven QTL were close to previously reported QTL, further studies are needed to determine whether they represent known or novel QTL. Two QTL on 2D and 3A identified at the seedling stage to race *Triticale2015a* did not confer field resistance. Once validated, these QTL could be used to develop and select varieties with high levels of YR resistance. Similarly, GP was highly effective (with prediction ability around 0.7) in predicting disease levels, positioning GP as a valuable tool for selecting parents in breeding programs, as well as for selecting lines. The methodology used for analyzing both phenotypic field data and genotypic data enabled the identification of genomic regions associated with YR resistance and the evaluation of GP models which can be applicable to projects on other wheat diseases and crop species. Moreover, it proved to be highly robust and capable of delivering high-quality data, which serves as the foundation for any solid breeding strategy. These findings provide valuable insights into the genetic basis of YR and offer robust tools for enhancing YR resistance breeding efforts in wheat.

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3.10. Supplementary material

Figure S1. Scatter and frequency distributions and Pearson's correlation coefficients for yellow rust (YR) area under the disease progress curve (AUDPC) across 366 wheat lines in the panel evaluated under field conditions. Panels represent data for: A) 2021, B) 2022, and C) combined years (2021 and 2022). Histograms along the diagonal represent frequency distributions of YR AUDPC, while scatter plots of YR AUDPC among replicates are shown in the lower-left panels. Pearson's correlation coefficients for AUDPC between corresponding replicates (A&B) and years (C) are displayed in the upper-right panels. *** Significant correlations at $P < 0.001$

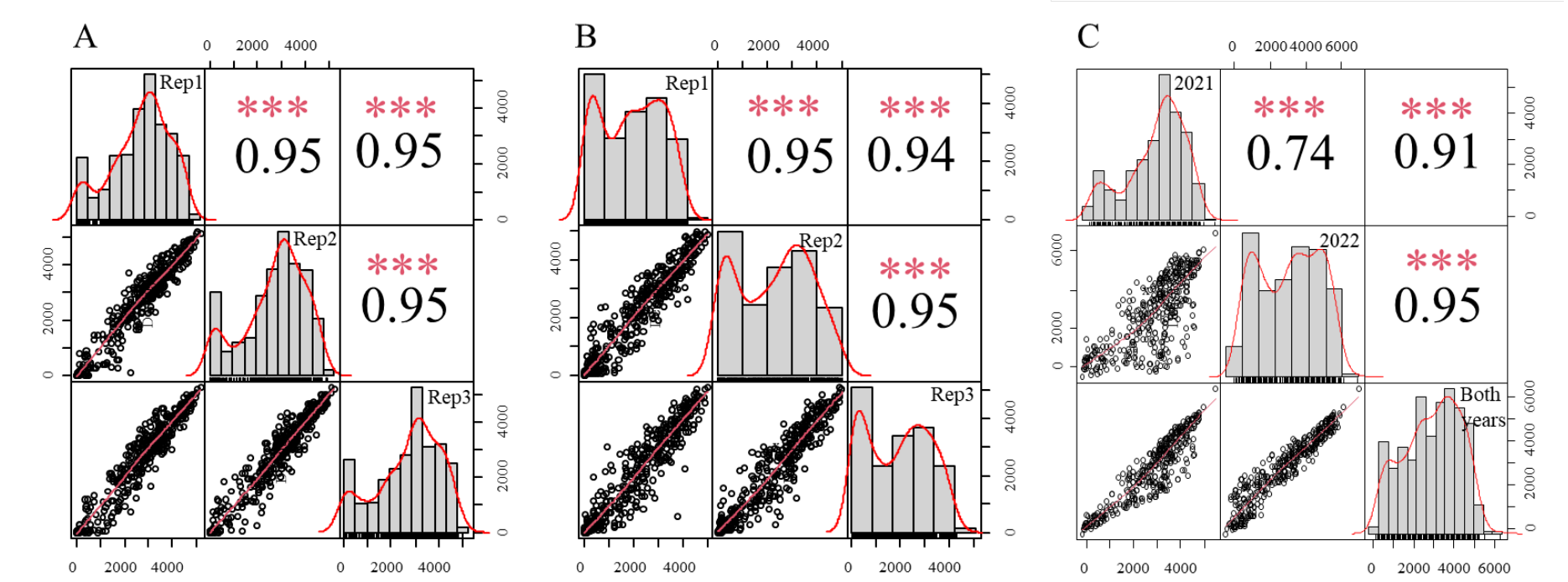


Figure S2. Frequency of lines with different seedling yellow rust infection type (IT) for the *Triticale2015a* and *Triticale2015b* races.

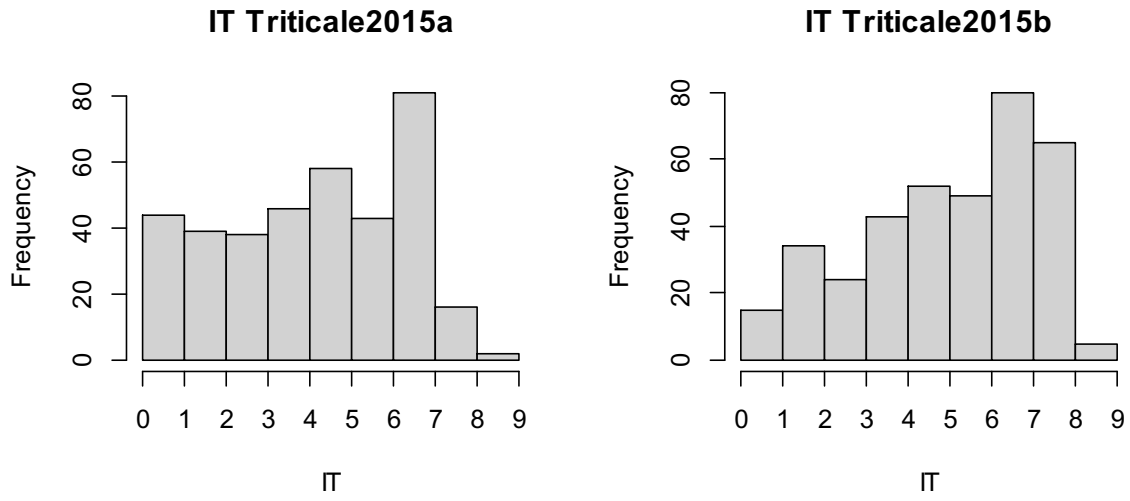


Figure S3. Distribution of field yellow rust residuals' values according to the base and models with spatial column information for 2021 (A) and 2022 (B). Histograms of residuals according to the base model and models with spatial information for 2021 (C) and 2022 (D) for the variable area under the disease progress curve (AUDPC) of the 366 wheat lines.

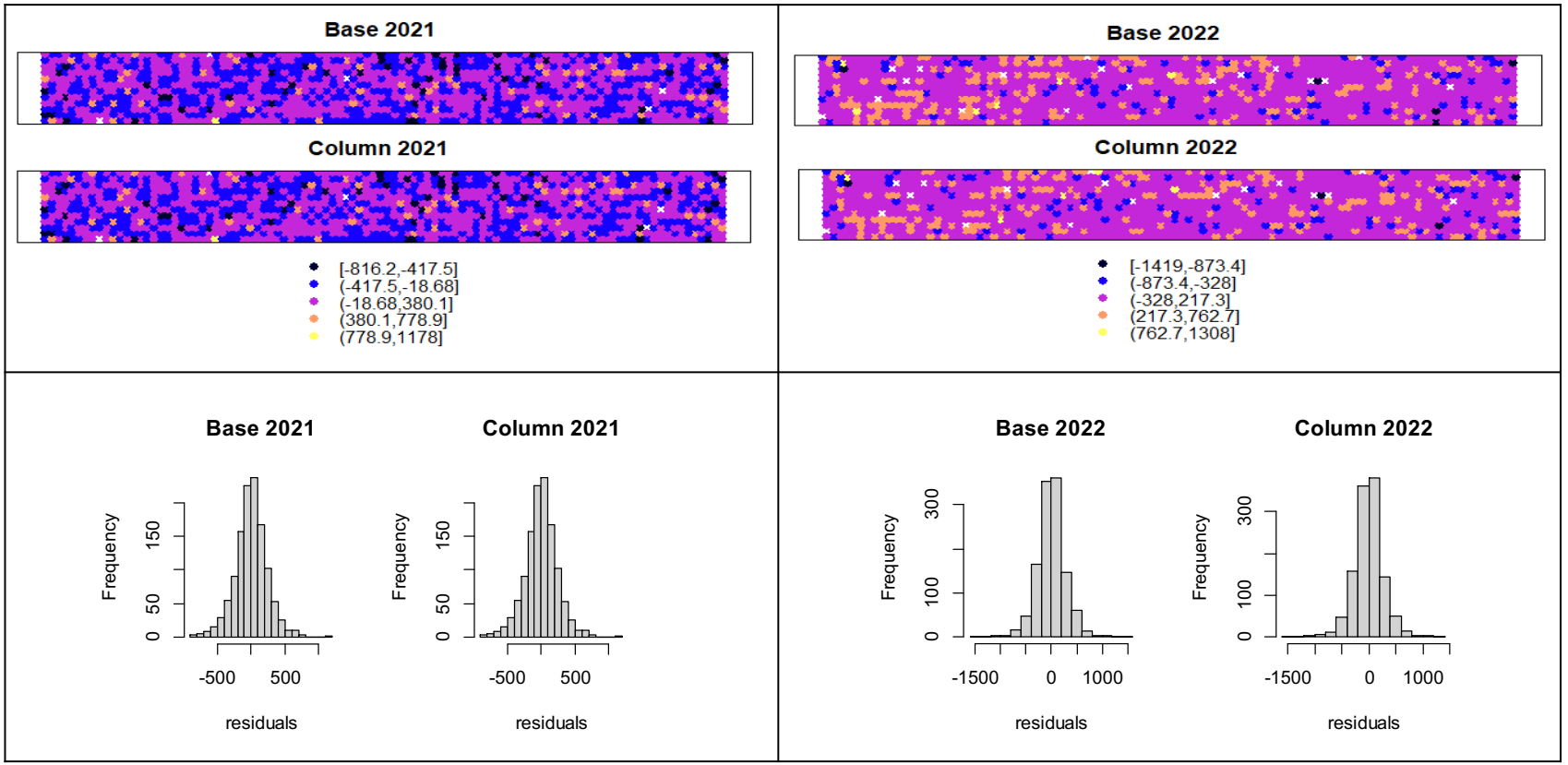


Figure S4. SNP density plot showing the distribution of the 156,034 SNPs by wheat chromosome.

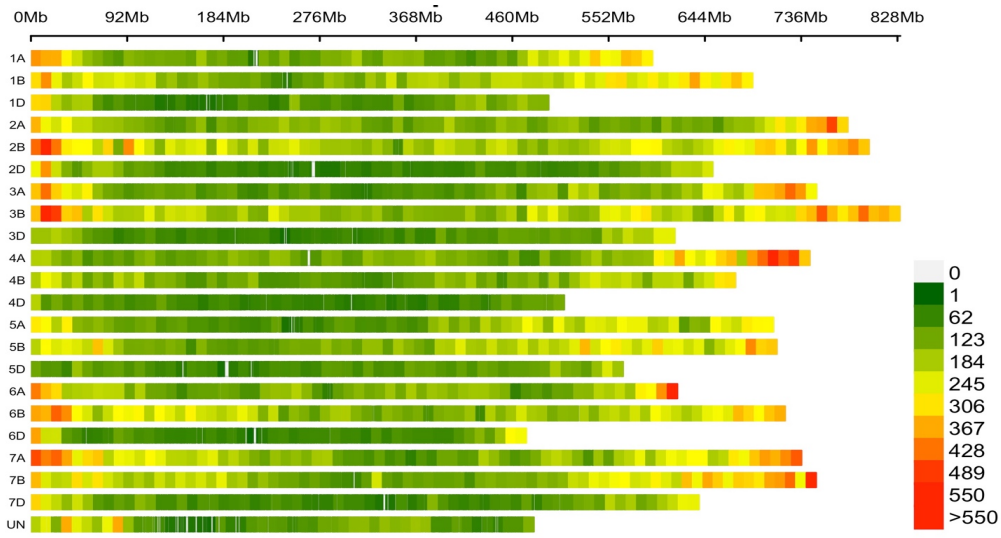


Figure S5. Heatmap of Euclidean distances where lines are ordered bases on cluster analysis among the 366 wheat lines of the GWAS and GP panel (A). Principal co-ordinate analysis (PCoA) for the 366 wheat lines of the GWAS and GP panel, colors indicate the AUDPC value for each line on a temperature scale from green (low) to red (high) (B). PCoA where colors indicate the origin of the lines (lines from National Institute of Agronomical Research (INIA) - Resistant Germplasm Development Program (INIA-RGDP); advanced, elite, and released lines of INIA - Wheat Breeding program (INIA-WBP); commercial varieties from other breeding programs that have been grown in Uruguay and checks) (C). Values of the delta k from Admixture (D).

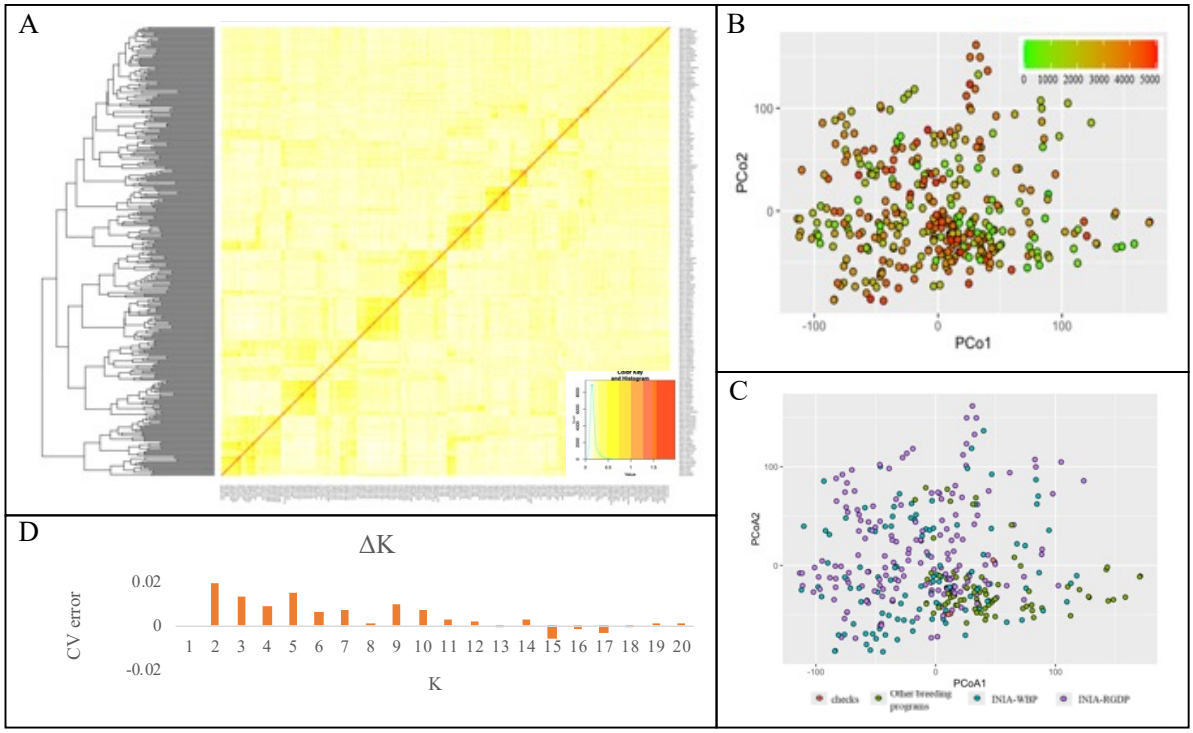


Figure S6. Manhattan plots for yellow rust (YR) resistance based on infection type (IT) values for race *Triticale2015a* (A) *Triticale2015b* (B) and for days to heading (C) in 366 wheat lines of the panel. Horizontal line indicating the genome-wide significance threshold.

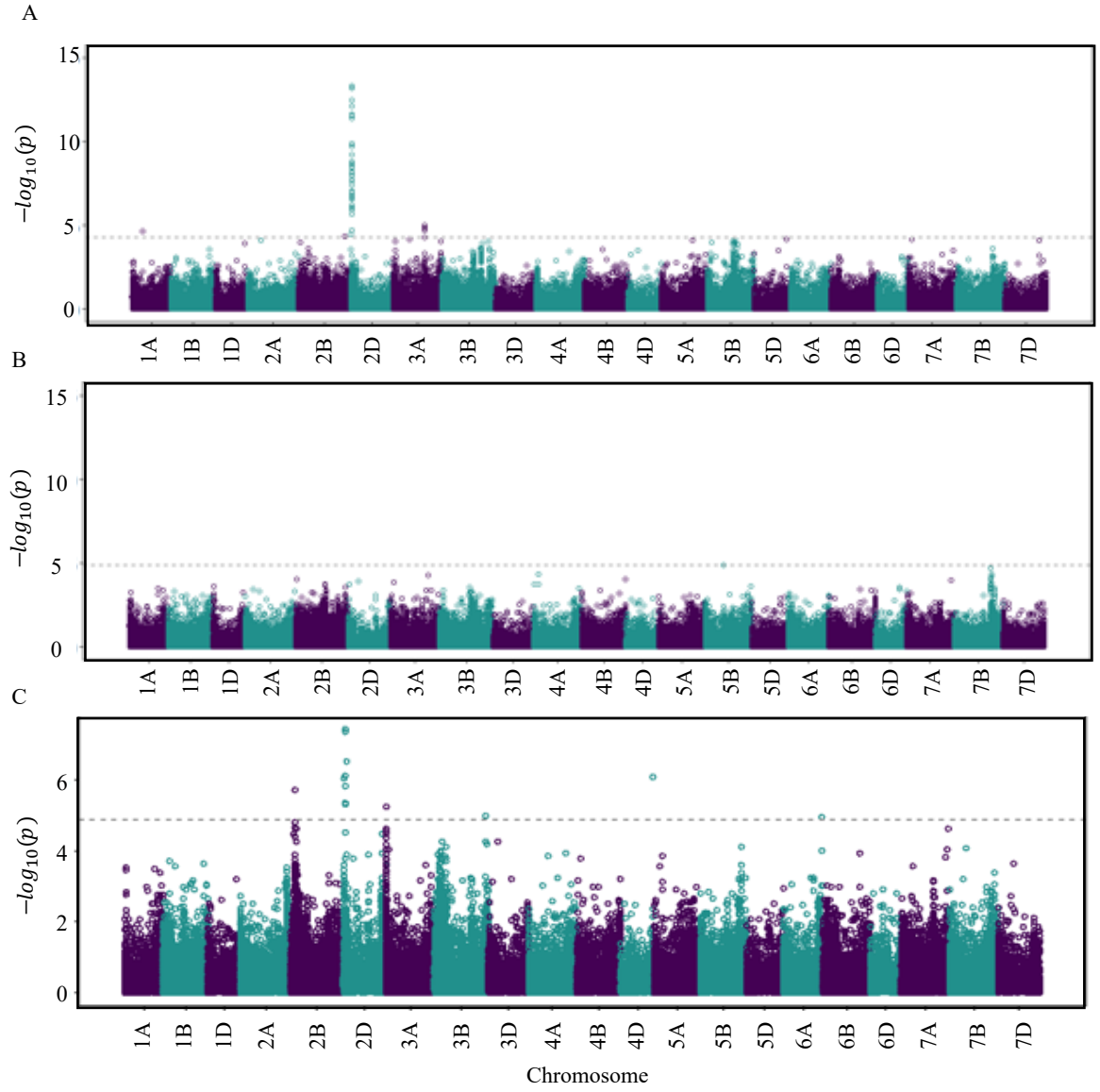
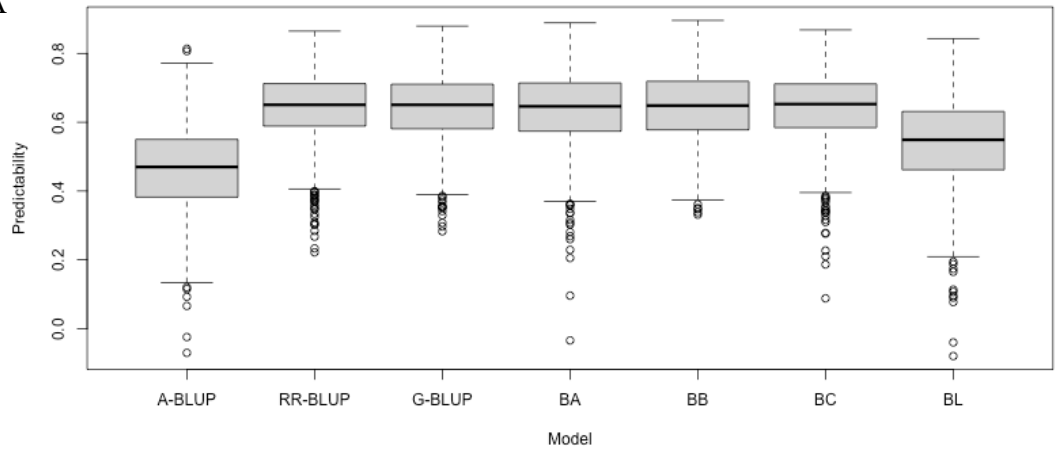


Figure S7. Predictive ability expressed as the Pearson's correlation between observed and predicted values (A), and mean squared error (MSE, B) of seven genomic prediction (GP) models for yellow rust (YR) resistance based on area under the disease progress curve (AUDPC) values in the field evaluations. Models compared include Pedigree-based (A-BLUP), G-BLUP, RR-BLUP, Bayesian A (BA), Bayesian B (BB), Bayesian C (BC), and Bayesian LASSO (BL). Results are based on a 10-fold cross-validation scheme with 100 iterations. Boxplots show the distribution of a dataset through five key summary statistics: minimum (lower whisker), first quartile (bottom of the box), median (line inside the box), third quartile (top of the box), and maximum (upper whisker). Points beyond the whiskers are values outside 1.5 times the interquartile range from the quartiles.

A



B

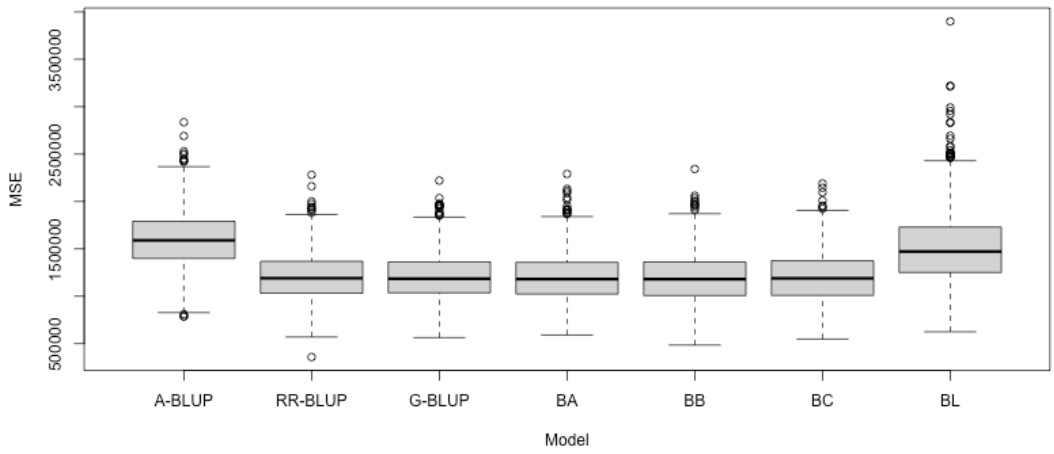


Figure S8. Predictive ability expressed as the Pearson's correlation between observed and predicted values (A), and mean squared error (MSE, B) of the G-BLUP model prediction for yellow rust (YR) resistance based on area under the disease progress curve (AUDPC) values in the field evaluations incorporating QTL as fixed effects. The model progressively incorporates up to five QTL identified via genome-wide association study (GWAS) as fixed effects. Results are based on a 10-fold cross-validation scheme with 100 iterations. Boxplots show the distribution of a dataset through five key summary statistics: minimum (lower whisker), first quartile (bottom of the box), median (line inside the box), third quartile (top of the box), and maximum (upper whisker). Points beyond the whiskers are values outside 1.5 times the interquartile range from the quartiles.

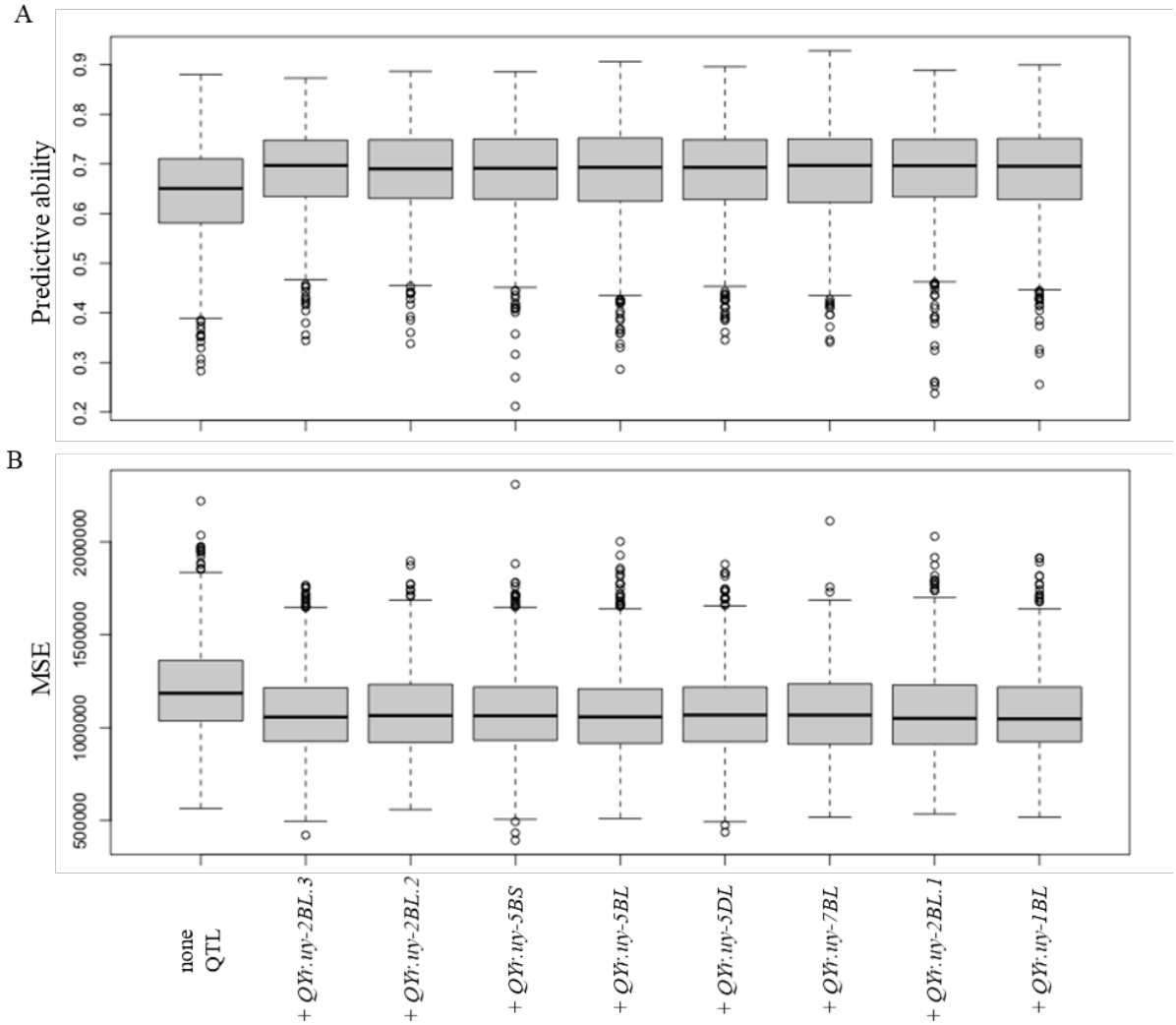


Figure S9. Quantile–quantile (QQ) plots of observed vs. expected p-values, obtained from the GWAS model used to detect QTL for the variables (A) field AUDPC, (B) seedling IT for the *Triticale2015a* race, (C) seedling IT for the *Triticale2015b* race.

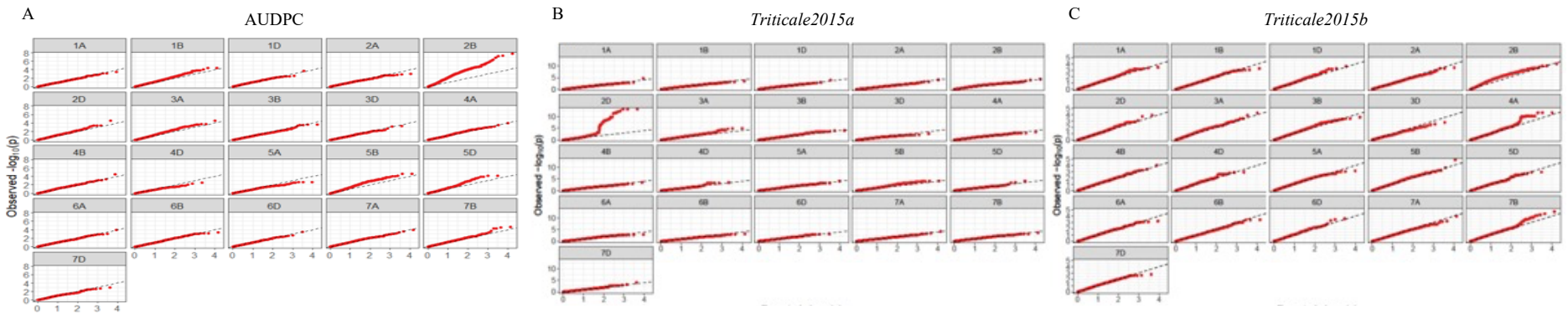


Table S1. Phenotypic data: adjusted means of field data from both years combined (AUDPC) and seedling test (IT), days to heading, presence/absence information for the identified QTL and information on the allele (1, favorable YR resistant allele; 2, YR susceptible allele), number of favorable alleles per line, and presence/absence of Yr18, Yr29 and Yr46 genes by KASP markers. NA, missing information.

		Field data			Seedling tests data		QTL for field AUDPC								Number of	L for seedling IT *Triticale201*		KASP markers		
line ID	**line origin**	**heading.date.21&22**	**AUDPC.base.21&22**	**DS.base.21&22.final**	**IT_*Triticale2015a***	**IT_*Triticale2015b***	***QYr.uy-1BL***	***QYr.uy-2BL.1***	***QYr.uy-2BL.2***	***QYr.uy-2BL.3***	***QYr.uy-5BS***	***QYr.uy-5BL***	***QYr.uy-5DL***	***QYr.uy-7BL***	**favorable QTL**	***QYr.uy-3AL***	***QYr.uy-2DS***	**KASP-*Yr18***	**KASP-*Yr29***	**KASP-*Yr46***
MA1_MOROCCO	included in the panel of 366 lines)	124.85	5142	100	8.5	8.5	*NA*	*NA*	*NA*	*NA*	*NA*	*NA*	*NA*	*NA*	*NA*	*NA*	*NA*	NA	NA	NA
MA3_THATCHER	checks	163.77	4808	89	7.5	7.75	1	2	2	2	2	1	2	2	2	2	2	NA	0	0
MA13_THATCHER*Yr18*	checks	159.77	3651	68	6	6.75	1	2	2	1	2	1	2	2	3	2	2	NA	0	0
MA15_THATCHER*Yr46*	checks	158.12	3315	62	5.5	5	1	2	2	2	2	1	2	2	2	2	2	NA	0	1
MA19_R08F54808	INIA-RGDP	134.49	2819	61	5	5.5	1	2	2	1	2	1	1	1	5	2	2	1	0	NA
MA20_R07F54236	INIA-RGDP	129.85	2551	45	7	7	2	1	2	1	2	1	2	1	4	2	2	0	1	0
MA21_R08F5862	INIA-RGDP	135.51	5211	96	6.5	6.5	1	2	2	2	2	2	1	1	3	2	2	NA	1	0
MA22_R08F53972	INIA-RGDP	128.82	2363	55	7	6	1	2	2	1	1	1	1		5	2	2	NA	1	0
MA23_R09F55051	INIA-RGDP	127.20	2326	42	4	4.5		2	2	2	2	2	1		1	2	2	NA	1	0
MA24_R09F54583	INIA-RGDP	160.06	3917	67	2	5	1	2	2	2	1	1	1	1	5	2	1	0	1	0
MA25_R07F53696	INIA-RGDP	137.87	4603	91	6.25	7.25	1	2	2		1	2	1	1	4	2	2	0	1	NA
MA26_R11F510405	INIA-RGDP	127.60	4659	90	6.75	7	1	2	2	2	2	1	1	1	4	2	2	1	1	0
MA27_R10F58420	INIA-RGDP	128.00	848	17	3.25	2.75	1	2	2	1	1	1	1		5	1	2	0	1	0
MA28_R12F56063	INIA-RGDP	158.17	2707	42	4.75	6.25	1	2	2	1	2	1	1		4	1	2	0	1	0
MA29_R12F56156	INIA-RGDP	156.27	4633	91	3.5	6	1	2	2	2	2	1	1	2	3	1	1	0	1	0
MA30_R12F56172	INIA-RGDP	126.18	4547	87	3.5	5.5	1	2	2	1	2	1	1	2	4	NA	2	NA	1	0
MA31_R12F56218	INIA-RGDP	145.02	2912	46	3.5	3.5	1	2	2		2	1	1	1	4	NA	2	0	1	0
MA32_R12F56549	INIA-RGDP	120.50	4630	89	1.5	6.25	1	2	2	2	2	1	1	1	4	2	1	1	1	0
MA33_R08F51944	INIA-RGDP	133.12	4759	91	6	6	1	2	2	2	2	2	2	1	2	2	2	0	1	0
MA34_R11F10775	INIA-RGDP	124.68	5373	93	7	7	1	2	2	2	2	1		2	2	2	2	1	1	0
MA35_R15F56228	INIA-RGDP	137.72	4856	88	8	8	1	2	2		2	2	2	1	2	2	2	0	1	0
MA36_R15F56982	INIA-RGDP	161.12	4436	83	1.5	6.25	1	2	2	2	2	2	1	1	3	2	1	0	1	0
MA37_R15F57058	INIA-RGDP	127.22	2342	53	2.25	2.25	1	2	2	2	1	1	1	2	4	2	2	1	1	0
MA38_R08F52344	INIA-RGDP	132.22	876	14	1.75	4	1	2	2	1	2	1	1	1	5	1	1	1	1	0
MA39_R13F56299	INIA-RGDP	132.26	1437	26	3.75	3.25	1	2	2	1		1	1		4	1	2	0	1	0
MA40_R15F56735	INIA-RGDP	144.74	3031	64	1.75	3.75	1	2	2	2	2	2	1	2	2	2	1	0	1	NA
MA41_R08F54562	INIA-RGDP	132.96	4231	85	6	8	1	2	2		2	1	1	1	4	2	2	1	1	0
MA42_R08F5971	INIA-RGDP	151.48	3223	61	7	8	1	2	2	2		1	1	2	3	2	2	1	0	0
MA43_R08F51512	INIA-RGDP	157.26	1291	25	0.5	2.5	1	2	2	1	1	1	1	2	5	1	1	0	1	0
MA44_R08F51349	INIA-RGDP	159.83	2767	52	6	6.25	1	2	2		2	1	1		3	1	2	0	1	0
MA45_R08F54448	INIA-RGDP	129.13	2618	37	4.25	6	1		2		1	1	1	1	5	2	2	0	1	0
MA46_R07F53531	INIA-RGDP	133.01	4258	85	7	8	1	2	2	2	1	1	2	1	4	2	2	0	1	0
MA47_R08F53750	INIA-RGDP	119.66	3099	57	8	8	2	2	2	2	2	2	1	1	2	1	2	NA	1	0
MA48_R10F58365	INIA-RGDP	128.09	3108	56	3	5	1	2	2	1	2	1	2	2	3	1	2	0	1	0
MA49_R10F58449	INIA-RGDP	123.57	3039	56	3	4.5	1	2	2	1	2	1	1	1	5	1	1	0	1	0
MA50_R10F58463	INIA-RGDP	123.20	1756	27	3.5	4.25	1	2	2	1	2	1	1	2	4	1	1	0	1	0
MA51_R10F58531	INIA-RGDP	155.94	4202	78	6.5	7.5	1	2	2	2	2	1	1	2	3	2	2	0	1	0
MA52_R11F510277	INIA-RGDP	118.26	309	4	3.75	6.75	1	1	2	1	2	1	1	1	6	2	1	1	0	NA
MA53_R11F510319	INIA-RGDP	121.34	934	9	4.25	3	1	1	2	2	2	1	1	2	4	2	1	1	0	0
MA54_R11F510940	INIA-RGDP	130.67	5224	93	7	8	1	2	2	2	2	1	2	2	2	2	2	1	1	0
MA55_R08F52346	INIA-RGDP	135.20	672	10	1	3.75	1		2	1	1	1	1	1	6	1	2	1	1	0
MA56_R09F54604	INIA-RGDP	130.43	3708	68	3	3.75		2	2	2	1	1	1	1	4	1	2	0	1	0
MA57_R09F54722	INIA-RGDP	153.24	1356	22	1.5	2.25	1	2	2	2	1	1	1	2	4	2	1	1	1	0
MA58_R10F58440	INIA-RGDP	125.57	1959	49	4.25	3.75	1	2	2	1	2	1	1	1	5	1	2	1	1	0
MA59_R10F58560	INIA-RGDP	127.05	3631	65	2.75	5.75	1	2	2	2	1	1	1	2	4	2	NA	0	1	0
MA60_R10F58407	INIA-RGDP	127.40	1633	33	2	3.25	1	2	2	1	1	1	2	1	5	1	2	0	NA	0
MA61_R12F56013	INIA-RGDP	126.67	4018	82	4.5	4	1	2	2		2	1	1	1	4	2	2	1	1	0
MA62_R12F56017	INIA-RGDP	122.88	3818	79	4.5	4.5	1	2	2	2	2	1	1	1	4	2	2	1	1	0
MA63_R12F56481	INIA-RGDP	134.08	3120	62	5	4.5	1	2	2	1	2	1	1	1	5	2	2	1	1	0
MA64_R12F56548	INIA-RGDP	118.18	3901	75	7	6.5		2	2	2	1	1	1	1	4	2	2	1	1	0
MA65_R13F56001	INIA-RGDP	134.86	1356	22	2.25	2.25	2	2	2	2	1	1	1	1	4	1	2	0	1	0
MA66_R13F56011	INIA-RGDP	128.99	4366	91	4	4	2	2	2	2	2	2	1		1	2	2	0	NA	NA
MA67_R13F56076	INIA-RGDP	152.31	2633	58	4.5	5	1	2	2	1	1	1	1	1	6	1	2	0	1	0
MA68_R13F56079	INIA-RGDP	152.38	4271	77	6.5	7	1	2	2	2	2	1	1	1	4	2	2	0	1	NA
MA69_R13F56248	INIA-RGDP	126.14	1802	38	5	4.5	1	1	2	1	1	1	1	1	7	2	2	1	1	0
MA70_R13F56389	INIA-RGDP	122.27	3476	70	4	4.5	1	2	2	2		1	2	2	2	2	1	0	1	0
MA71_R13F56566	INIA-RGDP	126.65	3760	72	5.5	6.75	1	1	2		2	1	2	2	3	2	NA	1	1	0
MA73_R09F5891	INIA-RGDP	151.62	2561	48	1.5	4	1	1	2	2	1	1	1	2	5	2	1	1	1	0
MA74_R09F54821	INIA-RGDP	158.17	2579	50	1	4.5	1	2	2	2	1	1	1	1	5	2	1	0	1	0
MA75_R09F54877	INIA-RGDP	153.41	1596	25	1	2	1		2	2	1	1	1	2	4	NA	1	0	1	0
MA77_R10F58488	INIA-RGDP	122.05	2242	40	1.5	3.75	1	2	2	2	1	1	1	1	5	2	NA	0	1	0
MA78_R12F56169	INIA-RGDP	128.44	2213	41	3.5	4.75	1	2	2	2	2	1	1	1	4	1	2	0	1	0
MA79_R12F56349	INIA-RGDP	142.73	3743	73	4.5	6.75		2	2	2	2	1	1	1	3	2	2	NA	1	0
MA80_R12F56485	INIA-RGDP	123.04	3302	67	5	5	2	2	2	2	2	1	1	1	3	2	2	NA	1	0
MA81_R12F56554	INIA-RGDP	124.98	3600	73	5	5	1	2	2	2	2	1	1	1	4	2	2	0	1	0
MA82_R12F56586	INIA-RGDP	127.68	3688	68	5.75	6	1	2	2	1	2	1	1	2	4	2	2	0	1	0

MA83_R13F56043	INIA-RGDP	156.51	949	10	1.5	2	1	2	2	1	2	1	1	1	5	1	2	0	1	0
MA84_R13F56055	INIA-RGDP	151.14	2414	47	6	5.5	1	1	2	2	1	1	1	1	6	2	2	0	1	0
MA85_R13F56263	INIA-RGDP	123.00	3645	67	7.5	7.25	1	2	2	2	1	1	1	1	5	2	2	0	1	0
MA86_R13F56443	INIA-RGDP	152.41	772	5	0.5	3.75	1	2	2		1	1	1	2	4	2	1	0	1	NA
MA87_R13F56460	INIA-RGDP	127.92	3115	62	5	5.5	1	2	2	1	2	1	1	2	4	2	NA	0	1	0
MA88_R13F56607	INIA-RGDP	127.33	1975	35	2	2	1	2	2	2	2	1	2	2	2	2	1	0	1	0
MA89_R10F57559	INIA-RGDP	156.55	3873	72	3.25	4	1	2	2	2	1	1	1	2	4	2	2	NA	1	NA
MA90_R10F57568	INIA-RGDP	162.75	3870	69	2.75	2	1	2	2	2	1	1	1	1	5	2	2	0	1	0
MA91_R14F512139	INIA-RGDP	154.71	3761	69	4.75	4.75	2	1	2	2	2	1	2	2	2	2	2	0	1	0
MA92_R14F512206	INIA-RGDP	123.43	4991	87	6.25	6.25	2		2	2	2	1	1	2	2	1	2	0	1	0
MA93_R14F512230	INIA-RGDP	154.12	2904	55	5.5	5.5	1	2	2	1	1	1	2	1	5	2	2	NA	1	0
MA94_R14F512238	INIA-RGDP	134.99	3761	72	8.25	8	2	2	2	2	2	1	1	1	3	2	2	1	1	0
MA95_R14F512252	INIA-RGDP	134.50	5014	95	8	7.5	2	2	2	2	2	2	1	1	2	2	2	0	1	0
MA96_R14F512281	INIA-RGDP	130.43	4849	86	7.5	7	2	2	2	2	2		2	1	1	2	2	NA	1	0
MA97_R14F512289	INIA-RGDP	137.34	4036	72	7	7	2	2	2	2	2	1	1	2	2	2	2	NA	1	0
MA98_R14F512381	INIA-RGDP	131.78	2368	48	6	6	2	2	2	2	2	1	1	1	3	2	2	1	1	0
MA99_R14F512522	INIA-RGDP	121.56	1466	22	5.5	5	2	2	2	2	2	2		1	1	2	2	1	1	0
MA100_R15F56081	INIA-RGDP	129.43	3788	68	7	6.25	1	2	2		2	1	2	2	2	2	2	1	1	0
MA102_R15F56324	INIA-RGDP	122.51	3890	72	7	7.5	2	2	2	2	1	1	2	1	3	2	2	1	1	0
MA103_R15F56334	INIA-RGDP	135.63	3468	73	6.5	6.75	1	2	2	2	1	1	2	1	4	2	2	1	0	0
MA104_R15F56341	INIA-RGDP	142.15	2019	52	5	5	2	2	2	2	1	1	2	1	3	2	2	1	1	0
MA105_R15F56382	INIA-RGDP	143.31	1703	34	4.5	4.5	1		2	2	2	1	2	1	3	2	2	1	NA	0
MA106_R15F56468	INIA-RGDP	125.80	3691	66	7.5	7.5	1	1	2	2	1	1	2	1	5	2	2	0	1	0
MA107_R15F56594	INIA-RGDP	157.53	2053	40	3.75	3.5	1	1	1	2	1	1	1	1	7	2	2	0	1	0
MA108_R15F56598	INIA-RGDP	160.06	2190	47	1	5.75	1	1	1	1	1	1	2	2	6	1	1	NA	1	0
MA109_R15F56664	INIA-RGDP	154.87	3937	81	3.5	7.25	1	2	2	2	2	2	2		1	2	1	NA	0	0
MA110_R15F56673	INIA-RGDP	148.81	4112	86	5.25	7.25	1	2	2	2	2	2	2	1	2	2	2	0	1	0
MA111_R15F56731	INIA-RGDP	149.73	3349	69	2.25	7.25	1	2	2	2	2	2	1	2	2	2	1	NA	1	0
MA112_R15F56797	INIA-RGDP	131.81	2552	48	5.75	5.25	2	2	2	2	2	2	2	1	1	2	NA	0	1	0
MA113_R15F56868	INIA-RGDP	141.28	3629	66	7.25	7.5	1	2	2		2	1	2	1	3	2	2	1	0	0
MA114_R15F56960	INIA-RGDP	129.84	3991	74	6.5	6.75	1	2	2	2	2	2	1	1	3	2	2	0	1	0
MA116_R15F57247	INIA-RGDP	125.11	2861	56	5.5	7.5	1	1	2	2	2	1	2		3	1	2	1	1	0
MA117_R19F57474	INIA-RGDP	154.24	3920	83	3.5	6.5	1	2	2	1	2	1	2	1	4	2	1	NA	1	0
MA118_R16F57064	INIA-RGDP	152.20	3197	63	4	4	1	2	2	2	2	2	2		1	2	2	NA	0	1
MA119_R16F57073	INIA-RGDP	151.65	2346	42	3	3.5	1	2	2	1	2	1	2	2	3	2	2	NA	0	0
MA120_R16F57122	INIA-RGDP	128.39	3817	80	6.75	7.5	1	1	1	2	1	1	1	1	7	1	2	0	1	0
MA121_R17F57066	INIA-RGDP	139.32	699	8	3.25	3.5	1	1	1	1	1	1	1	2	7	2	2	0	1	0
MA122_R17F57103	INIA-RGDP	134.17	623	14	2.75	3.25	1	2	1	1	1	1	1	1	7	2	2	1	1	0
MA123_R17F57130	INIA-RGDP	137.83	2237	43	2.75	3	1	2	2	2	2	1	1	1	4	2	2	0	1	0
MA124_R17F57132	INIA-RGDP	122	0	0	2.5	2	1	1	1	1	1	1	1	1	8	2	2	1	1	0
MA125_R17F57318	INIA-RGDP	136.92	2212	36	0.5	1.5	1	2	2		2	1	1	1	4	1	2	1	1	0
MA126_R17F57367	INIA-RGDP	132.25	1382	31	3.5	2.5	1	1	1	2	2	1	1	1	6	1	2	1	1	0
MA127_R18F57005	INIA-RGDP	137.08	1229	14	7	6.25	1	2	2	1	2	1	1	1	5	2	2	NA	1	0
MA128_R18F57010	INIA-RGDP	128.80	342	0	4	4	1	2	2	1	2	1	1	1	5	2	2	0	1	0
MA129_R18F57014	INIA-RGDP	129.87	483	6	0	0	1	2	2	1	2	1	1	1	5	1	2	0	1	0
MA130_R18F57033	INIA-RGDP	137.85	1246	21	4.75	5.5	1	2	2	1	2	1		1	4	2	2	0	1	0
MA131_R18F57049	INIA-RGDP	132.48	1527	26	6	6	1	2	2	1	2	1	1	1	5	2	2	0	1	0
MA133_R18F57179	INIA-RGDP	138.86	1628	39	2.75	3.75	1	2	2	2	2	1	1	2	3	2	2	1	1	0
MA134_R18F57252	INIA-RGDP	123.67	2444	63	0.5	1	1	2	2	2	2	1	1	1	4	2	1	1	1	0
MA135_R18F57270	INIA-RGDP	121.97	1778	36	0.5	1	1	2	2	2	2	1	1	1	4	2	2	NA	1	0
MA136_R18F57294	INIA-RGDP	127.49	2186	44	1.25	1.5	1	2	2	2	1	1	1	1	5	1	1	0	1	0
MA137_R18F57342	INIA-RGDP	139.57	2143	41	1	1.5	1	2	2	2	1	1	1	1	5	1	NA	1	1	0
MA138_R18F57360	INIA-RGDP	129.98	3182	63	1	6.5	1	2	2	2	2	1	1	1	4	1	1	1	1	0
MA139_R18F57386	INIA-RGDP	138.01	1307	27	1	2	1	2	2	2	1	1	1	1	5	1	2	1	1	0
MA140_R18F57463	INIA-RGDP	118.91	1537	28	1	2	1	2	2	2	1	1	1	1	5	1	2	1	1	0
MA141_R18F57543	INIA-RGDP	136.16	3960	84	3.75	5.5	1	2	2	2	1	1	1		4	2	2	0	1	0
MA142_R18F57612	INIA-RGDP	134.08	1174	15	5.25	5.75	1	2	2	1	1	1	1	1	6	2	2	0	1	0
MA143_R18F57641	INIA-RGDP	139.16	430	1	3.75	3.75	1	1	1	1	2	2		1	5	2	2	NA	1	0
MA144_R18F57671	INIA-RGDP	131.50	723	9	5	5	1	1	1		1	1	2	1	6	2	2	NA	1	0
MA145_R18F57778	INIA-RGDP	131.90	1157	30	1	2	1	1	1	1	2	1	1	1	7	2	2	1	1	0
MA146_R18F57786	INIA-RGDP	128.89	1647	36	1.25	2	1	2	2	2		1	1	1	4	2	2	1	1	0
MA147_R18F57790	INIA-RGDP	134.08	1304	27	1.25	1	1	1	1		1	1	2	1	6	2	2	1	NA	0
MA149_R18F57923	INIA-RGDP	134.43	2867	62	4	5	1	2	2	2	1	1	1	1	5	2	2	1	1	0
MA150_R18F57931	INIA-RGDP	130.45	3600	76	2.25	7	1	2	2	2	1	1	1	1	5	1	1	0	1	0
MA151_R18F58178	INIA-RGDP	130.76	1916	40	5	5	1	1	1	2	1	1	1	1	7	2	2	1	1	0
MA153_R18F58283	INIA-RGDP	135.61	2681	59	6.5	7	1	2	2	2	1	1	1	1	5	2	2	NA	1	0
MA154_R18F58293	INIA-RGDP	119.34	1556	25	1.5	7.5	1	2	1	1	1	1	1	1	7	2	2	1	1	0
MA155_R18F58337	INIA-RGDP	135	1621	25	1	1	1	1	1	1	1	1	1	1	8	2	2	0	1	0
MA156_R18F58441	INIA-RGDP	131.34	1995	43	1	1	1	1	1	2	1	1	1	1	7	2	2	0	1	0
MA157_R18F58510	INIA-RGDP	148.96	357	3	0.25	1.5	1	2	1	2	2	1	1	1	5	2	1	0	1	0
MA158_R18F58512	INIA-RGDP	156.43	768	10	1.75	2.5	1			2	2	1	1	1	4	2	2	0	1	0
MA159_R07F421370	INIA-RGDP	133.80	762	8	4	4.75	2	1	2		1	1	2	1	4	2	NA	1	1	0
MA160_R07F422985	INIA-RGDP	155.20	3180	59	7	7	1	2	2	2	2	1	1	2	3	2	2	1	0	0
MA161_R07F53736	INIA-RGDP	155.25	2835	53	6.75	7.25	1	2	2	1	1	1	1	2	5	2	2	1	0	0
MA162_R09F54906	INIA-RGDP	159.55	1616	31	2	6.75	1	2	2	2	2	1	1	2	3	1	1	0	1	0
MA163_R08F61257	INIA-RGDP	149.88	3423	69	7	7.25	1	2	2	2	2	1	1	2	3	2	2	0	1	0
MA164_R13F56591	INIA-RGDP	128.39	4029	76	4	8.25	1	1	2	2	2	1	1	1	5	2	1	1	1	0
MA165_R13F57859	INIA-RGDP	123.11	4700	86	7	7.5	1	2	2	2	2	1	2	2	2	2	2	1	1	0
MA166_R14F512232	INIA-RGDP	148.96	2284	34	7.25	6.75	1	2	2	1	1	1	2	1	5	2	2	1	1	0
MA167_R16F57998	INIA-RGDP	155.13	3823	70	6.5	8	1	2	2	2	2	1	1	1	4	2	2	NA	NA	0
MA168_R16F59384	INIA-RGDP	139.54	2486	45	4	5	1	2	2	2	1	1	1	1	5	2	2	0	1	0
MA169_R17F57034	INIA-RGDP	127.99	3340	72	7.5	7.5	1	2	2	1	2	1	2	1	4	2	2	1	0	0
MA170_R19F57046	INIA-RGDP	131.50	3507	71	0.5	6.75	1	2	2	2	1	1	1	2	4	1	1	1	1	0
MA171_R19F57142	INIA-RGDP	155.26	2356	46	2.75	3	1	2	2	2	1	1	1	2	4	2	2	1	1	0
MA172_R19F57176	INIA-RGDP	126.36	2996	65	1.5	5.75	1	2	1	2	1	1	1	1	6	2	1	1	1	0
MA173_R19F57238	INIA-RGDP	130.49	1700	29	2.75	4	1	1	1	2	1	1	1	1	7	2	2	0	1	0
MA174_R19F57313	INIA-RGDP	156.76	1925	41	3	2	1	1	1	2	1	1	1	1	7	2	2	1	1	0
MA175_R19F57414	INIA-RGDP	136.24	3565	77	2.25	4	1	2	2	2	2	1	1	1	4	1	2	NA	1	0
MA176_R19F57426	INIA-RGDP	135.90	700	4	4.25	7.25		1	1	1	1	1	1	1	7	2	2	0	1	0
MA177_R15F57341	INIA-RGDP	138	436	1	3.25	5	1	1	1	1	1	1	1	1	8	2	2	1	1	0
MA178_R19F57511	INIA-RGDP	136.92	3957	82	1	6.75	1		2	2	1	1	1	2	4	2	1	0	1	0
MA179_R19F57515	INIA-RGDP	131.81	1839	41	2.25	4.5	1	2	1	1	1	1	1		6	2	2	0	1	0
MA180_R19F57592	INIA-RGDP	136.48	2346	43	5	6.5	1	2	2	1	2	1	1	2	4	2	2	0	1	0
MA181_R19F57619	INIA-RGDP	136.71	1342	16	5.5	5.5	1	2	2	1	2	1	1	1	5	2	2	0	1	0
MA182_R19F57638	INIA-RGDP	140.84	766	16	4.75	4.5	1	2	2	2	1	1	1	1	5	2	2	1	NA	NA
MA183_R19F57754	INIA-RGDP	134.30	1638	34	0.5	2	2		1	1	1	1	1	1	6	2	2	0	1	0
MA184_R19F57767	INIA-RGDP	132.16	2371	50	0.5	1.5	2	1	1	1	1	1	1	1	7	2	2	0	1	0
MA185_R19F57810	INIA-RGDP	132.12	3396	76	1	4	2	2	2	2	1	1	2	1	3	2	1	1	1	0
MA186_R19F57862	INIA-RGDP	138.06	1076	15	3.25	3.75	1	2	2	1	1	1	1	1	6	1	2	0	1	0
MA187_R19F57890	INIA-RGDP	133.64	2456	48	5.25	6	1	2	2	1	1	1	1	1	6	1	2	0	1	0

MA188_R19F57999	INIA-RGDP	132.69	2922	62	4.25	4	2	1	2	2	2	1	1	1	4	2	2	0	1	0
MA189_R19F58041	INIA-RGDP	133.44	3028	61	6.75	6.75		1	2	2		1	1	1	4	2	2	0	1	0
MA190_R19F58054	INIA-RGDP	137.17	2636	55	3.5	4.25	1	2	2	2	1	1	1	1	5	2	2	1	0	0
MA191_R19F58132	INIA-RGDP	155.61	2984	65	3.75	5.5	1	2	2	1	2	1	1	2	4	1	2	NA	1	0
MA192_R19F58178	INIA-RGDP	146.71	3630	77	2.25	6	1	2	2		1	1	1	2	4	2	1	NA	1	0
MA193_R19F58285	INIA-RGDP	132.68	355	1	2.75	2	1	2	2	2	2	1	1	1	4	1	2	NA	1	0
MA194_R19F58337	INIA-RGDP	127.74	2322	39	3.5	4	1	2	2	2	2	1	1	1	4	1	2	0	1	0
MA195_R19F58339	INIA-RGDP	126.27	823	6	3	5.5	1	2	2	2	2	1	1	1	4	1	2	0	1	0
MA196_R19F58520	INIA-RGDP	132.77	365	0	1.75	1	1	2	2	2	1	1	1	1	5	1	2	1	1	0
MA197_R19F58527	INIA-RGDP	132.39	349	2	2	1.5	1	2	2	2	1	1	1	1	5	1	2	1	1	0
MA198_LE2092-ESTANZUELA_CALANDRIA	INIA-WBP	160.45	4724	90	7.5	7	1	2	2	2	2	1	2		2	2	2	NA	1	0
MA199_LE2118-ESTANZUELA_FEDERAL	INIA-WBP	163.21	4119	75	3.25	7	1	1	2	2	1	1	1	2	5	2	1	0	0	0
MA200_LE2119-ESTANZUELA_JILGUERO	INIA-WBP	120.00	4146	81	5.5	8	2	2	2	2	2	1	1		2	2	NA	1	1	0
MA201_LE2145-ESTANZUELA_CHAJA	INIA-WBP	160.91	4667	91	0.5	9	2	2	2	2	2	2	1	2	1	1	1	0	1	0
MA202_LE2164-ESTANZUELA_HALCON	INIA-WBP	161.83	4516	88	1	8	2	2	2	2	2	2	1	2	1	1	1	NA	NA	NA
MA203_LE2172-INIA_BOYERO	INIA-WBP	124.43	489	9	3	2.5	1	2	2	1	1	1	2	1	5	1	2	1	1	0
MA204_LE2189-INIA_MIRLO	INIA-WBP	119.61	2918	62	1.5	4	1	2	2			1	1		3	1	1	1	1	0
MA205_LE2193-INIA_CAIURE	INIA-WBP	137.43	4641	90	5	5.5	1	1	2	2	2	1	2	2	3	2	2	0	1	0
MA206_LE2210-INIA_TIJERETA	INIA-WBP	155.47	3400	61	7	7.25	1	2	2		2	1	1	2	3	2	2	1	0	0
MA207_LE2245-INIA_GORRION	INIA-WBP	156.58	3854	69	2.25	6.25	1	2	2	2	1	1	1	2	4	2	1	NA	1	0
MA208_LE2249-INIA_CHURRINCHE	INIA-WBP	131.98	4621	88	5	5	1	2	2	1	1	2	1	2	4	2	2	0	0	0
MA209_LE2255-INIA_GAVILAN	INIA-WBP	161.13	3708	68	1	5.25	1	2	2	2	2	1	1	2	2	1	1	0	1	0
MA210_LE2271-INIA_TORCAZA	INIA-WBP	159.67	3087	54	3.5	5.25	1	2	2	1	2	1	1	2	4	1	2	0	1	0
MA211_LE2303-INIA_TERO	INIA-WBP	140.41	2469	39	3.75	5.5	2	2	2	2		1	1	1	3	2	2	1	1	0
MA212_LE2310-INIA_CARANCHO	INIA-WBP	138.28	4242	78	3.5	6.5	1	2	2	2	2	2	1	2	2	2	1	NA	1	0
MA213_LE2313-INIA_GARZA	INIA-WBP	161.32	2274	37	4.5	4.5	1	1	2	2	2	1	1	2	4	2	2	1	0	0
MA214_LE2325-INIA_CHIMANGO	INIA-WBP	162.04	4600	90	7	7	1	2	2	2	1	2	1	1	4	2	2	NA	0	0
MA218_LE2346-GENESIS2346	INIA-WBP	154.90	3273	67	4	4.25	1	2	2	1	2	1	1	1	5	2	1	0	1	0
MA219_LE2354-GENESIS2354	INIA-WBP	138.37	4724	89	6.75	8	2	2	2	2	2	1	1	1	3	2	NA	0	1	0
MA220_LE2358-GENESIS2358	INIA-WBP	154.97	4096	74	6.25	7.5	1	1	2	2	2	1	1	1	5	2	1	0	1	0
MA221_LE2359-GENESIS2359	INIA-WBP	155.22	4406	76	6.5	6.25	1	2	2	2	2		2		2	2	2	1	1	0
MA222_LE2366-GENESIS2366	INIA-WBP	155.30	4631	90	6.5	7.5		2	2		2	1	1	2	2	2	2	0	0	0
MA223_LE2375-GENESIS2375	INIA-WBP	130.98	4299	93	2.5	2.5	1	2	2	2	2	1	1	1	4	1	2	1	1	0
MA224_LE2377-GENESIS877	INIA-WBP	161.19	4367	87	6.5	8	2	2	2	1	2	2	1	2	2	2	NA	0	1	0
MA225_LE2381-GENESIS681	INIA-WBP	139.66	4780	97	6.75	7.5	1	2	2	2	2	1	1	1	4	2	NA	0	1	0
MA226_LE2387-GENESIS687	INIA-WBP	130.58	3863	78	6	7.5	1	2	1	2	2	2	1	1	4	2	2	0	1	0
MA227_LE2394-GENESIS794	INIA-WBP	147.01	4307	83	5.75	6.5	1	2	2	2	2	2	1	2	2	2	2	0	1	0
MA228_LE2425-GENESIS725	INIA-WBP	142.90	3597	68	5.75	7	1	1	2	2	1	1	1	2	5	2	1	0	1	0
MA229_LE2428-GENESIS628	INIA-WBP	139.52	4739	92	6.5	7.5		2	2	2	2	1	1	1	3	2	2	0	1	0
MA230_LE2433-GENESIS433	INIA-WBP	126.02	4165	87	6.5	7.5	1	2	2	2		1	1	1	4	2	2	0	NA	0
MA231_LE2438-GENESIS638	INIA-WBP	148.68	4091	73	7.5	7.5	1	1	2	2	2	1	2	2	3	2	2	1	0	0
MA232_LE2455-GENESIS555	INIA-WBP	130.04	2446	49	1.75	2	1	2	2	2	1	1	1	2	4	2	1	0	NA	0
MA233_LE2252	INIA-WBP	131.78	2650	44	5.25	7	2	1	2		2	1	2	1	3	2	NA	1	1	0
MA234_LE2294	INIA-WBP	126.07	4085	72	6.5	7.25	1	2	2	1	1	1	2	1	5	1	2	1	1	0
MA235_LE2304	INIA-WBP	139.08	2696	38	4.5	4.5	2	2	2	2		1	1	1	3	2	2	1	1	0
MA236_LE2326	INIA-WBP	153.40	3204	55	6	6.75	1	2	2	1		1	1	2	4	2	2	1	0	0
MA237_LE2349	INIA-WBP	159.30	2363	36	7	6.75	1	2	2	1	2	1	1	1	5	2	2	1	0	0
MA238_LE2357	INIA-WBP	129.03	3822	72	6.25	7	1	1	2	2	2	1	2	1	4	2	2	1	0	0
MA239_LE2378	INIA-WBP	140.06	1997	53	4.25	5	1	2	2		2	1	1	1	4	2	2	0	1	0
MA240_LE2379	INIA-WBP	156.91	4833	90	2.5	7	1	1	2	2	1	1	2	2	4	2	1	0	1	0
MA241_LE2380	INIA-WBP	156.11	2591	63	2	1.5	2	1	2	2	2	1	1	1	4	1	1	0	NA	0
MA242_LE2382	INIA-WBP	128.35	4621	88	6.75	8	2	2	2	2	2	1	1	1	3	2	1	0	1	0
MA243_LE2383	INIA-WBP	133.69	570	10	4.75	4.5	1	1	1	1	2	1	1	1	7	2	2	0	1	0
MA244_LE2384	INIA-WBP	131.59	5030	94	6.5	8	1	2	2	2	2	1	1	1	4	2	2	0	1	0
MA245_LE2385	INIA-WBP	131.84	4068	87	1.75	1	1	1	2	2	1	2	1	1	5	2	2	0	1	0
MA246_LE2386	INIA-WBP	127.22	1903	39	6.5	7	1	1	2	1	1	1	1	2	6	2	2	0	1	0
MA247_LE2389	INIA-WBP	120.22	1951	42	1	2.75	2	1	1		2	1	2	1	4	1	1	0	1	0
MA248_LE2390	INIA-WBP	125.71	3320	70	4.5	5.5	1	2	2		2	2	1	1	3	2	2	0	1	0
MA249_LE2391	INIA-WBP	151.13	2303	55	4.75	6.75	1	2	2	1	2	1	1	1	5	1	2	1	0	0
MA250_LE2392	INIA-WBP	155.71	1389	28	4.75	5	1	2	2	2	2	1	1	1	4	1	NA	0	1	0
MA251_LE2393	INIA-WBP	158.37	2687	54	4.25	7.5	2	1	2	2	1	1	1	1	5	2	1	0	1	0
MA252_LE2395	INIA-WBP	127.14	170	4	4	2.5	1	1	2	2	2	1	1	2	4	1	2	0	1	0
MA253_LE2396	INIA-WBP	128.47	213	3	2.5	3.25	1	1	2	2	2	1	1	2	4	1	2	0	1	0
MA254_LE2397	INIA-WBP	139.28	5063	96	7	8	1	2	2	2	2	1	1	1	4	2	2	0	1	0
MA255_LE2398	INIA-WBP	130.16	5043	93	6.5	6.25	1	2	2		2	1	2	2	2	1	2	0	1	0
MA256_LE2399	INIA-WBP	131.09	5338	96	6	6	1	2	2		2	1	2	2	2	1	2	0	1	0
MA257_LE2400	INIA-WBP	126.28	4438	87	6.5	6.5	1	2	2	2	2	1	2	2	2	2	2	1	1	0
MA258_LE2401	INIA-WBP	139.02	3502	68	5.75	6.25	1	2	2		2	1	1	2	3	2	2	0	1	0
MA259_LE2402	INIA-WBP	126.01	1305	38	2	4	1	2	2		2	1	1	1	4	1	1	0	1	0
MA260_LE2403	INIA-WBP	154.34	2182	49	2.5	4	1	1	2	2	2	1	1	1	5	2	1	0	1	0
MA261_LE2404	INIA-WBP	160.39	2559	40	4.5	5	1	2	2	1	2	1	1	1	5	1	1	0	NA	0
MA262_LE2405	INIA-WBP	158.17	3350	71	4.25	5	1	2	2	2	2	1	1	1	4	1	2	1	1	0
MA263_LE2406	INIA-WBP	137.73	5114	94	6.5	7	1	2	2	2	2	1	1	1	4	2	NA	1	1	0
MA264_LE2407	INIA-WBP	136.39	3067	61	6.25	7.5	2	2	2	2	2	1	1	1	3	2	2	1	0	0
MA265_LE2408	INIA-WBP	138.61	4722	92	7	7.5	1	2	2	2	2	1	1	1	4	2	NA	1	1	0
MA266_LE2409	INIA-WBP	132	492	5	2	3.5	1	1	1	1	1	1	1	1	8	2	2	0	1	0
MA267_LE2410	INIA-WBP	131.49	4717	89	6.75	7.5	2	2	2	2	2	2	1		1	2	2	1	1	0
MA268_LE2413	INIA-WBP	155.12	3850	83	2	7.5	1	2	2		1	1	1	1	5	2	NA	0	1	0
MA269_LE2414	INIA-WBP	163.51	5046	96	6.25	6.25	1	2	2	2	2	1	2	2	2	2	2	0	1	0
MA270_LE2416	INIA-WBP	128.65	784	4	4	4	1	2	2	1	2	1	1	1	5	1	2	0	1	0
MA271_LE2417	INIA-WBP	132.04	3618	71	4.75	6	2		2	2		1	2	1	2	2	2	1	1	0
MA272_LE2418	INIA-WBP	136.30	4758	93	4.75	7	1	2	2	2	2	2	1	1	3	2	2	0	1	0
MA273_LE2419	INIA-WBP	130.08	3486	67	5.75	6.5	1	2	2	2	2	1	1	1	4	2	2	0	1	0
MA274_LE2420	INIA-WBP	127.17	4213	90	6.75	8	1	2	2	2	2	1	1	1	4	2	2	0	1	0
MA275_LE2421	INIA-WBP	127.57	2800	67	3.75	4.5	1	2	2	2	2	1	1	1	4	2	2	0	1	0
MA276_LE2422	INIA-WBP	133.70	3614	65	4.25	4.5	1	2	2	2	2	1	1		3	2	2	NA	NA	0
MA277_LE2423	INIA-WBP	128.64	4745	91	5.5	6.25	1	2	2	2	2	2	1	1	3	2	2	1	NA	0
MA278_LE2424	INIA-WBP	152.26	2761	48	5.25	6	1	2	2	1	2	1	1	2	4	1	1	0	1	0
MA279_LE2426	INIA-WBP	153.31	4343	83	6.25	5.5	1	1	2	2	2	1	2	2	3	1	1	0	1	0
MA280_LE2427	INIA-WBP	131.23	4300	80	5	5.25	1	2	2		2	2	2		1	2	2	0	NA	0
MA281_LE2429	INIA-WBP	141.21	4353	84	4.5	5.75	1	1	2	2	2	1	2	2	3	2	2	0	0	0
MA282_LE2430	INIA-WBP	136.34	4344	92	3.25	4.25	1	1	2	2	2	1	1	2	4	2	2	0	0	0
MA283_LE2431	INIA-WBP	134.30	811	11	4.75	7		1	1	1	1	2	1	1	6	2	2	0	1	0
MA284_LE2432	INIA-WBP	137.49	4309	80	5.5	8	1	1	1	2	1	1	2	2	5	2	2	0	1	0
MA285_LE2434	INIA-WBP	130.36	5189	94	7.5	7.5	1	2	2		2	1	2	1	3	1	2	0	1	0
MA286_LE2435	INIA-WBP	161.08	3048	50	1.5	6	1	1	2	2	2	1	1	2	4	2	1	0	1	0
MA287_LE2436	INIA-WBP	155.46	3729	79	1.75	4.5	1	1	2	2	2	1	1	1	5	2	1	0	1	0
MA288_LE2437	INIA-WBP	136.99	2544	52	2	4.75	1	1	2	2	2	1	1	2	4	2	2	1	1	0
MA289_LE2439	INIA-WBP	133.87	2529	57	4.75	4.75	1	1	1	1	1	1	1	2	7	2	2	0	1	0
MA290_LE2440	INIA-WBP	138.22	2559	43	2	6.5	1	2	2	2	2	1	1	1	4	2	1	0	1	0

MA295_LE2445	INIA-WBP	150.01	3529	68	6.25	6.5	1		2	2	2	1	1	2	3	2	2	1	1	0
MA296_LE2447	INIA-WBP	143.88	5188	98	6.75	7.25	1	2	2	2	2	1	1	1	4	2	2	0	0	0
MA297_LE2448	INIA-WBP	131.42	4195	81	6.75	7	1	2	2	2	2	1	1		3	2	2	1	1	0
MA298_LE2449	INIA-WBP	131.17	3534	78	7	8	1	2	2	2	1	1	1	1	5	2	2	NA	1	0
MA303_LE2454	INIA-WBP	160.15	4783	95	7	8	2	2	2		2	1	1	2	2	2	2	0	0	0
MA304_LE2456	INIA-WBP	137.33	4887	92	7.5	8.5	1	1	2	2	1	1	1	1	6	2	2	0	0	0
MA305_LE2457	INIA-WBP	147.25	2600	48	4	4.5	1	2	2	1	1	1	1	2	5	2	2	0	0	0
MA306_LE2458	INIA-WBP	154.26	1996	42	4.75	5.25	1	1	1	2	2	1	1	1	6	1	2	0	1	0
MA307_LE2459	INIA-WBP	136.31	4218	88	4.5	4.75	1	1	2	2	1	1	1	1	6	2	1	0	1	0
MA308_LE2460	INIA-WBP	125.18	3758	77	5	5.5	1		1	2	2	1	1		4	2	NA	0	1	0
MA309_LE2461	INIA-WBP	128.18	5004	89	7	7	1	2	2	1	1	1	2	2	4	2	2	NA	1	0
MA310_LE2462	INIA-WBP	124.12	3391	78	0.5	1	1	2	2		2	1	1	1	4	1	2	1	1	0
MA311_LE2463	INIA-WBP	156.09	4564	90	6	7.5	2	1	2		1	2	2		2	NA	1	0	1	0
MA312_LE2464	INIA-WBP	159.96	2130	33	0.5	1.5	2	1	1		2	1	1	1	5	2	1	1	1	NA
MA313_LE2465	INIA-WBP	154.27	3555	68	3.25	4.75	1	2	2	2	2	1	1	1	4	1	1	1	1	0
MA314_LE2466	INIA-WBP	137.44	4568	92	4	2	2	2	2	2	2	1	2	1	2	2	2	1	1	0
MA315_LE2467	INIA-WBP	152.05	3114	57	2.25	4.5	1	2	2	1	2	1	1	1	5	1	1	1	1	0
MA316_LE2468	INIA-WBP	124.95	220	1	4.5	4	1	1	1	1	1	1	1	2	7	2	2	1	1	0
MA317_LE2469	INIA-WBP	150.71	3281	64	4.25	4	1	2	2		2	1	1	2	3	1	2	0	1	0
MA318_LE2470	INIA-WBP	145.10	3985	88	4.5	3.5	1	2	2	2	2	1	1	1	4	1	2	0	1	0
MA319_LE2471	INIA-WBP	133.61	3984	86	5.75	6.5	1	2	2	1	2	1	2	2	3	2	2	0	0	0
MA320_LE2473	INIA-WBP	134.47	958	23	5.5	5	1	1	1	1	2	1	1	1	7	2	NA	0	1	0
MA321_LE2474	INIA-WBP	130.94	3148	68	6.5	7	1	1	2		1	2	1	1	5	2	2	0	1	0
MA322_LE2475-GENESIS110	INIA-WBP	133	357	5	1	1	1	1	1	1	1	1	1	1	8	1	2	1	1	0
MA323_LE2476	INIA-WBP	138.61	4858	90	6.5	7		2	2	2	2	2	1	2	1	2	2	0	1	0
MA324_LE2477	INIA-WBP	131.59	2090	49	1.75	2	1	1	1		1	1	1	1	7	2	2	1	1	0
MA325_LE2478	INIA-WBP	132.95	429	0	2.75	2.75	1	1	2	2	2	2	1	2	3	2	2	0	1	0
MA326_ACA320	other breeding programs	149.29	4065	77	6.5	6.5	2	1	2	2	2	2	1	2	2	2	2	0	1	0
MA327_ACA360_CURUPAY	other breeding programs	147.75	3806	75	4.5	4	2	1	2	2	2	2	2	1	2	2	2	0	1	0
MA328_ACA602	other breeding programs	127.79	411	8	4.5	4.5	1	1	2		2	2	1	2	3	2	2	1	1	0
MA329_ACA908_ZEUS	other breeding programs	127.22	5177	94	5.5	6	1	1	2	2	2	2	2	2	2	2	2	0	1	0
MA330_AGPFAST_SYNFAST	other breeding programs	121.95	5491	92	7	7.5	2	1	2	2	2	2	2	2	1	2	2	0	1	0
MA331_DM_ALGARROBO	other breeding programs	139.14	5112	92	4.5	4.5	2	1	1	2	2	2	2	1	3	2	2	0	1	0
MA332_AREX	other breeding programs	120.82	5204	92	6.25	7	2	2	2	2	2	1	1		2	2	2	0	1	0
MA333_ATLAX	other breeding programs	129.10	2137	43	0.5	2	1	1	2	2	2	1	1	1	5	1	NA	NA	1	0
MA334_BAGUETTE17	other breeding programs	133.24	536	5	2.5	3	1	1	2		2	1	1	1	5	2	2	0	1	0
MA335_BAGUETTE18	other breeding programs	136.75	3923	80	3.75	3		1	1	2	2	1	2	1	4	2	2	0	1	0
MA336_BAGUETTE501	other breeding programs	127.09	457	3	4	5.25	1	1	2	1	1	1	1	1	7	1	2	0	NA	NA
MA337_BAGUETTE601	other breeding programs	133.92	4145	88	0.5	2	1	1	2	1	2	1	2	1	5	1	1	0	1	0
MA338_BAGUETTE620	other breeding programs	134.56	2777	68	0.5	1	2	1	1	2	1	2		1	4	2	2	0	1	0
MA339_BAGUETTE701	other breeding programs	153.25	3303	60	2.5	3	1	1	2	2	2	2	2		2	NA	2	0	1	0
MA340_BAGUETTE801	other breeding programs	140.25	3500	78	1	2	1	1	1	2	2	1	2	1	5	1	2	0	1	0
MA341_BAGUETTE11	other breeding programs	139	767	13	6.5	4	1	1	1	1	1	1	1	1	8	2	2	0	1	0
MA342_BAGUETTE13	other breeding programs	132.49	1882	36	0.5	1		1	1	1	2	1	2	1	5	2	2	0	1	0
MA343_BASILIO	other breeding programs	137	289	1	1.5	2	1	1	1	1	1	1	1	1	8	1	2	0	1	0
MA344_BIOINTA1001	other breeding programs	133.05	2637	45	3	8	1	1	1	2	1	1	2	2	5	1	1	0	1	0
MA345_BIOINTA1006	other breeding programs	125.83	3208	64	5.75	5.5	1	1	2	2	2	1	1	2	4	1	2	0	1	0
MA346_BIOINTA3000	other breeding programs	153.35	2611	44	6	5.75	1	1	1	2	1	1	2	1	6	2	2	0	1	0
MA347_BIOINTA3006	other breeding programs	140.50	1009	15	0	3.75	1	2		1	2	1	1	1	5	1	1	0	1	0
MA348_BUCK_GUAPO	other breeding programs	159.67	4240	84	0.5	4.75	2	1	2	2	2	2	2	2	1	2	1	0	1	0
MA349_BUCK_SYN_METEORO	other breeding programs	150.65	4385	80	0.5	6.25	2	2	2	2	2	2	2	2	0	2	1	0	1	0
MA350_BUCK_SYN_PLENO	other breeding programs	123.17	1651	30	0.5	2	1	1	2	1	2	1	1	1	6	2	1	0	1	0
MA351_DM_CEIBO	checks	123.77	4578	89	6	5.5	1	1	1	2	2	2	1	1	5	1	2	0	1	0
MA352_FUNDACEP_BRAVO	other breeding programs	132.18	4663	88	7	7.5	2	2	2		2	2	1		1	2	2	0	1	0
MA353_FUSTE	other breeding programs	139.02	5464	98	7.25	7.5		2	2	2	2	1	1	2	2	2	2	0	1	0
MA354_GINGKO	other breeding programs	125.66	881	13	2.5	2.5	1	1	1	1	2	1	1	1	7	2	2	0	1	0
MA355_GUAYABO	other breeding programs	140	688	9	0	1	1	1	1	1	1	1	1	1	8	1	2	0	1	0
MA356_JACARANDA	other breeding programs	136	460	1	5.5	5.5	1	1	1	1	1	1	1	1	8	1	2	0	1	0
MA357_KLEIN_CAPRICORNIO	other breeding programs	152.51	3642	80	6.75	6.75	1	2	2	2	2	1	2	2	2	2	2	1	1	0
MA358_KLEIN_CASTOR	other breeding programs	124.96	4911	92	7	8	2	2	2		2	2	1	2	1	2	2	0	1	0
MA359_KLEIN_FLAMENCO	other breeding programs	154.65	504	6	3.25	2.5	1	1	2	1	1	1	1	1	7	1	2	0	1	0
MA360_KLEIN_FLECHA	other breeding programs	124.01	449	3	3	2.5	1	1	1	2	1	1	2	1	6	2	1	0	1	0
MA361_KLEIN_GLADIADOR	other breeding programs	155.46	771	5	4.5	4.25	1	1	1	2	1	1	2	1	6	1	1	0	1	0
MA362_KLEIN_LEON	other breeding programs	125.42	4197	79	5.25	6		1	2	2	2	1	2	1	3	2	2	0	1	0
MA363_KLEIN_YARARA	other breeding programs	152.16	4817	91	6.25	7	1	2	2	2	2	1	1	2	3	2	2	0	1	0
MA364_KLEIN_LIEBRE	other breeding programs	134.35	2655	49	1	2	1	1	2	2		1	2	2	3	1	NA	0	1	0
MA365_KLEIN_MARTILLO	other breeding programs	153.78	3971	81	7	6.75	1	1	2	2	1	1	1	2	5	2	2	0	1	0
MA366_KLEIN_MERCURIO	other breeding programs	158.05	5374	104	1.5	7	2	1	2	2	2	2	2		1	2	2	0	1	0
MA367_KLEIN_NUTRIA	other breeding programs	122.77	2594	63	4.75	5.5	1	1	2	2	2	1	1	2	4	1	2	1	1	0
MA368_KLEIN_POTRO	other breeding programs	130.81	2929	60	2.25	2.25	1	1	2	2	2	1	1	2	4	1	2	0	0	0
MA369_KLEIN_PROMETEO	other breeding programs	135.19	4919	89	5.25	3.75	2	1	2	2	2	1	2	2	2	2	2	0	0	0
MA370_KLEIN_PROTEO	other breeding programs	142.43	4922	97	5.75	5.75	1	1	2	2	2	1	2	2	3	2	2	0	0	0
MA371_KLEIN_RAYO	other breeding programs	122.83	4394	82	1	4	2		2	2	2	2	2	1	1	2	NA	0	1	0
MA372_KLEIN_TAURO	other breeding programs	124.98	5396	96	6	6	2	1	2		2	2	1	2	2	2	2	NA	1	0
MA373_LG_LAPACHO	other breeding programs	138.91	3262	75	7	8.25	1	1	1	1	2	1	1	1	7	NA	2	NA	1	0
MA374_LG_ARLASK	other breeding programs	136	1053	9	7.5	8	1	1	1	1	1	1	1	1	8	2	2	0	1	0
MA375_LG_WA110169	other breeding programs	130.64	3494	76	6.5	6.75	1	1	1	2	1	1	1	1	7	2	2	0	1	NA
MA377_MSINTA116	other breeding programs	138.94	3857	71	7	7	2	1	1	1	2	1	1	1	6	2	NA	0	1	0
MA378_MSINTA415	other breeding programs	139.70	1595	32	2	2	1	1	2	2	1	1	1	1	6	2	2	0	1	0
MA379_MSINTA416	other breeding programs	129.90	4463	88	6	7	1	1	1	2	2	1	2	2	4	2	2	0	1	0
MA380_MSINTA815	other breeding programs	120.82	2503	46	7	6.5	2	1	1	1	2	1	1	2	5	2	2	0	1	0
MA381_NOGAL	other breeding programs	136.18	3397	75	7	8	1	1	1	2	1	1	1	1	7	1	2	0	1	0
MA382_NST_BERRETIN	other breeding programs	137.62	3764	84	6.5	6.5	1	2	2		2	1	2	1	3	1	2	0	1	0
MA383_NST_MALEVO	other breeding programs	139.40	3715	79	7	7	1	2	2	2	2	1	2	1	3	1	2	0	1	0
MA384_DM_NANDUBAY	other breeding programs	134.70	4456	92	7	8	1	2	2	2	1	1	1	2	4	2	2	0	1	0
MA385_ONIX	other breeding programs	125.15	5058	93	7	8	1	2	2	2	2		1	1	3	2	2	0	1	0
MA386_DM_PEHUEN	other breeding programs	129.52	1444	33	2.5	3.75	1	1	1	1	2	1		1	6	2	2	0	1	0
MA387_PROINTA_MOLINERO	other breeding programs	138.36	2966	52	7	7.5	1	2	2		1	1	1	2	4	1	2	1	1	0
MA388_DM_SAUCE	other breeding programs	137.90	2848	66	6.25	7	1	1	1	2		1	1	1	6	1	2	0	1	0
MA389_SY110_SYN110	other breeding programs	132.43	2430	35	5	1.5	1	2		2	1	1	1	1	5	2	1	0	1	0
MA390_SYN200	other breeding programs	132	644	6	2.5	2.25	1	1	1	1	1	1	1	1	8	2	2	0	1	0
MA391_SY211_SYN211	other breeding programs	130.68	1573	32	0.5	1	1	1	1	2	1	1	1	1	7	2	2	0	1	0
MA392_SY300_SYN300	other breeding programs	126.16	2303	44	4.5	5.5	1	1	1	1	1	1		1	7	2	2	0	1	0
MA393_SY330_SYN330	other breeding programs	123.88	3444	82	5	7	1	1	2	2	1	1	1	1	6	2	2	0	1	NA
MA394_DM_TBIO_AUDAZ	other breeding programs	119.94	4279	88	4.25	7	2	1	1	2	2	2	1	1	4	1	2	0	1	0
MA395_FRONTANA	other breeding programs	128.45	3043	62	1.5	7	1	1	2	2	1	1	1	2	5	2	1	1	1	0
MA396_TOROPI	other breeding programs	144.00	2122	37	1.25	2.25		2	2		1	1	1	1	4	2	2	0	1	0
MA397_QUP25462009	other breeding programs	130.56	2191	39	1	1	2	1	1	1	1	1	1	1	7	1	2	0	1	0
MA398_QUP24182007	other breeding programs	133.23	444	5	1.75	2	1	1		1	1	1	1	1	7	1	2	0	1	0
MA399_QUP25292009	other breeding programs	123.18	340	1	3.25	2	1	1	2	2	1	1	1	1	6	2	2	1	0	0
MA400_PANTERA	other breeding programs	126.39	271	1	1.75	1.5	1	2	2	2	2	1	1	1	4	2	2	0	1	0

Table S2. Fit indicators of the area under the disease progress curve (AUDPC) data analysis models for the 366 wheat lines from 2021, 2022, and both years combined.

Year	Model	AIC[a]	BIC[b]	Heritability	Correlation (obs. vs. pre.)[c]
2021	Base	12657	14496	0.9834	0.9841
	Column	12659	14503	0.9834	0.9841
	Base + Cor. Gaussian	12659	14503	0.9834	0.9841
	Base + Cor. Spherical	12659	14503	0.9834	0.9841
	Base + Cor. Exponential	12659	14503	0.9834	0.9841
2022	Base	13134	14569	0.9813	0.9819
	Column	13126	14565	0.9826	0.9839
	Base + Cor. Gaussian	13136	14576	0.9813	0.9819
	Base + Cor. Spherical	13136	14576	0.9813	0.9819
	Base + Cor. Exponential	13136	14576	0.9813	0.9819
	Column + Cor. Gaussian	12734	13128	0.9826	0.9839
	Column + Cor. Spherical	12734	13128	0.9826	0.9839
	Column + Cor. Exponential	12734	13128	0.9826	0.9839
Both years	Base	33373	35583	0.9658	0.9125
	Column	33374	35589	0.9658	0.9105

[a]AIC: Akaike information criterion; [b]BIC: Bayesian information criterion; [c]correlation between observed phenotypic values for each line and the value predicted by the model

Table S3. Comparison of the physical position of the quantitative trait loci (QTL) identified as regions associated with resistance to yellow rust and genes and QTL, based on Tong e t al. (2024) previous review.

Reported QTL or gene[a]	Chromosome	Position physical (Mb)[b]	PVE[c]	Reference
QYrpd.swust-1BL	1B	527.37	12.6	Zhou et al. 2022b
/	1B	532.62	5.1	Roseswarne et al. 2012
QYr.uy-1BL	1B	540.93	4.9	
QYr.spa-1B	1B	578.97	15.2	Bokore et al. 2017
QYrsv.swust-1BL.1	1B	646.95	3.3	Zhou et al. 2021b
QLr.caas-1BL	1B	653.33	28.5	Ren et al. 2012c
Lr46/Yr29/Sr58	1B	661.86	46.8	Li et al. 2020
QYr.caas-2BS.1	2B	417.62	29.9	Bin et al. 2012
QYr.ifa-2BL	2B	423.75	16.8	Buerstmayr et al. 2014
QYr.uy-2BL.1	2B	432.33	5.7	
/	2B	470.78	26.20	Yuan et al. 2018
QYr.inra-2BL	2B	519.48	24.3	Paillard et al. 2012
QYr.nafu-2BL	2B	556.62	17.7	Zhou et al. 2015a
QYr.uy-2BL.2	2B	564.65	12.1	
QYrqn.nwafu-2BL	2B	579.36	39.1	Zeng et al. 2019c
QYr.inra-2BL	2B	594.03	70	Mallard et al. 2005
Yr53	2B	605.84		Xu et al. 2013
QYr.inra-3Bcent	2B	609.6	15.3	Dedryver et al. 2009
QYr.inra-2BS	2B	629.2	61	Mallard et al. 2005
QYrdr.wgp-2BL	2B	636.84	13.7	Hou et al. 2015
Yr41	2B	660.73		Luo et al. 2005
QYrSM155.1	2B	661.11	47.9	Zhou et al. 2022a
QYrsnb.nwafu-2BL	2B	691.08	17.3	Zeng et al. 2019b
Yr7[d]	2B	693.31		Marchal et al. 2018
QYr.uy-2BL.3	2B	700.09	21.2	
Qyr.gaas.2B.1	2B	710.42	1.9	Cheng e al. 2022
QYraq.cau-2BL	2B	713.16	61.5	Guo et al. 2008
QYrns.orz-2BL	2B	714.81	30	Vazquez et al. 2015
QYr.caas-2BL	2B	721.88	12.2	Ren et al. 2012a
Yrq1	2D	16.99	48.1	Cao et al. 2012
QYr.uy.2DS	2D	16.82	19.1	
QYr.hbau-2DS	2D	20.57	7.50	Gebrewahid et al. 2020
QYr.nmbu.3A.2	3A	481.5		Lin et al. 2023
QYr.uy.3AL	3A	489.3	7.9	
QYr.hbaas-3AL	3A	502.8		Jia et al. 2020
Yr47	5B	8.25		Bansal et al. 2011
QYrPI181410.wgp-5BL.1	5B	23.21	15.10	Liu et al. 2020b
QYr.ufs-5B	5B	26.63	5.70	Agenbag et al. 2012
QYr.uy-5BS	5B	72.33	10.6	
QYrPI181410.wgp-5BL.2	5B	178.16	17.30	Liu et al. 2020b
QYr.caas-5BL.1	5B	510.98	7.20	Lu et al. 2009
QYr.YBZR-5BL	5B	529.16	10.90	Deng et al. 2022
QYr.uy-5BL	5B	538.22	8.6	
QYr.AYH-5BL	5B	539.15	21.40	Long et al. 2021
QYrdr.wgp-5BL.2	5B	545.54	13.29	Hou et al. 2015
QYr.dms-5B	5B	549.92	6.7	Zou et al. 2017
Yr70	5D	8.86		Bansal et al. 2017
QYr.uy-5DL	5D	550.06	7.3	
QYrdr.wgp-5DL	5D	554.21	11.94	Hou et al. 2015
QYrbr.wpg-5D	5D	555.41	12.00	Case et al. 2014
QYr.hebau-7BL	7B	624.58	5.90	Zhang et al. 2019c
QYr.uy-7BL	7B	637.44	6.6	
QYr.niab-7B	7B	648.00	13.00	Powell et al. 2013
QYr.cim-7BL	7B	657.16	12.00	Calvo-Salazar et al. 2015
Yr67	7B	677.49		Bariana et al. 2022

[a]The designated or cloned resistance genes were highlighted as blue, QTL reported in this work were highlighted in yellow. The reported names of QTL were collected from corresponding references. "/" indicates that the name was not provided by the literature; [b]Physical positions (Mb) of flanking markers are based on the Chinese Spring reference IWGSC RefSeq v1.0; [c]PVE, phenotypic variance explained; [d]*Yr5* and *YrSP* are located in te same gene cluster as *Yr7* (Marchal et al., 2018)

4. Discusión general

4.1. Estudio de la diversidad del patógeno

El análisis preliminar de 124 aislamientos del patógeno *Pst* recolectados en Uruguay entre 2017 y 2021 permitió identificar once fenotipos de virulencia distintos, diferenciados con base en diez genes de resistencia. A partir de esta caracterización, se seleccionaron veintisisete aislamientos representativos por su diversidad en fenotipo de avirulencia/virulencia, origen geográfico, año de recolección y cultivar hospedero para un análisis genético y fenotípico en profundidad.

El análisis genético mediante diecinueve marcadores SSR reveló la presencia de tres grupos genéticos previamente descritos en otras regiones del mundo: *PstS13* (78 % de los aislamientos), *PstS7* (15 %) y *PstS10* (8 %). El grupo *PstS13* se encontró en todos los años de muestreo, mientras que *PstS7* apareció por primera vez en 2018 y nuevamente en 2020 y 2021. *PstS10* fue detectado únicamente en 2020. Cada grupo presentó fenotipos de virulencia específicos. Dentro de *PstS7* se identificaron aislamientos con virulencia similar a la raza warrior, mientras que los dos aislamientos de *PstS10* coincidieron con la raza benchmark. En el grupo *PstS13* se detectaron cuatro fenotipos, incluyendo la raza triticale2015, ya reportada en Europa y Argentina, y tres nuevas variantes identificadas entre 2019 y 2021, que mostraron un incremento progresivo en la virulencia sobre *Yr3*, *Yr17*, *Yr25*, *Yr27* e *Yr32* (Riella et al., 2024). Con base en el fenotipado realizado localmente de otras muestras de 2021 y muestras de 2022, se determinó la presencia de los mismos fenotipos identificados previamente. Prevaleció el fenotipo correspondiente al grupo genético *PstS13*, en particular la variante virulenta sobre *Yr17* e *Yr32*, seguido por la variante virulenta sobre *Yr17*, *Yr27* e *Yr32*.

Estos resultados proporcionan evidencia del establecimiento en Uruguay de razas, así como del surgimiento de nuevas variantes locales con mayor amplitud de virulencia. Este patrón sugiere la ocurrencia de evolución regional del patógeno.

La aparición de estos grupos genéticos en el Cono Sur, previamente identificados en Europa, podría explicarse tanto por actividades humanas (Ali et al., 2014; Brown y Hovmøller, 2002; Stubbs, 1985; Wellings, 2007; Zadoks, 1961) como por dispersión

aérea a larga distancia (Hovmøller et al., 2002; Zadoks, 1961). La continuidad geográfica entre Argentina y Uruguay, ambos pertenecientes a la misma zona epidemiológica de royas (Rajaram y Campos, 1974), facilita la dispersión de urediniosporas, lo cual puede explicar la aparición simultánea de epidemias severas en ambos países a partir de 2017 (Campos et al., 2016; Carmona et al., 2019; Germán et al., 2018). Antes de ese año, *Pst* no era un patógeno predominante en esta región, en parte porque no sobrevivía durante el verano en las áreas agrícolas (Germán et al., 2007). Sin embargo, desde 2017 se ha observado un cambio en la dinámica epidemiológica, atribuido a una mayor capacidad del patógeno para sobrevivir fuera del ciclo del cultivo, lo que facilita infecciones tempranas y epidemias severas.

PstS7 y *PstS10*, aunque menos frecuentes en este estudio, han sido responsables de importantes epidemias desde 2011 (Ali et al., 2017; Sørensen et al., 2014). El grupo genético predominante en Uruguay, *PstS13*, se detectó por primera vez en Europa en 2015 (Hovmøller et al., 2018) y se ha diseminado rápidamente a otros continentes. En Sudamérica, se identificó en Argentina y Chile en 2017 y 2018 (Carmona et al., 2019; Hovmøller et al., 2018, 2020) y más recientemente en Paraguay (Fernández-Gamarra et al., 2023). En Uruguay se identificaron nuevas variantes que demuestran evolución local del patógeno. Las tres nuevas variantes encontradas en Uruguay entre 2019 y 2021 adquirieron virulencia sobre *Yr3*, *Yr17*, *Yr25, Yr27 e Yr32*, siguiendo un patrón compatible con mutaciones secuenciales de ganancia de virulencia. *Triticale2015b* virulenta sobre *Yr17* e *Yr32*, *Triticale2015c* sobre *Yr27*, *Yr27* e *Yr32*, y *Triticale2015d* virulenta sobre *Yr3*, *Yr17*, *Yr25* e *Yr32*.

Esta evolución gradual sugiere una presión de selección ejercida por los genes de resistencia desplegados en los cultivares locales. Sin embargo, el surgimiento de estas nuevas variantes no se tradujo necesariamente en un aumento dramático de la susceptibilidad en campo. Algunas variedades mostraron un cambio de nivel de resistencia de bajo a intermedio (Génesis 6.87, Génesis 4.33, SYN 211) o de intermedio a alto (Algarrobo), probablemente como consecuencia de la aparición de estas nuevas variantes.

Aunque este estudio permitió detectar tres grupos genéticos ampliamente distribuidos, no puede descartarse la presencia de otros grupos no detectados, como

PstS14, reportado en Argentina en 2017 (Carmona et al., 2019), pero ausente en esta colección. Esto sugiere la necesidad de continuar con monitoreos más amplios y sistemáticos a escala regional para mejorar el conocimiento de la distribución y evolución de *Pst* en el Cono Sur.

4.2. Identificación de regiones genómicas asociadas a la resistencia a roya estriada

El panel utilizado para el análisis de GWAS consistió en 366 genotipos diversos de trigo de diferentes orígenes, comprendiendo 172 líneas del Programa de Desarrollo de Germoplasma de Trigo Resistente a enfermedades de INIA (INIA-RGDP), desarrolladas para introgresar RP a roya de la hoja, principalmente a partir de germoplasma del CIMMYT; 117 líneas del Programa de Mejoramiento de Trigo de INIA (INIA-PMGT), que incluyen líneas avanzadas y élite, así como variedades liberadas, que representan un siglo de mejoramiento genético en el país; 73 cultivares de otros programas de mejoramiento utilizadas en Uruguay; y cuatro líneas testigos seleccionadas por su diversidad en fecha de espigazón y susceptibilidad a RE.

El fenotipado en planta adulta para RE se llevó a cabo en experimentos de campo en INIA La Estanzuela durante 2021 y 2022. Los ensayos se establecieron bajo un diseño alfa lattice con tres repeticiones, se utilizaron parcelas de dos surcos de 1 m separadas por 0,30 m. Para asegurar una presión uniforme de inóculo, se sembraron bordes de infección con una mezcla de cultivares susceptibles, inoculados con urediniosporas de las razas triticale2015*a* y *b* (grupo *PstS13*; Riella et al., 2024). En 2021 el experimento fue en secano, mientras que en 2022 se implementó riego suplementario por aspersión.

Se registraron días a espigazón y la severidad de la enfermedad en seis evaluaciones por año, se calculó el área bajo la curva de progreso de la enfermedad (AUDPC), que se utilizó como la variable para los análisis estadísticos. Los datos fenotípicos se analizaron en R mediante modelos lineales mixtos ajustados por año y se incorporó los días a espigazón como covariable. Dado el alto grado de correlación entre años, se ajustó un modelo combinado para estimar medias ajustadas (BLUE) de AUDPC por genotipo.

En 2023 se realizaron ensayos en invernáculo para caracterizar la resistencia en plántula (ASR). Se inocularon plántulas con las mismas razas utilizadas a campo y se evaluó el tipo de infección (TI) a los 15-20 días, según la escala 0-9 de McNeal et al. (1971). Se utilizaron dos repeticiones por genotipo y raza y se incluyeron líneas diferenciales para verificar identidad y pureza de las razas. Las medias ajustadas se obtuvieron mediante modelos lineales simples.

El genotipado de los materiales del panel se realizó mediante GBS en la Universidad de Wisconsin (Madison). Se generaron lecturas *paired-end* de 150 pb y se procesaron en Tassel 5.2 para alineación al genoma de referencia (Glaubitz et al., 2014), llamado de SNP y filtrado. Se eliminaron SNP con más de 80 % de datos faltantes o frecuencia del alelo menor (MAF) < 1 %. Los datos faltantes se imputaron con Beagle 5.4. Se calcularon distancias genéticas con el coeficiente de Jaccard y se realizó un PCA para evaluar estructura poblacional.

El análisis GWAS se realizó con el paquete GWASpoly, se utilizaron 156.032 SNP y BLUE de AUDPC como variable respuesta. Se ajustó un modelo mixto con matriz de parentesco (modelo K) para controlar estructura poblacional. Se utilizaron gráficos Manhattan y cuantil-cuantil (QQ) para visualizar resultados y FDR = 0,1 como umbral de significancia. Se definieron QTL como regiones con al menos dos SNP en LD significativo. Los QTL fueron nombrados siguiendo la nomenclatura de Boden et al. (2023) complementado con el brazo del cromosoma donde se ubicaron y se compararon con genes *Yr* y QTL previamente reportados (McIntosh, 2024; Tong et al., 2024).

Finalmente, se evaluó el efecto acumulativo de alelos favorables en AUDPC se agruparon líneas según el número de alelos y se compararon las medias mediante la prueba de Tukey ($P < 0,05$).

Se observaron altos niveles de infección en los cultivares testigo y una amplia variabilidad en los valores de AUDPC (0-5491). Los modelos estadísticos ajustados mostraron alta precisión y los BLUE de AUDPC fueron fuertemente correlacionados entre años ($r = 0,74$), lo que permitió combinar los datos en un único análisis. La heredabilidad amplia para AUDPC fue muy alta en ambos años (0,98), con fuertes correlaciones entre repeticiones y entre años ($r = 0,74$), lo cual respalda la solidez del

conjunto de datos para estudios de asociación genómica (GWAS). El análisis de estructura poblacional reveló una estratificación genética débil, lo que permitió controlar asociaciones espurias al usar un modelo K.

El análisis genotípico retuvo 156.032 SNP distribuidos homogéneamente en el genoma, aunque con menor cobertura en el subgenoma D. No se detectó estructura poblacional y el desequilibrio de ligamiento decayó rápidamente (<0,2 en 0,12 Mb).

El GWAS identificó ocho regiones genómicas asociadas a resistencia a RE en campo, consistentes entre años, que no se deben a diferencias en ciclo a espigazón, dado que el QTL para días a espigazón encontrado (cromosoma 2D) no colocalizó con QTL de resistencia. El GWAS identificó QTL asociados a resistencia RE a campo en los cromosomas 1BL, 2BL (tres QTL), 5BS, 5BL, 5DL y 7BL. *QYr.uy-2BL.3* explicó la mayor proporción de varianza fenotípica (21,2 %) con una reducción del 61 % del AUDPC en líneas con el alelo favorable, seguido por *Qyr.uy-2BL.2* (12,1 %). *Qyr.uy-5BS*, *Qyr.uy-5BL*, *Qyr.uyt-5DL*, *Qyr.uyt-7DL, Qyr.uyt-2BL.1* y *Qyr.uy-1BL* explicaron progresivamente menor proporción de la variancia fenotípica. Entre los QTL encontrados, solo *QYr.uy-2BL.3* colocalizó con genes previamente reportados (cluster *Yr5, Yr7, YrSp*), pero se demostró que no es ninguno de estos genes. *QYr.uy-5BS* parece ser novedoso, ya que la región más cercana asociada a resistencia a RE previamente reportada está ubicada a 40 Mb (Tong et al., 2024). *QYr.uy-5BL* podría ser *QYrdr.wgp-5BL.2* (Hou et al., 2015) ubicado a menos de 1 Mb. Los otros QTL identificados están localizados cercanos (10 Mb o menos) de QTL previamente reportados (Tong et al., 2024), por lo que se requieren estudios adicionales para determinar si estos QTL corresponden a loci conocidos o son novedosos.

Se identificaron dos QTL asociados a resistencia frente a triticale2015a (2DS y 3AL) en el estado de plántula que no fueron detectados para la raza triticale2015b con mayor rango de virulencia. Los QTL detectados para resistencia en plántula para la raza triticale2015a no coincidieron con los detectados a campo, lo que indica que los QTL detectados a campo confieren RP. El apilamiento de QTL redujo significativamente el AUDPC, lo que confirma efectos aditivos que han sido reportados previamente para RP.

La presencia de alelos favorables fue variable entre germoplasma de distinto origen. El germoplasma de INIA-RGDP, seleccionado por resistencia APR a roya de la hoja, mostró menores valores de AUDPC para RE, lo que sugiere pleiotropía entre genes de APR para ambas royas. Once líneas presentaron los alelos favorables de todos los QTL encontrados y tuvieron muy baja infección de RE a campo. Estos recursos genéticos podrían ser utilizados como fuentes de resistencia a RE y representan una oportunidad para desarrollar cultivares con resistencia durable y de amplio espectro.

4.3. Evaluación de la precisión en los modelos de predicción genómica

Los programas modernos de mejoramiento, especialmente en un contexto de costos de genotipado cada vez más accesibles, requieren optimizar estrategias tanto para la selección de líneas como para la identificación eficiente de progenitores para cruzamientos en etapas tempranas. Esto es clave para desarrollar cultivares adaptados y resistentes en el menor tiempo posible. Este estudio tuvo como objetivo determinar la capacidad predictiva de diferentes modelos de PG al utilizar información genómica y fenotípica de las líneas del panel. Además, se evaluó si la incorporación de QTL identificados por GWAS (asociados al AUDPC para RE) como efectos fijos podría mejorar dicha capacidad predictiva.

Se evaluaron siete modelos de PG que asumen diferentes distribuciones de los efectos de los marcadores: A-BLUP, RR-BLUP (usa toda la información de marcadores), G-BLUP (considera distancias genéticas entre líneas) y cuatro modelos bayesianos (BA, BB, BC y BL). Los resultados mostraron que no hubo diferencias significativas entre RR-BLUP y G-BLUP, ambos con correlaciones de aproximadamente 0,7 entre los valores observados y predichos. Los modelos bayesianos BA, BB y BC tampoco mostraron diferencias notables, mientras que BL tuvo un desempeño inferior (correlación $\approx$ 0,5), similar al modelo A-BLUP. Resultados similares fueron reportados por Tehseen et al. (2021) y Manickavelu et al. (2016). La similitud entre G-BLUP y los modelos bayesianos (que requieren mayor tiempo computacional) indica que suposiciones simples sobre varianzas iguales de efectos de marcadores pueden ser suficientes para predecir AUDPC de RE. Este

hallazgo es consistente con estudios previos en roya del tallo y fusariosis de la espiga en trigo (Ornella et al., 2012; Rutkoski et al., 2014).

La predicción genómica resultó eficaz para predecir AUDPC en el panel de GP, con correlaciones de alrededor de 0,7 para los modelos equivalentes RR-BLUP y G-BLUP. La inclusión de QTL identificados por GWAS como efectos fijos mejoró la precisión predictiva. En particular, incluir el QTL con mayor efecto fue suficiente para lograr esta mejora, sin beneficios adicionales al añadir otros QTL. La inclusión del QTL *QYr.uy-2BL.3* redujo el error cuadrático medio (MSE) en un 10,4 % en comparación con G-BLUP sin dicho QTL, probablemente debido a su fuerte efecto sobre la variable de respuesta. Estudios de simulación demostraron que modelar loci de gran efecto como efectos fijos es ventajoso cuando la heredabilidad del rasgo es superior a 0,5 y el locus explica más del 25 % de la varianza genética (Bernardo, 2014). Además, datos reales muestran que G-BLUP con efectos fijos supera a G-BLUP estándar para rasgos donde estos loci explican una proporción sustancial de la variación (Juliana et al., 2017; Rutkoski et al., 2014). Sin embargo, para muestras nuevas, G-BLUP sin efectos fijos puede ofrecer resultados comparables a aquellos obtenidos con marcadores de efectos fijos.

5. Conclusiones

La presencia en nuestro país y otros países del Cono Sur de América de razas de *Pst* originarias de otros continentes confirma la hipótesis de migración intercontinental del patógeno. Estas razas agresivas de *Pst* están asociadas a las actuales epidemias observadas en la región. La detección de estas razas exclusivamente en relevamientos realizados desde 2017 indica que se establecieron en la región, permiten sobrevivencia en el verano en zonas donde se realizan cultivos o cercanas a estos, lo que genera infecciones tempranas y el desarrollo de epidemias anualmente.

La identificación en Uruguay de razas de *Pst* no descritas previamente es evidencia de evolución reciente del patógeno en Uruguay, adquiriendo virulencia sobre algunos de los genes de resistencia previamente efectivos. La aparición de estas nuevas razas de *Pst* tiene implicancias importantes para el manejo de la enfermedad y para los programas de mejoramiento genético enfocados en resistencia en Uruguay y países vecinos. A corto plazo, el manejo local de epidemias en variedades susceptibles de trigo se limita a la aplicación de fungicidas. Sin embargo, la utilización de cultivares resistentes aparece como la estrategia más amigable con el medioambiente y sin costos adicionales para los productores. El desarrollo de cultivares resistentes implica un monitoreo permanente de las razas de *Pst* para generar materiales con resistencia efectiva frente a las razas presentes en una zona epidemiológica específica, principalmente si se utilizan genes de resistencia específicos a razas en particular. La ocurrencia de dispersión a larga distancia de razas desde continentes distantes resalta la importancia de la coordinación global en los esfuerzos de monitoreo. Las nuevas variantes raciales reportadas en este trabajo podrían migrar hacia otras áreas y potencialmente causar pérdidas de rendimiento importantes en regiones donde se cultivan variedades que poseen genes de resistencia que han sido superados por dichas razas.

Los resultados de este estudio sobre identificación de regiones genómicas asociadas a la resistencia a RE sientan las bases para comprender el fundamento genético de la resistencia a la RE presente en un panel diverso de trigo y pueden aplicarse al desarrollo de nuevos cultivares localmente adaptados con mejor resistencia

a la enfermedad. Reportamos ocho regiones genómicas asociadas con resistencia a campo; ninguna de estas regiones fue identificada en etapa de plántula. Los dos QTL identificados en plántula frente a la raza triticale2015*a* no fueron detectados frente a la raza triticale2015*b* ni tampoco a campo. Por lo tanto, todos los loci detectados a campo confieren RP. Ninguno de los QTL correspondió a genes *Yr* conocidos. *QYr.uy-5BS* es probablemente un nuevo locus de resistencia a RE. La posición de los otros siete QTL fue cercana a QTL previamente reportados; para los cuales se requieren más estudios para determinar si representan QTL conocidos o novedosos. Es relevante identificar si los QTL detectados son novedosos, dado que su utilización en el desarrollo de cultivares resistentes permite incrementar la diversidad genética en la base de resistencia, en el ámbito local y global, lo que representa un seguro frente a la capacidad de variación que posee *Pst*.

La disminución significativa del nivel de infección de RE al incrementar el número de QTL presentes en los genotipos indica que poseen efecto aditivo. Esta característica sumada a la expresión de la resistencia en planta adulta sugiere que el tipo de resistencia conferida por los loci encontrados es de tipo cuantitativa. En el caso de las royas de trigo, este tipo de resistencia se considera que no es específica para cada raza y, por lo tanto, durable. La mayoría de los genes conocidos de resistencia a royas con esta característica poseen además efecto pleiotrópico, ya que confieren resistencia a varias enfermedades, lo que posee gran implicancia para el mejoramiento genético por resistencia. Una vez validados, los QTL identificados podrán ser utilizados para desarrollar y seleccionar variedades con altos niveles de resistencia a RE, como es el caso de las líneas que combinan todos los QTL encontrados. Estos QTL deben además ser combinados con otros genes o QTL diferentes para ampliar la variabilidad genética frente a la enfermedad.

De manera similar, la PG fue altamente efectiva (con una capacidad de predicción cercana a 0,7) para predecir los niveles de enfermedad, lo que posiciona a la PG como una herramienta valiosa para seleccionar padres en programas de mejoramiento, así como para seleccionar líneas.

La metodología utilizada para analizar tanto los datos fenotípicos de campo como los datos genotípicos permitió identificar regiones genómicas asociadas a la

resistencia a RE, los cuales pueden ser aplicables a proyectos sobre otras enfermedades del trigo y en otras especies cultivadas. Además, esta metodología demostró ser altamente robusta y capaz de generar datos de alta calidad, los cuales constituyen la base de cualquier estrategia de mejoramiento sólida. Estos hallazgos brindan información valiosa sobre la base genética de la resistencia a la RE y ofrecen herramientas robustas para fortalecer los esfuerzos de mejoramiento en trigo.

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