Status of Ug99 resistance in current Australian wheat cultivars and breeding materials

Park RF, Bariana HS
The University of Sydney, Faculty of Agriculture, Food and Natural Resources, Plant Breeding Institute, PMB 11, Camden, NSW 2570, AUSTRALIA.

INTRODUCTION

Stem rust of wheat (caused by Puccinia graminis f. sp. tritici; Pgt) occurs in all wheat growing regions of Australia, and has at times caused serious yield losses. Reports of economic losses in Australian wheat production due to stem rust include £2–3 million in 1889 (McAlpine, 1906), £400,000 in 1903, £2 million in 1916 (Waterhouse, 1929), £7 million in 1947 (Butler, 1948), and AUD$200 to 300 million in 1973 (Watson and Butler, 1984). The losses from the epidemic in 1973, centered on south eastern Australia, were considered by Watson and Butler (1984) to be the most severe in the history of the Australian wheat industry. A workshop convened in September 1974 by the NSW Department of Agriculture in response to the 1973 epidemic addressed the concept of a nationally coordinated approach to wheat breeding for rust resistance, and led to the establishment of the National Rust Control Program (currently the Australian Cereal Rust Control Program; ACRCP).

CURRENT EXOTIC RUST THREATS TO AUSTRALIA

There are four major current exotic rust threats to the Australian cereal industries: stripe rust of barley caused by Puccinia striiformis f. sp. hordei, leaf rust of durum wheat caused by Puccinia sp. Group II Type A, crown rust of barley caused by P. coronata var. hordei, and Pgt isolates within the “Ug99” lineage. “Ug99” was first detected in Uganda in 1999, and subsequently in Kenya, Ethiopia, Yemen and Iran. Since its first detection, several other races have been found that are believed to be closely related to “Ug99”, together forming the “Ug99” lineage:

1. race TTKSK (“Ug99”; found in Uganda, Kenya, Ethiopia, Yemen and Iran)
2. race TTKST (“Ug99” +Sr24)
3. race TTKSK (“Ug99” +Sr36)
4. race TTKSF (identical to “Ug99” but lacking virulence for Sr31; found only in South Africa)
5. race TTKSP (identical to race TTKSF but with added virulence for Sr24; found only in South Africa)

STEM RUST RESISTANCE BREEDING IN AUSTRALIA

Brennan and Murray (1988) estimated that efforts to breed wheat for resistance to Pgt saved the Australian grains industry about $128 million per year. Comprehensive reviews of efforts to breed for resistance to Pgt in Australia were published by Macindoe and Walkden Brown (1968) and Luig (1983), and Luig and Watson (1970) reviewed not only resistance breeding per se but also its impact on virulence in Australian populations of Pgt.

The second of the three phases identified by Luig and Watson (1970), from 1938 to 1964, was characterised by the releases of cultivars carrying single genes for resistance (Sr6, Sr11, Sr17, Sr9b, Sr36) and the subsequent detection of mutant pathotypes with corresponding virulence. The third phase (1965 to 1970), in which the genetic base of resistance was broadened and cultivars with multiple genes for resistance to Pgt were deployed, began with the release of Mendos (Sr11, Sr17, Sr36) in 1964 (Luig and Watson 1970). Over the past 40 years, the most effective sources of resistance to Pgt have been based on the genes Sr24, Sr26, Sr30, Sr36 and Sr38, and genes imparting partial protection such as Sr2, Sr12 and Sr13 have been important components of many gene combinations. Of these, the most important have been and continue to be Sr2, Sr24 and Sr26, for which virulence has not been detected in Australia. At least 38 cultivars have been released carrying Sr2, 35 carrying Sr24, and 31 carrying Sr26, including cultivars carrying two of these genes (Sr24+Sr26, Sr2+Sr24). These genes have also been deployed in combinations with genes for which virulence is rare or no longer detected (e.g. Sr24 or Sr26 with Sr30, Sr36, or Sr38). Despite the detection of virulence for Sr30, Sr36 and Sr38, these genes have also remained important in combinations for which matching virulence either does not occur or is no longer detected (e.g Sr9g+Sr30, Sr9e+Sr36, Sr36+Sr38).

RESPONSE OF AUSTRALIAN WHEAT GERMPLASM TO “Ug99”

Following the disastrous stem rust epidemic of 1973/74, a stronger national focus was placed on stem rust resistance by rust testing of germplasm, research on genetics of resistance including reducing linkage drag associated with alien Sr genes, and the development and
application of markers linked to important Sr genes. This strategy, coordinated by (currently) the ACRCP and largely funded by (currently) the Grains Research and Development Corporation, has led to a robust understanding of deployed Sr genes and a resulting ability to predict response of Australian germplasm to “Ug99”. These predictions have been refined by field testing germplasm in Kenya with the assistance of the Kenyan Agricultural Research Institute from 2005-07. Because Sr31 has not been used widely in Australia, the greatest impact of “Ug99” on germplasm to date has been due to virulence for Sr30, combined virulence for Sr38 with other genes, and more recently, virulence for Sr24 and Sr36. While virulences for Sr30, Sr36 and Sr38 have been detected in Australia, virulence for Sr24 has not. The genes Sr2, Sr12, Sr13, Sr22 and Sr26, effective against “Ug99” and derivatives, are important contributors to the resistance present in current germplasm.

Efforts to ensure genetic diversity in the resistances deployed in Australia and to avoid over-reliance on genes such as Sr24 and Sr26 are important considerations for the future. Already, wheat cultivars carrying the resistance genes Sr22 (Schomburgk), Sr33 (Lorikeet) and Sr45 (Thornbill) have been released and many backcross derivatives carrying these genes and others like Sr39 have been produced. The development of linked markers for Sr2, Sr26 and SrR (Ellis et al. 2007), all effective against “Ug99”, are also important advances that will allow more efficient gene pyramiding. Identifying new sources of resistance and dissociating negative traits associated with alien-derived resistances are also crucial (Dundas et al. 2007). Above all, continued industry commitment to Minimum Disease Standards (Wallwork 2007) for all released cultivars is essential to ensure Australian wheat growers have sustained genetic protection from this potentially devastating disease.

ACKNOWLEDGEMENTS

The authors would like to thank the Australian Grains Research and Development Corporation and predecessors for long-term financial support of cereal rust research at the University of Sydney.

REFERENCES


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