Australia’s Preparedness for Ug99

A review of the Australian grains industry’s ability to respond to the arrival of stem rust of wheat (Puccinia graminis f. sp. tritici) pathotype Ug99

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and Plant Health Australia

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Further information

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1 Key findings

- Stem rust (*Puccinia graminis* f. sp. *tritici*) is the most damaging of the three rust diseases that affect wheat.
- Australia currently has many pathotypes of wheat stem rust; however prevalence of the disease has declined due to the widespread use of resistant wheat varieties.
- A new pathotype of stem rust, Ug99, is of concern as it has overcome 17 resistance genes, including *Sr31*, which is one of the world’s most widely used and previously effective source of resistance.
- Should Ug99 be detected in Australia, it is unlikely eradication or containment will be technically feasible, as spores are readily spread by wind and the time required to survey and definitively differentiate this pathotype from existing stem rust pathotypes is likely to be too long to consider either option.
- Currently, 28% of wheat cultivars in Australia are rated as moderately susceptible to susceptible to existing stem rust strains. If Ug99 becomes established, up to 60% of cultivars will become moderately susceptible to susceptible.
- Stem rust can mutate readily and since the initial detection of Ug99, two new derivatives have been identified. Further mutations could continue to occur, potentially affecting other resistance genes in use in Australia.
- As at December 2008, Ug99 had been confirmed in Africa (Ethiopia, Sudan, Kenya and Uganda) and the Middle East (Iran and Yemen). Surveillance is being undertaken throughout the world to track the distribution of Ug99 through the Global Cereal Rust Management System (GCRMS).
- In Australia, surveillance for cereal rusts is linked to the GCRMS through the Australian Cereal Rust Control Program (ACRCP) and, providing funding for this program is maintained at the current levels, ACRCP will detect any new pathotypes of stem rust that establish in Australia.
- Preparedness activities for Ug99 being undertaken in Australia include significant work in surveillance and pre-breeding for germplasm resistance.
- Should Ug99 become established in Australia, management could potentially include the use of chemical (fungicide) application. It is unlikely sufficient chemical stockpiles will be available in the event of an epidemic.
- Should Ug99 become established, extensive communication within the grains industry will be required to encourage the use of resistant cultivars. Resources will be required to ensure that sufficient quantities of seed of resistant cultivars are available for sowing in the years following initial detection of a new rust pathotype.
- Ug99 is not the only cereal rust threat in Australia. In particular, Stripe rust of barley and Leaf rust in durum wheat have the potential to cause very significant damage.
2 Purpose of this document

This business continuity plan was developed to provide an overview of the Australian grains industry’s preparedness for an incursion of *Puccinia graminis* f. sp. *tritici* pathotype Ug99 and/or its derivatives. This document contains background information on the pest biology, diagnostic and surveillance activities in place to respond to an incursion, as well as possible control measures and management strategies. The information contained within this document is designed to minimise the disruption to agricultural industries following the entry and establishment of the pest.

3 Eradication or containment decision matrix

Eradication of *P. graminis* f. sp. *tritici* pathotype Ug99 would only be technically feasible if the rust is detected while still contained within a very small area and the spore load is light. Determination of the extent of the incursion should be completed quickly and commence as soon as there is a reasonable suspicion of the presence of Ug99, without waiting for confirmation, as any delay may be critical in allowing further spread. While it is possible an initial detection maybe contained within an area small enough and/or isolated enough that eradication is considered feasible.

Past experience in the detection and monitoring of exotic cereal rust pathogen isolates have shown that eradication is not likely to work. With current surveillance protocols, the threshold of detection of new rust isolates is such that by the time a new pathogen has been detected, it has already spread over significant distances. Stripe rust of wheat was first detected in Australia in 1979, and this example is instructive in this context. The initial detection of this pathogen and its subsequent spread and establishment were well documented by O’Brien et al. (1980) and Wellings (2007). The disease was first reported on 25 October 1979 near Charlton and Dooen (Victoria), and had been observed 3 days earlier at Darlington Point (NSW). Detailed surveys established that by November 16, the disease was already well established throughout in commercial wheat crops throughout the Mallee and Wimmera, as well as south of Hamilton and near Geelong (O’Brien et al. 1980). At the time, it was suggested that the pathogen would not be able to survive the non-cropping harsh Australian summer; however, it has managed to do so every summer since, often surviving in more than one location (Wellings 2007).

No specific eradication matrix has been determined for Ug99, however the general decision process as outlined in Figure 1 should be followed in determining if an incursion of this pest will be eradicated or managed/contained. The final decision between eradication and management will be made through the National Management Group.
Figure 1. Decision outline for the response to an exotic pest incursion
4 Pest information/status

4.1 Pest details

<table>
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<tr>
<th>Scientific name</th>
<th><em>Puccinia graminis f. sp. tritici</em></th>
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<tr>
<td>Pathotype</td>
<td>Ug99 pt. TTKSK, pt. TTKST, pt. TTKSF, pt. TTKSP</td>
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<td>Common names</td>
<td>Stem rust of wheat, stem rust of cereals, wheat rust, wheat stem rust, stem rust</td>
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4.1.1 Background

Rust fungi are plant pathogens that pose a particularly high biosecurity threat because they can travel large distances, build up rapidly, and those that attack economically important plant species are very damaging. A single epidemic of wheat stem rust during 1973 in south eastern Australia caused some $300 million in damage. The ability of rust pathogens to spread and build up rapidly also makes them extremely difficult to eradicate once introduced.

Australia faces threats not only from exotic rust species, but also from exotic isolates of endemic rust species. Long-term (80+ years) national studies of cereal rust pathogens conducted at the University of Sydney have documented 10 incursions of new races of endemic rust pathogens as well as two incursions of new cereal rusts. The rate of exotic cereal rust incursions has increased steadily since 1925 when these surveys began. Rust introductions have had serious implications for Australia's plant-based industries, and in the cereal industry have hindered attempts to control rust by genetic resistance.

The major current threats of exotic rust isolates to the Australian cereal industries are:

1. Stripe rust of barley caused by *Puccinia striiformis* f. sp. *hordei*. Overseas tests of Australian barley germplasm have shown that about 80% of current cultivars and advanced breeding lines are susceptible to stripe rust.
2. Leaf rust of durum wheat caused by *Puccinia* sp. Group II Type A. Pre-emptive breeding for resistance to leaf rust in durum wheat is currently being undertaken by one Australian durum wheat breeder and colleagues at ICARDA.
3. Crown rust of barley caused by *P. coronata var. hordei*. While barley crown rust must be regarded as an important quarantine threat, it does not impact greatly on barley production in North America where it occurs and therefore may not be damaging if introduced into Australia.
4. Wheat stem rust (caused by *Puccinia graminis* f. sp. *tritici*; *Pgt*) pathotype Ug99, first detected in Uganda in 1999 and now causing yield losses in Kenya and Ethiopia. Pathotype Ug99 has had a large impact on a wide range of wheat germplasm.

The fungal pathogen stem rust (*Puccinia graminis*) attacks many grasses, including the economically important cereals wheat, barley, oats and rye. Wheat stem rust is one of the most feared plant diseases, at times causing total crop failure. Recent studies of rDNA sequence data have confirmed the long-held belief that *P. graminis* is a genetically variable complex species (Zambino & Szabo
1993; Abbasi et al. 2005). It comprises variants known as formae speciales (“special forms”; f. sp.), which are morphologically identical but are specialised to different host species.

4.1.1.1 Puccinia graminis F. Sp. Tritici and pathotype Ug99

Of the three rust diseases that attack wheat, stem rust is the most damaging. It was the threat of this disease that led to the establishment of Rockefeller Foundation sponsored wheat breeding in Mexico, later to become the International Wheat and Maize Centre (CIMMYT). It has been said that it took this program about 10 years to produce wheats with durable resistance to stem rust, and a further 25 years for global adoption of this germplasm.

It is not known how Pgt was first introduced into Australia; however, it was suggested by Watson (1981) that it may have been present before European colonization, surviving on susceptible native grasses. McAlpine (1906) stated that rust had been present in Australia for at least 80 years, quoting the head librarian of the Public Library of NSW as referring to a publication thought to contain the earliest record of rust in wheat in Australia, from 1825: “rust sometimes appears, but is not very common” (Atkinson 1826). Although few reliable estimates of economic losses in wheat due to stem rust are available, reports do exist of losses of £2–3 million in 1889 (McAlpine 1906), £400,000 in 1903, £2 million in 1916 (Waterhouse 1929), £7 million in 1947 (Butler 1948), and $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.

Wheat stem rust epidemics were experienced in Kenya in the early 1990s following the breakdown of resistance (Sr36) in the popular cultivar Enkoy. A new pathotype of Pgt was detected in eastern Africa in 1999 (Ug99) (Pretorius et al. 2000). Since then, Ug99 has been detected in Uganda, Kenya and Ethiopia. This pathotype has caused considerable concern because of its broad virulence spectrum that includes gene Sr31, one of the most widely deployed stem rust resistance genes that remained effective until the detection of Ug99. Genes that are rendered ineffective by this pathotype are:

Sr5, Sr6, Sr7b, Sr8a, Sr8b, Sr9a, Sr9b, Sr9d, Sr9e, Sr9g, Sr11, Sr15, Sr17, Sr21, Sr30, Sr31, and Sr38

Those that remain effective are:

Sr7a, Sr13, Sr22, Sr24, Sr25, Sr26, Sr27, Sr28, Sr29, Sr32, Sr33, Sr35, Sr36, Sr37, Sr39, Sr40 and Sr44

Ug99 has had a large impact on a wide range of wheat germplasm, including that originating from the CIMMYT breeding programs. Accordingly, an expert panel was assembled and met in Nairobi Kenya on 29 May 2005, with Rockefeller funding. In essence, the meeting acknowledged the threat of Ug99 to stable wheat production in eastern Africa, and also recognised the threat posed to many other parts of the world given experience with stripe rust during the 1990s when circumstantial evidence supported the progressive migration of virulence for Yr9 from eastern Africa to south Asia. A detailed report was prepared and circulated, which proposed the formation of a “Borlaug Global Rust Initiative” (BGRI) to be led by CIMMYT/ICARDA to deal with the immediate problems in eastern Africa and to prepare other regions for the possible introduction of Ug99. Ten recommendations were proposed as a strategy for the BGRI. The BGRI was launched on 8th September 2005 at a meeting in Nairobi, which brought together a range of scientists, policy makers, and representatives of several funding agencies.
4.1.2 Life cycle

The stem rust pathogen *Puccinia graminis* has a complex life cycle. In regions where this species undergoes its entire life cycle, it produces five spore types and has two hosts. In Australia, the life cycle is restricted to the uredinial stage, which is in essence a succession of asexual spore production on cereal hosts. Teliospores are produced toward the end of the cereal (uredinial) host growing cycle, however, in Australia, teliospores are non-functional due to the absence of the alternate host *Berberis vulgaris*.
4.2 Affected Hosts

4.2.1 Host range

The fungal pathogen stem rust *Puccinia graminis* attacks many grasses. Three cereal-attacking formae specialae of *P. graminis* are recognised in Australia: *P. graminis* f. sp. *tritici*, *P. graminis* f. sp. *avenae* (*Pga*) and *P. graminis* f. sp. *secalis* (*Pgs*) (Park 2007). Whilst *Pgt* and *Pga* were recognised and studied in detail from the early 1900s, it was not until sometime later that *Pgs* was positively identified, when it was recorded from not only cereal rye but also from several grass hosts (Waterhouse 1957). Subsequent work based on pathogenicity analyses (Watson and Luig 1959) and isozyme analyses (Burdon et al. 1981) demonstrated the existence of another cereal attacking form of *P. graminis*, known locally as “scabrum” rust. This pathogen is often associated with the endemic grass *Elymus scaber* (formerly *Agropyron scabrum*) and also barley. A fourth form of *P. graminis* reported in Australia, *P. graminis* f. sp. *lolii* (*Pgl*), is associated with *Lolium* spp. and other grasses (Waterhouse 1951). While these four formae specialae and “scabrum” rust have been clearly distinguished in Australian studies of *P. graminis*, the identities of isolates of this species from other wild and cultivated grass species are not as clear. Comparative studies of the pathogenicities of isolates of *P. graminis* from *Dactylis* spp. and *Phalaris* spp. in particular provided evidence of two further formae specialae (*P. graminis* f. sp. *dactylidis*, *Pgd*; *P. graminis* f. sp. *phalaridis*, *Pgp*), however, Luig and Watson (1972) considered that at least one of these, *Pgp*, could be the product of a hybridisational event between two of the four recognised formae specialae.
The host range of \textit{Pgt} includes a wide range of cereal species, including those in Table 1.

\textbf{Table 1. Host range of \textit{P. graminis} f. sp. tritici.}

| Major hosts                  | \textit{Triticum aestivum} (wheat), \textit{Triticum turgidum} (durum wheat), \textit{Hordeum vulgare} (barley), \textit{Triticale} |

\section{4.3 Geographic distribution}

\textit{P. graminis} f. sp. \textit{tritici} is found in most countries worldwide, including Australia. The virulent strain Ug99 was first discovered in a Ugandan nursery in 1999 (Pretorius et al., 2000), and has since been detected in Kenya, Ethiopia, Sudan and Yemen, and in 2007 it was detected in Iran (Nazari et al. 2009). Since its first detection, two presumed mutational derivatives with virulence, one with virulence for \textit{Sr24} (Jin et al. 2008) and one with virulence for \textit{Sr36} (Jin et al. 2009), have been detected in Kenya (Jin et al. 2008), and a race with identical virulence but lacking virulence for \textit{Sr31} has been detected in South Africa (Visser et al. 2009) (Figure 4).

Publications in the international scientific literature are increasingly referring to Ug99 and derivative pathotypes by use of the North American \textit{Pgt} pathotype (pt; races or strains) nomenclature system:

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

As at December 2008, the following countries had confirmed presence of Ug99:

- Africa: Ethiopia, Kenya, Sudan, Uganda
- Middle East: Iran, Yemen
4.4 Symptoms

*P. graminis* can attack all above-ground parts of the plant, including the stem, leaves and inflorescence (Figure 5). Stem rust pustules are larger than those formed by leaf rust and tend to extend to both leaf surfaces. Infected wheat plants may also produce shrivelled grain. Symptoms also vary with the fungal life cycle stages (shown in Figure 3).

Symptoms by affected plant part:

- Inflorescence
- Leaves
- Seeds
- Stems
The virulent pathotype Ug99 presents the same symptoms as other pathotypes of *Pgt* on susceptible wheat plants. Differences between fungal pathotypes are seen as symptom development on wheat lines containing resistance genes effective against other pathotypes of stem rust.

**Figure 5.** Typical symptoms of stem rust infection in wheat

### 4.5 Pest risk analysis

#### 4.5.1 Entry pathways for exotic rust pathogens

**Rating: High**

Long-term (ca 80 years) national studies of cereal rust pathogens in Australia conducted at the University of Sydney have documented eight incursions of new pathotypes of endemic cereal rust pathogens as well as two incursions of new cereal rust diseases (Table 2). The origins of these fungi are generally unknown, as are the means of introduction. Watson & Sousa (1982) concluded that introductions 3 and 4 in Table 2 were transported to Australia from central Africa by high altitude winds. Wellings and McIntosh (1987) considered it likely that the wheat stripe rust pathogen was introduced to Australia from France by contaminated clothing. While the exact reason(s) are unknown, the increase in frequency in exotic incursions observed since surveys began could be associated with the increased international travel and contaminated clothing.
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<td></td>
<td></td>
</tr>
<tr>
<td>1. Wheat stem rust/Pgt</td>
<td>1925</td>
<td>?</td>
<td>Waterhouse (1952)</td>
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<tr>
<td><strong>Newly introduced cereal rust pathogens</strong></td>
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### 4.5.2 Spread and Establishment potential

**Rating: High**

Previous experience has shown that the cereal rust fungi have tremendous potential for spread once introduced. Examples exist of the pathogens causing wheat stem rust (Zwer et al. 1992), wheat leaf rust (Park et al. 1995) and wheat stripe rust (Wellings 2007) being dispersed across the Australian continent in as little as 12 months, and in many cases with subsequent dispersal to New Zealand. These situations have demonstrated quite clearly that once the threshold of detection is reached, eradication is not likely to work.

### 4.5.3 Economic impact

**Rating: High**

Wheat stem rust epidemics have the potential to cause large economic losses to the grains industry. For example, a single outbreak in south eastern Australia in 1973 caused $300 million in damage. If Ug99 were to become established, there would be an increase in the number of stem rust susceptible wheat varieties (more information provided in Section 7.2), increasing the likelihood of new epidemics occurring.

### 4.5.4 Overall risk

**Rating: High**
4.6 Preparedness for Ug99 in other countries

International efforts are already underway in many countries to address potential incursion of Ug99. While some developed countries have allocated direct funding for pre-emptive breeding for resistance (e.g. USA and Canada), most efforts in the developing world are targeting breeding for resistance surveillance, with funding from international agencies such as the Bill and Melinda Gates Foundation via the project “Durable Rust Resistance in Wheat” (DRRW), which is being managed by Cornell University (wheatrust.cornell.edu), and a range of other funding sources being administered by the UN-FAO.

5 Surveillance

5.1 Overview of surveillance systems for stem rust in Australia

Of all pathogens causing diseases in plants, Pgt is arguably the most closely monitored. Annual surveys of stem rust in wheat crops and pathotype analyses have been conducted on a continuous basis at the University of Sydney since the early 1900s. Surveys typically involve random inspections of crops and roadside self sown cereals and weed species, along with experimental plots, and are conducted by staff of the Australian Cereal Rust Control Program (ACRCP) at the University of Sydney and state based cereal pathologists, cereal breeders, extension staff, and in some cases cereal growers.

5.2 Australian pathotype identification and history

Identification of P. graminis to the species and formae specialis levels is usually done using symptoms and the host on which the rust occurs. It is also possible to identify some f. spp. based on ITS sequence information (e.g. Waipara et al. 2005).

Annual surveys of the cereal rusts not only document presence/absence and severity of rust diseases, they also provide viable rust isolates for pathotype identification. It is these pathogenicity analyses that led to the identification of three occasions on which exotic stem rust isolates entered Australia (Table 2), and also the subsequent evolution of new pathotypes from the exotic founding isolates. Pgt pathotypes detected in annual surveys over the past 40 years in Australia are all believed to have been derived from incursions 2, 3 and 4 in Table 2.

Physiologic races (pathotypes) of Pgt were first described by Stakman (1914). Annual pathogenicity surveys of Pgt conducted at the University of Sydney since 1919 have provided a sound basis for Australian stem rust resistance breeding efforts. The three incursions of four exotic Pgt isolates identified in these surveys have all had significant impacts on wheat production, highlighting the importance of current exotic threats such as Pgt race Ug99 and its variants. In each case, the introduced isolate acted as a “founding ancestor” that in time, via sequential mutations in genes conferring pathogenicity, gave rise to clonal lineages comprising closely related pathotypes. The clonal lineages are believed to have developed as follows:

- Lineage 1 – derived from the introduction of standard race 126 (first detected in Western Australia in 1925)
- Lineage 2 – derived from introduction of standard race 21 (first detected in 1954)
- Lineage 3 – derived from introduction of standard race 326
Studies of pathogenic variability in Pgt in Australia initiated by Waterhouse in 1919 utilised the 12 standard Stakman differentials\(^1\) (Stakman et al. 1962). Watson and Waterhouse (1949) demonstrated that pathogenic variability occurred within Australian isolates for Pgt for resistance genes not represented in the Stakman differentials, and consequently added differentials that became known as the Australian supplemental differential genotypes: Sr6 (introduced in 1941), Sr11 (1947), Sr9b, Sr36, Sr17, Sr8a and Sr15 (all during the 1950s), Sr30 (1960s), SrAgi (1971), SrEm (1973), Sr8b (1973), Sr27 (1982), and SrSatu (1983). Over time, it also became apparent that some of the Stakman differentials did not discriminate between isolates of Pgt in Australia, and consequently, the use of some of these differential genotypes was discontinued. Routine standard race designations are currently determined by pathogenicity on the varieties Reliance, Marquis, Acme, Vernal Emmer, Einkorn, and Line S (substituting for Khapli emmer), but periodically all Stakman differentials are used to confirm the identity of new pathotypes.

In reviewing surveys of pathogenicity for Pgt extending from 1919 to 1970, Luig and Watson (1970) identified three periods based on cultivar resistance and pathogenic variability. From 1919 to 1938, cultivars lacked effective resistance genes; from 1938 to 1964, cultivars with single genes for resistance were released (Eureka, Sr6; Gabo, Sr11; Festival and Gamenya, Sr9b; Mengavi, Sr36; Spica, Sr17), and new pathotypes with corresponding virulences were detected soon after. During the third period (1965 to 1970), the genetic base of resistance was broadened and cultivars with multiple factors for resistance were deployed, significantly reducing the occurrence of new pathotypes and consequent yield losses due to the disease.

Pathogenicity surveys from 1919 to 1925 detected six pathotypes (Waterhouse 1952). Standard race 126 was first detected in Western Australia in 1925, and was attributed to be of exotic origin. This race subsequently spread to eastern Australia, and by 1929 had virtually replaced the six originally detected. Race 126, along with several derivative pathotypes, predominated until 1954, when a new pathotype (21-0) was detected and quickly became widespread throughout eastern wheat growing regions. Pt 21-0 was very distinct from the 126- group and was regarded as having originated from infected Berberis in Tasmania (Watson 1958) or from Africa (Luig 1977). The frequency of this pathotype increased rapidly in the eastern wheat belt over the next few years, and concurrently, frequencies of those in the 126- group declined. Over 50 new pathotypes, all considered to have arisen via step-wise mutations tracing back to pt 21-0, were detected throughout the remainder of the 1950s and 1960s (Luig & Watson 1970). Pathotype 34-2,11, first detected in northern NSW in 1957, combined certain pathogenic features of isolates in both the 126- and the 21- groups, and on this basis was regarded as the product of somatic hybridisation between the two groups (Watson 1981). This was supported by subsequent studies in which isozymic variability was examined (Burdon et al. 1982). This pathotype, along with several derivative pathotypes, were common during the late 1950s and throughout the 1960s (Luig & Watson 1970), however, only pt 34-2,4,5,11 was of significance because it combined virulences for several resistance genes present in the cultivar Mendos (Sr7a, Sr11, Sr17, Sr36; Watson 1981).

The results of pathogenicity surveys for Pgt over the period 1969-1985 were reviewed by Zwer et al. (1992). The most striking trend during this period, continuing up to 2006 (R. F. Park, unpublished), was a decline in overall inoculum levels and in pathotype diversity in all wheat growing regions. Whereas pathogenicity surveys identified 41 pathotypes in 1973 (Luig and Watson 1977), only 24 pathotypes were detected from 1990 to 2005, and in some of these years, stem rust was not recorded.

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\(^1\) Varieties of wheat used to identify differences between pathotypes of stem rust.
in any Australian commercial wheat crop (R. F. Park, unpublished). The decline in levels of stem rust throughout Australia is thought to be a consequence of the release of cultivars carrying combinations of resistance genes such as \(Sr2, Sr24\) and \(Sr26\), and the continued effectiveness of these genes. The diversity and frequency of \(Pgt\) pathotypes in the years 1969 to 2005 were strongly shaped by three factors: the detection of two distinct pathotypes (194-1,2,3,5,6 and 326-1,2,3,5,6) in 1969, severe stem rust epidemics throughout south eastern Australia in 1973 and 1974, and the development of two \(Pgt\) pathotypes with the ability to attack certain triticale genotypes.

5.3 What additional surveillance is required to successfully detect Ug99?

The annual surveillance activities undertaken by the ACRCP (i.e. surveying the presence / absence of rust diseases and pathotype analyses) use standard methods that are well established and have proven successful in the timely detection of exotic rust incursions. Experience with this system has shown that the systemic deployment of trap plots to supplement information gained from commercial crops and experimental plots adds very little information, and that the effort involved in establishing such a system does not justify the small if any increase in resolution of detection. While it could be argued that more extensive crop inspections could lead to earlier detection, the benefit from doing so is by no means clear.

5.4 Modelling scenarios for Ug99

There is no way of knowing if and when Ug99 will occur in Australia – it may already be here or it may never get here. In deciding whether or not effort should be invested in modelling rust pathogen movement, it is vital to acknowledge that Ug99 represents but one of many dangerous exotic cereal rust threats to Australia (see Section 4.1.1). There may even be pathotypes of \(Pgt\) present in other parts of the world that pose a greater threat to the Australian wheat industry than does Ug99.

The DRRW project, funded by the Gates Foundation, has in Phase I (2008-10) undertaken a concerted effort to establish the current distribution of Ug99 and to track its spread, using a combination of stem rust surveillance, pathotype analysis, and GIS technologies ([wheatrust.cornell.edu/about/objective3.html](http://wheatrust.cornell.edu/about/objective3.html)). The leader of this work is Prof Robert Park at the University of Sydney. The aim was to establish a single system to service the world’s needs for rust surveillance. While this has proven to be “theoretically” straightforward, is has “in principle” been challenging because of the need for strong international co-operation, particularly in the sharing of information. Similar challenges were faced in establishing an international system for desert locust monitoring, which was successfully achieved by developing the Desert Locust Information Service (DLIS), located within the FAO’s Emergency Prevention System for Transboundary Animal and Plant Pests and Diseases (EMPRES). The long-term success of DLIS and FAOs ability to enter into dialogue with UN-member countries provided an excellent framework within which to develop an international rust surveillance system. A Global Cereal Rust Management System (GCRMS) was therefore based on the organizational model and data flows of the DLIS. It comprises a UN-FAO based International Focal Point (IFP), National Focal Points (NFPs), NARS supported National Surveillance Teams (drawn from both Plant Protection and Research Institutions), a Regional Pathologist based at ICARDA, a Coordinating Pathologist based at the University of Sydney, and various international partners (including Agriculture Agri-Foods Canada, CIMMYT, ICARDA, University of Free State [South Africa] and USDA-ARS).
The GCRMS is underpinned by an information platform that includes standardized protocols for methods and systems used in surveys, preliminary virulence testing, data, sample transmission and management at the field, national, and global levels. In order to provide access to stem rust information in a timely fashion, two web-based visualization tools were developed that are linked to a centralized database and released in the public domain: Rustmapper (www.cimmyt.org/gis/RustMapper/index.htm), a networked Google Earth application and RustMapper Web (www.cimmyt.org/gis/rustmapper/RustMapper_Web.html), a browser-based tool. Both tools incorporate updated stem rust survey data, near-real time wind trajectories, country level germplasm susceptibility estimates and distribution of major wheat growing areas. Both the tools and database are being updated on a routine basis, hence delivering the most recent information relating to stem rust in a timely manner.

A proposal to extend the funding for this surveillance work for another 5 years is currently under consideration by the Gates Foundation.

5.5 Delimiting survey and epidemiology study

Wheat stem rust, _Pgt_, is present in Australia, and delimiting survey and epidemiology study outlined in Sections 5.5.1-5.5.4 would only be required if an exotic or mutated pathotype, such as Ug99, was suspected.

5.5.1 Sampling method

Once initial samples have been received and preliminary diagnosis made, follow up samples to confirm identification of the pathotypes may be necessary. This will involve sampling directly from the infected crop, and sampling crops over a larger area to determine the extent of disease distribution.

From each crop sampled, at least 100 plants should be taken at random. However, preference may be given to symptomatic plants in fields where the disease incidence is low. All plants surveyed should be assessed for the presence of the stem rust symptoms.

Any personnel collecting samples for assessment should notify the diagnostic laboratory prior to submitting samples to ensure expertise is available to undertake the diagnosis. General protocols for collecting and dispatching samples are available within Appendix 3 of PLANTPLAN (Plant Health Australia, 2008).

A large number of samples may be collected and it is vital that a system of sample identification is determined early in the procedure to allow for rapid sample processing and accurate recording of results. Follow up samples will be forwarded to the nominated diagnostic laboratories for processing.

Samples should be initially collected over a representative area of the infected crop to determine the pathogen distribution. The disease may appear as patches within the crop depending on the source of the pathogen. It is important to note the distribution of disease in the initial crop, as this will indicate whether the pathogen has been seed-borne, carried on trash from adjacent paddocks or originated from contaminated machinery or human movement.

It is vitally important that all personnel involved in crop sampling and inspections take all precautions to minimise the risk of pathogen spread between crops and human health impacts by decontaminating between paddocks.

Samples should be collected from plants that represent a range of symptoms observed in the infected crop. Preferably enough material should be collected to allow for immediate processing and retention of a portion that can be placed into long term storage as a reference.
Samples should be treated in a manner that allows them to arrive at the laboratory well-preserved state. In this instance samples can be collected and transported between pieces of dry paper. The viability of the rust spores will not be compromised. In addition, in a dry state the sample is unlikely to become infected with saprophytic fungi and bacteria, which may render the sample unviable.

Samples should be processed as quickly as possible after sampling from the field if sub-cultures are to be made from infected tissue. Once removed from the field, fresh plant samples can deteriorate and become contaminated by other mould, fungi and bacteria, which may prevent successful sub-culturing of the pathogen. Sub-culturing should be done within three to four days after sampling from the field.

Long term storage of isolates can occur as fungal cultures that can be freeze dried for future reference (without loss of viability) or as deep frozen specimens maintained at -80°C.

It is important to record the precise location of all samples collected, preferably using GPS, or if this is not available, map references including longitude and latitude and road names should be recorded. Property and owners names should also be included where possible.

### 5.5.2 Epidemiological study

The number of infected plants within a crop will depend on the amount of inoculum available and whether conditions have been favourable for the pathogen to spread from the initial foci.

Sampling of crops within a district and beyond will be based upon the origins of the initial suspect sample(s). Factors to consider will be:

- The proximity of other susceptible crops to the initial infected crop, both in the current growing season and previous season. This will include the growers own crops and those on neighbouring properties. Alternative and wild host species should also be considered.
- What machinery or vehicles have been in the infected crop
- The extent of human movements in the infected crop. A possible link to recent overseas travel or visitors from other regions should also be considered

### 5.5.3 Models of spread potential

Spread may occur in the following ways:

- Spread through fungal spore movement is the major pathway for pathogen spread. Long distance movement of spores occurs by wind and by contaminated machinery, equipment, clothing and plant debris. Within a crop the spores are usually dispersed relatively short distances by rain splash but can sometimes be carried to neighbouring crops by windblown rain.
- Mechanical transmission through movement on contaminated vehicles and machinery
- Small fragments of plant material can be blown into surrounding paddocks during harvesting and allow the pathogen to move considerable distances away from the infected crop
5.5.4 Pest Free Area guidelines

The establishment and maintenance of Pest Free Areas (PFAs) would be a resource-intensive process, especially as the pathogen already occurs within Australia. Prior to development of a PFA due consideration should be given to alternative methods (e.g. treatments or enclosed quarantine) that achieve an equivalent biosecurity outcome. A benefit-cost analysis is useful for this purpose.

Additional information is provided by the IPPC (1995) in Requirements for the Establishment of PFA. This standard describes the requirements for the establishment and use of PFAs as a risk management option for phytosanitary certification of plants and plant products. Establishment of maintenance of a PFA can vary according the biology of the pest, pest survival potential, means of dispersal, availability of host plants, restrictions on movement of produce, as well as PFA characteristics (size, degree of isolation and ecological conditions).

Points to consider are:

- Design of a statistical delimiting field survey for symptoms on host plants
- Plant sampling should be based on at least 100 plants taken at random per crop
- Preliminary diagnosis can be based on leaf symptoms and fungal morphology
- Cereal rust pathotypes can only be identified by using seedling based greenhouse host assays
- Surveys should also consider alternative host plants

6 Diagnostic information

6.1 Current diagnostics for Ug99 (local and worldwide)

The only way to identify cereal rust pathotypes is by using seedling based greenhouse assays. Provided a good rust sample is received, these tests can be completed within 3 weeks. However this period of time for positive diagnosis is likely to be too long if eradication or containment were to be considered.

Progress has been made in recent years in the development of DNA-based fingerprinting using microsatellite or Simple Sequence Repeat (SSR) markers for Pgt. The application of some of these markers has shown clearly that the Ug99 lineage is distinct from North American (L. Szabo, personal communication) and South African (Visser et al. 2009). Pgt isolates, and preliminary testing in Australia using these markers and an additional set developed locally have confirmed its distinctiveness from Australia Pgt isolates (H. Karaoglu & R. F. Park unpublished).

Despite the distinctiveness of the Ug99 lineage, these studies have failed to identify SSR alleles that are unique to the Ug99 lineage. While not diagnostic for Ug99, the SSR markers that are now available do however provide a rapid means of demonstrating a given isolate of Pgt does not belong to the Ug99 lineage. The usefulness of this was recently demonstrated in establishing that stem rust outbreaks in Pakistan in early 2009 were not associated with Ug99. In this case, dead Pgt urediniospores were sent with AQIS approval to the University of Sydney, where DNA was extracted and SSR fingerprinting was conducted against a standard DNA sample from the original Ug99 accession, provided by Prof Z. A. Pretorius (University of the Free State, South Africa) (Figure 6).
Diagnostic protocol to determine Ug99

As already indicated, SSR analysis can indicate quickly that a given isolate does not belong to the Ug99 lineage. It does not, however, provide conclusive proof that a given isolate is Ug99, nor does it provide any indication of the virulence characteristics of a given isolate. Without the latter information, it is impossible to assess the potential impact of an exotic isolate on current wheat cultivars. The only way of doing this is a seedling based greenhouse assay, and it is highly unlikely that a DNA-based test that allows accurate discrimination of pathotypes will be developed in the next 10 years. A major problem with DNA-based tests will always be the possibility of contamination; rust samples collected from crops often comprise more than one pathotype, and the only way of establishing a pure isolate is to subculture onto seedlings in the greenhouse, a process that requires essentially the same infrastructure and time as a seedling based assay of virulence. The other difficulty with developing a DNA-based method of identifying pathotypes, or specific virulences, is that the difference between avirulence and virulence for a specific resistance gene could be a simple base pair change in the pathogen genome, which for Pgt is estimated to be about 85mb (Park 2000).
6.3 Diagnostic capabilities for stem rust in Australia and overseas

Staff at the University of Sydney’s Plant Breeding Institute have undertaken national pathogenicity surveys for all cereal rust pathogens since the early 1900s, and continue to do so with GRDC and University funding. There has however been an alarming decline in the global skill-base in cereal rust genetics, pathology and pathotype analysis over the past 30 years. Internationally, the only countries undertaking routine wheat rust pathotype analysis are Australia, Canada, Denmark (wheat stripe rust only), India, South Africa, and the USA, with only five people in the world with significant expertise in identifying pathotypes of *Pgt* (Australia, Prof R. Park; Canada, Dr T. Fetch; India, Dr M. Prashar; South Africa, Prof Z. Pretorius; USA, Dr Y. Jin).

7 Control methods

7.1 Chemical management of Ug99

Prior to the introduction of stripe rust in 1979, fungicides had not been used to control foliar pathogens in Australian wheat crops. Research on fungicidal control of stripe rust in wheat was conducted in the years after 1979, and has now become an important means of stripe rust control in situations where new pathotypes have arisen. For example, in 2008 some $40-50 million was spent on fungicidal control of stripe rust in NSW alone. Fungicides have also been used to control leaf rust in wheat in Australia particularly during epidemics experienced in WA in 1992 and again in 1999.

Fungicides have not been used to control stem rust in commercial wheat crops in Australia. Few studies have examined the efficacy of fungicides in controlling this disease, although those that have been conducted have shown fungicidal activity in some chemicals (Mayfield 1985, Afshari 2000, Loughman et al. 2005) and further demonstrated yield improvements that would justify the expense of chemical application. In experiments in which yield losses of up to 45% were recorded, Loughman et al. (2005) found that Folicur 430SC (a.i. tebuconazole) was more effective than Impact 250SC (a.i. flutriafol) or Triad (a.i. triadimefon) in reducing stem rust or improving yield or grain quality, however the timing of fungicide application was an important determinant in efficacy.

State Departments of Primary Industries maintain regularly updated lists of fungicides registered for controlling rust diseases in cereal crops. For example, chemicals currently recommended for rust control in wheat in Queensland are listed in Table 3.
Table 3. Fungicides registered (R) for control of rust disease in wheat in Queensland

<table>
<thead>
<tr>
<th>Active Ingredient</th>
<th>Trade name</th>
<th>Stripe rust</th>
<th>Stem rust</th>
<th>Leaf rust</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flutriafol</td>
<td>Force fungicide®</td>
<td>R</td>
<td>R</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Impact®</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Jubilee®</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Propiconazole</td>
<td>Aurora 250 EC®</td>
<td>R</td>
<td>R</td>
<td>R</td>
</tr>
<tr>
<td></td>
<td>Bumper 250 EC®</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Prestige®</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Propiconazole 250 EC®</td>
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<td></td>
<td></td>
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<tr>
<td></td>
<td>Slipstream 250EC</td>
<td></td>
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<tr>
<td></td>
<td>Tilt 250EC®</td>
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<tr>
<td></td>
<td>Tower 250EC®</td>
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<td></td>
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<tr>
<td></td>
<td>Tyrant®</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tebuconazole</td>
<td>Folicur 430 SC®</td>
<td>R</td>
<td>R</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Orius 430 SC®</td>
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<tr>
<td></td>
<td>Stingray®</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Tebuconazole 430 SC®</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triadimefon</td>
<td>Accord®</td>
<td>R</td>
<td>R</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bayleton 125 EC®</td>
<td></td>
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<tr>
<td></td>
<td>Slingshot®</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Triad 125 EC®</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Triadimefon 125 EC®</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Turret®</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Azoxystrobin + Cyproconazole</td>
<td>Amistar Xtra®</td>
<td>R</td>
<td>R</td>
<td></td>
</tr>
<tr>
<td>Propiconazole + Cyproconazole</td>
<td>Tilt Xtra®</td>
<td>R</td>
<td>R</td>
<td>R</td>
</tr>
</tbody>
</table>

While there are examples of some fungal plant pathogens developing insensitivities to fungicides, there are no verified reports of a rust pathogen of wheat developing insensitivity to a fungicide. On this basis, it is reasonable to assume that Ug99 will show the same sensitivity to fungicides as do Australian isolates of Pgt. Studies on chemical control of Ug99 have been initiated in Kenya as part of the DRRW project.

A problem associated with fungicidal control of rust diseases in broad-acre crops like wheat in Australia has been supply. Predicting requirement in a given year is very difficult and chemical suppliers do not like to maintain large stockpiles of active ingredient. It is believed that it is not likely Australia would have sufficient stockpiles of fungicide in the country should a stem rust epidemic of a new pathotype occur (Bodnurak pers. comm.).

7.2 Management of Ug99 – breeding for resistance

There has been a world-wide decline in the incidence of stem rust since breeding for resistance began in earnest, attributable largely to the widespread deployment of resistance genes, Sr2 and Sr31 in particular. More localised stem rust epidemics have nonetheless still occurred, albeit at a much reduced frequency.

Brennan & Murray (1988) estimated that efforts to breed wheat for resistance to Pgt have 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 & Walkden Brown (1968) and Luig (1983), and Luig & 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 & 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 & 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).

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 virulence’s 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.

Based on the knowledge of the stem rust resistance genes present in current Australian wheat cultivars (Table 4), an attempt was made to quantify the potential impact of Ug99 on the Australian wheat industry (Park & Bariana 2008). Data for grain receivals for 2005-06 were provided by the Australian Wheat Board, and from these, approximations of the area sown to cultivars with the same stem rust response based on the resistance genes present (Table 5) were estimated for three agroecological zones: north, south and west.

**Table 4.** List of commercially popular cultivars of wheat, with details of their stem rust resistance genes and their resistance to stem rust (both endemic strains and Ug99). Table modified from Bariana *et al.*, 2007.

<table>
<thead>
<tr>
<th>Queensland</th>
<th>Stem rust response&lt;sup&gt;3&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baxter</td>
<td>Sr2, Sr30, Sr36, MR, MS-S, R?</td>
</tr>
<tr>
<td>Kennedy</td>
<td>Sr2, Sr9g, Sr30, MR, -</td>
</tr>
<tr>
<td>Lang</td>
<td>Sr12, Sr24, Sr30, R, R</td>
</tr>
<tr>
<td>Strzelecki</td>
<td>Sr30, MS, MS-S</td>
</tr>
<tr>
<td>Sunco</td>
<td>Sr12, Sr24, Sr36, R, R</td>
</tr>
<tr>
<td>Sunstate</td>
<td>Sr2, Sr5, Sr8a, Sr12, Sr38, MR, MS-S</td>
</tr>
<tr>
<td>Sunvale</td>
<td>Sr5, Sr36, Sr38, R, R</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>New South Wales</th>
<th>Stem rust response&lt;sup&gt;3&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Babbler</td>
<td>Sr24, R-MR, R-MR</td>
</tr>
<tr>
<td>Chara</td>
<td>Sr9g, Sr30, MR-MS to MS, MS-S</td>
</tr>
<tr>
<td>Cunningham</td>
<td>Sr5, Sr8a, Sr12, Sr24, R-MR, -</td>
</tr>
<tr>
<td>Diamondbird</td>
<td>Sr2, Sr9g, Sr11, MS, MS</td>
</tr>
<tr>
<td>Drysdale</td>
<td>Sr2, Sr9g, Sr30, MR-MS, MS-S</td>
</tr>
<tr>
<td>EGA Wedgetail</td>
<td>Sr30, MS, MS-S</td>
</tr>
<tr>
<td>H45</td>
<td>Sr17, Sr30, MR-MS, MS-S</td>
</tr>
<tr>
<td>Janz</td>
<td>Sr5, Sr12, Sr24, R-MR, R-MR</td>
</tr>
<tr>
<td>Sunlin</td>
<td>Sr26, Sr34, MR, R</td>
</tr>
<tr>
<td>Wylah</td>
<td>Sr26, MR-MS, R</td>
</tr>
</tbody>
</table>

<sup>3</sup> R, resistant; R-MR, resistant to moderately resistant; MR, moderately resistant; MR-MS, moderately resistant to moderately susceptible; MS, moderately susceptible; MS-S, moderately susceptible to susceptible; S, susceptible

<sup>4</sup> Responses against the most appropriate pathotype based on the gene(s) present.
### Stem rust response

<table>
<thead>
<tr>
<th></th>
<th>Response</th>
<th></th>
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</thead>
<tbody>
<tr>
<td><strong>Victoria</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frame</td>
<td>Sr30</td>
<td>MR-MS</td>
<td>MS-S</td>
</tr>
<tr>
<td>Mitre</td>
<td>Sr24</td>
<td>R-MR</td>
<td>R</td>
</tr>
<tr>
<td>Yilpi</td>
<td>Sr30&lt;sup&gt;5&lt;/sup&gt;</td>
<td>MS-S</td>
<td>MS-S</td>
</tr>
<tr>
<td><strong>South Australia</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Excalibur</td>
<td>Sr8a, Sr15</td>
<td>MS</td>
<td>MS</td>
</tr>
<tr>
<td>Krickauff</td>
<td>Sr24</td>
<td>MR</td>
<td>-</td>
</tr>
<tr>
<td>Kukri</td>
<td>Sr2, Sr9g, Sr30</td>
<td>MR-MS</td>
<td>MR-MS to MS</td>
</tr>
<tr>
<td>Pugsley</td>
<td>Sr38, Sr30</td>
<td>MR-MS to MS</td>
<td>-</td>
</tr>
<tr>
<td>Trident</td>
<td>Sr12, Sr38</td>
<td>MS-S</td>
<td>-</td>
</tr>
<tr>
<td><strong>Western Australia</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arrino</td>
<td>Sr30</td>
<td>MS-S</td>
<td>MS-S</td>
</tr>
<tr>
<td>Calingiri</td>
<td>Sr30</td>
<td>MS</td>
<td>S</td>
</tr>
<tr>
<td>Carnamah</td>
<td>Sr2, Sr12, Sr30</td>
<td>MR-MS</td>
<td>MS-S</td>
</tr>
<tr>
<td>EGA Bonnie Rock</td>
<td>Sr30</td>
<td>MS</td>
<td>-</td>
</tr>
<tr>
<td>GBA Sapphire</td>
<td>Sr24, Sr36</td>
<td>R</td>
<td>R</td>
</tr>
<tr>
<td>Stiletto</td>
<td>Sr12, Sr13</td>
<td>MS</td>
<td>MS-S</td>
</tr>
<tr>
<td>Westonia</td>
<td>Sr9g</td>
<td>S</td>
<td>-</td>
</tr>
<tr>
<td>Wyalkatchem</td>
<td>Sr8a, Sr15</td>
<td>MS</td>
<td>MS</td>
</tr>
</tbody>
</table>

<sup>5</sup> Ineffective at the adult plant stage
Table 5. Approximate percentage of area sown to wheat cultivars carrying stem rust resistance genes in three agroecological zones in Australia (based on AWB receival data and stem rust genotype data from the ACRCP)

<table>
<thead>
<tr>
<th>Gene</th>
<th>North</th>
<th>South</th>
<th>West</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>% Area</td>
<td># cultivars</td>
<td>% Area</td>
</tr>
<tr>
<td>Sr2</td>
<td>33.4</td>
<td>12</td>
<td>15.0</td>
</tr>
<tr>
<td>Sr12</td>
<td>0.1</td>
<td>9</td>
<td>7.4</td>
</tr>
<tr>
<td>Sr13</td>
<td>0.01</td>
<td>1</td>
<td>0.6</td>
</tr>
<tr>
<td>Sr22</td>
<td>0</td>
<td>0</td>
<td>0.01</td>
</tr>
<tr>
<td>Sr24</td>
<td>15.7</td>
<td>9</td>
<td>25.5</td>
</tr>
<tr>
<td>Sr26</td>
<td>3.7</td>
<td>5</td>
<td>0.8</td>
</tr>
<tr>
<td>Sr30</td>
<td>6.5</td>
<td>10</td>
<td>38.2</td>
</tr>
<tr>
<td>Sr33</td>
<td>0</td>
<td>0</td>
<td>0.01</td>
</tr>
<tr>
<td>Sr36</td>
<td>30.3</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Sr38</td>
<td>17.5</td>
<td>7</td>
<td>10.7</td>
</tr>
</tbody>
</table>

The figures were then aggregated to come up with a national estimate of the area sown to cultivars considered to be resistant [R], moderately resistant [MR], moderately resistant to moderately susceptible [MR/MS], moderately susceptible [MS] and susceptible [S]. Cultivars rated as R to MR would not be expected to suffer yield loss in a stem rust epidemic year. The area was determined as it was in 2005-06, with current stem rust pathotypes, and what it would have been if Ug99 were present in Australia (Figure 7).
The data for the three regions on which Figure 7 is based are presented in Table 6, Table 7 and Table 8. Important points from this analysis include:

- Nationally the areas sown to MS and S cultivars was about 28%; this would have been 60% if Ug99 was present (Figure 7)

- Significant areas of the southern and northern regions were occupied by cultivars carrying Sr24 or Sr36, which would be vulnerable to the variants of Ug99 that have been found with virulence matching these genes (Table 5)

- The presence of Ug99 in Australia would not necessarily mean that an epidemic is imminent. For example, some 70% of the wheat acreage in WA in 2005-06 was either MS or S to the pathotypes of stem rust that occurred there at that time, and yet there was no epidemic during that year. In fact, only 33 samples were forwarded to PBI from WA for race analysis in 2005-06, and of these, only 4 came from commercial wheat crops (R. F. Park, unpublished). The 1973 stem rust epidemic in south eastern Australia was caused by a mutational derivative of pt 21-0, an exotic introduction first detected some 20 years earlier in 1954.
Table 6. Approximate percentage of area sown to wheat cultivars in the northern region in 2005-06 according to adult plant stem rust response (based on AWB\textsuperscript{6} receival data and stem rust genotype and field data from the ACRCP)

<table>
<thead>
<tr>
<th>Stem rust</th>
<th>- Ug99</th>
<th>+ Ug99</th>
</tr>
</thead>
<tbody>
<tr>
<td>R</td>
<td>83.8</td>
<td>44.6</td>
</tr>
<tr>
<td>MR</td>
<td>9.5</td>
<td>8.2</td>
</tr>
<tr>
<td>MR/MS</td>
<td>6.2</td>
<td>2.7</td>
</tr>
<tr>
<td>MS</td>
<td>0.5</td>
<td>34.3</td>
</tr>
<tr>
<td>S</td>
<td>0</td>
<td>6.3</td>
</tr>
</tbody>
</table>

Table 7. Approximate percentage of area sown to wheat cultivars in the southern region in 2005-06 according to adult plant stem rust response (based on AWB\textsuperscript{6} receival data and stem rust genotype and field data from the ACRCP)

<table>
<thead>
<tr>
<th>Stem rust</th>
<th>- Ug99</th>
<th>+ Ug99</th>
</tr>
</thead>
<tbody>
<tr>
<td>R</td>
<td>34.8</td>
<td>32.6</td>
</tr>
<tr>
<td>MR</td>
<td>34.1</td>
<td>0.7</td>
</tr>
<tr>
<td>MR/MS</td>
<td>14.4</td>
<td>1.2</td>
</tr>
<tr>
<td>MS</td>
<td>14.2</td>
<td>38.5</td>
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<tr>
<td>S</td>
<td>0</td>
<td>23.5</td>
</tr>
</tbody>
</table>

Table 8. Approximate percentage of area sown to wheat cultivars in the western region in 2005-06 according to adult plant stem rust response (based on AWB\textsuperscript{6} receival data and stem rust genotype and field data from the ACRCP)

<table>
<thead>
<tr>
<th>Stem rust</th>
<th>- Ug99</th>
<th>+ Ug99</th>
</tr>
</thead>
<tbody>
<tr>
<td>R</td>
<td>6.8</td>
<td>6.4</td>
</tr>
<tr>
<td>MR</td>
<td>19.3</td>
<td>0.9</td>
</tr>
<tr>
<td>MR/MS</td>
<td>3.3</td>
<td>0.1</td>
</tr>
<tr>
<td>MS</td>
<td>62.2</td>
<td>40.6</td>
</tr>
<tr>
<td>S</td>
<td>8.3</td>
<td>37.5</td>
</tr>
</tbody>
</table>

\textsuperscript{6} The author would like to acknowledge the support for the Grains Research and Development Corporation and the provision of wheat receival figures for 2005-06 by the Australian Wheat Board
8 Course of action

The information presented within this section is relevant only if eradication or containment of *Pgt* pathotype Ug99 will be attempted. This decision will be made by the NMG (see Section 3). Should the response to an incursion of Ug99 be the ongoing management of the pest with no attempt at containment, the required information can be found in the previous sections.

8.1 Infected crop destruction strategy

8.1.1 Destruction protocols

- Disposable equipment, infected plant material or soil should be disposed of by autoclaving, high temperature incineration or deep burial
- Any equipment removed from the site for disposal should be double-bagged

8.1.2 Decontamination protocols

Machinery, equipment and vehicles in contact with infected plant material or soil, or present within the Quarantine Area, should be washed to remove soil and plant material using high pressure water or scrubbing with products such as a farm degreaser or a 1% bleach (available chlorine) solution in a designated wash down. General guidelines for wash down areas are as follows:

- Located away from crops or sensitive vegetation
- Readily accessible with clear signage
- Access to fresh water and power
- Mud free, including entry and exit points (e.g. gravel, concrete or rubber matting)
- Gently sloped to drain effluent away
- Effluent must not enter water courses or water bodies
- Allow adequate space to move larger vehicles
- Away from hazards such as power lines
- Waste water, soil or plant residues should be contained (see PLANTPLAN 2008 Appendix 18)
- Disposable overalls and rubber boots should be worn when handling infected soil or plant material in the field. Boots, clothes and shoes in contact with infected soil or plant material should be disinfected at the site or double-bagged to remove for cleaning
- Skin and hair in contact with infected plant material or soil should be washed

8.1.3 Priorities

- Confirm the presence of the pest
- Prevent movement of vehicles and equipment through affected areas
- Priority of eradication/decontamination of infected host material
Determine the extent of infection through survey and plant material trace back

8.1.4 **Plants, by-products and waste processing**

- Infected plant material should be destroyed by (enclosed) high temperature incineration, autoclaving or deep burial (in a non-cropping area).
- As the fungus can be mechanically transmitted, killed crops should be ploughed in.

8.1.5 **Disposal issues**

- Particular care must be taken to minimize the transfer of infected soil or plant material from the area.
- No particular issues with resistance of disease to chemicals or physical treatments are known to exist.

8.2 **Quarantine and movement controls**

8.2.1 **Quarantine priorities**

- Plant material and soil at the site of infection to be subject to movement restrictions.
- Machinery, equipment, vehicles and disposable equipment in contact with infected plant material or soil to be subject to movement restrictions.
- Wind-borne inoculum can escape from stem rust infested crops, therefore the establishment of a quarantine area may be impractical

8.2.2 **Movement control for people, plant material and machinery**

Once symptoms of stem rust are observed the pathogen is usually well established in the crop and eradication difficult. Therefore, any zoning, quarantine or movement controls will usually pertain to containment and management.

Movement of people, vehicle and machinery, from and to affected farms, must be controlled to ensure that infected soil or plant debris is not moved off-farm on clothing, footwear, vehicles or machinery. This can be achieved through:

- Signage to indicate quarantine area and/or restricted movement in these zones
- Fenced, barricaded or locked entry to quarantine areas
- Movement of equipment, machinery, plant material or soil by permit only
- Clothing and footwear worn at the infected site should either be double-bagged prior to removal for decontamination or should not leave the farm until thoroughly disinfected, washed and cleaned
- Hay, stubble or trash must not be removed from the site
All machinery and equipment should be thoroughly cleaned down with a pressure cleaner prior to leaving the affected farm. The clean down procedure should be carried out on a hard surface, preferably a designated wash-down area, to avoid mud being re-collected from the affected site onto the machine.

8.3 **Zoning**

The size of each quarantine area will be determined by a number of factors, including the location of the incursion, biology of the pest, climatic conditions and the proximity of the infected property to other infected properties. This will be determined by the National Management Group during the production of the Response Plan. Further information on quarantine zones in an Emergency Plant Pest incursion can be found in PLANTPLAN, Appendix 10 (Plant Health Australia, 2008). These zones are outlined below.

8.3.1 **Destruction zone**

If destruction of hosts is considered, the entire crop should be destroyed after the level of infection has been established. The delimiting survey will determine whether or not neighbouring host crops are infected and need to be destroyed.

The Destruction Zone will usually be the entire crop but may be the entire farm or contiguous areas of management if spread is likely to have occurred prior to detection.

If the movement of air-borne inoculum to adjacent crops appears likely, they will also need to be destroyed.

8.3.2 **Quarantine zone**

The Quarantine Zone is defined as the area where voluntary or compulsory restraints are in place for the affected property(ies). These restraints may include restrictions or movement control for removal of plants, people, soil or contaminated equipment from an infected property.

8.3.3 **Buffer zone**

A Buffer Zone may or may not be required depending on the incident. It is defined as the area in which the pest does not occur but where movement controls or restrictions for removal of plants, people, soil or equipment from this area are still deemed necessary. The Buffer Zone may enclose an infested area (and is therefore part of the Control Area) or may be adjacent to an infested area.

8.3.4 **Restricted Area**

The Restricted Area is defined as the zone immediately around the infected premises and suspected infected premises. The Restricted Area is established following initial surveys that confirm the presence of the pest. The Restricted Area will be subject to intense surveillance and movement control with movement out of the Restricted Area to be prohibited and movement into the Restricted Area to occur by permit only. Multiple Restricted Areas may be required within a Control Area.
8.3.5 Control Area

The Control Area is defined as all areas affected within the incursion. The Control Area comprises the Restricted Area, all infected premises and all suspected infected premises and will be defined as the minimum area necessary to prevent spread of the pest from the Quarantine Zone. The Control Area will also be used to regulate movement of all susceptible plant species to allow trace back, trace forward and epidemiological studies to be completed.

8.4 Decontamination and farm clean up

Decontaminant practices are aimed at eliminating the pest thus preventing its spread to other areas.

8.4.1 Decontamination procedures

General guidelines for decontamination and clean up:

- Keep traffic out of affected area and minimize it in adjacent areas
- Adopt best-practice farm hygiene procedures to retard the spread of the pest between fields and adjacent farms
- Machinery, equipment, vehicles in contact with infected plant material or soil or present within the Quarantine Area, should be washed to remove soil and plant material using high pressure water or scrubbing with products such as a detergent, farm degreaser or a 1% bleach solution in a designated wash down area. Plant material should be destroyed using herbicide. Only recommended materials are to be used when conducting decontamination procedures, and should be applied according to the product label

Refer to PLANTPLAN (Plant Health Australia 2008) for further information

8.4.2 General safety precautions

For any chemicals used in the decontamination, follow all safety procedures listed within each MSDS.

8.5 Surveillance and tracing

8.5.1 Surveillance

Detection and delimiting surveys are required to delimit the extent of the outbreak, ensuring areas free of the pest retain market access and appropriate quarantine zones are established.

Initial surveillance priorities include the following:

- Surveying all host growing properties in the pest quarantine area
- Surveying all properties identified in trace-forward or trace-back analysis as being at risk
- Surveying all host growing properties that are reliant on trade with interstate or international markets which may be sensitive to Ug99 presence
- Surveying other host growing properties

### 8.5.2 Survey regions

Establish survey regions around the surveillance priorities identified above. These regions will be generated based on the zoning requirements (see Section 8.3), and prioritised based on their potential likelihood to currently have or receive an incursion of this pest. Surveillance activities within these regions will either allow for the area to be declared pest free and maintain market access requirements or establish the impact and spread of the incursion to allow for effective control and containment measures to be carried out.

Steps outlined in Table 9 form a basis for a survey plan. Although categorised in stages, some stages may be undertaken concurrently based on available skill sets, resources and priorities.

**Table 9. Phases to be covered in a survey plan**

| Phase 1 | Identify properties that fall within the buffer zone around the infested premise  
|         | Complete preliminary surveillance to determine ownership, property details, production dynamics and tracings information (this may be an ongoing action) |
| Phase 2 | Preliminary survey of host crops in properties in buffer zone establishing points of pest detection |
| Phase 3 | Surveillance of an intensive nature, to support control and containment activities around points of pest detection |
| Phase 4 | Surveillance of contact premises. A contact premise is a property containing susceptible host plants, which are known to have been in direct or indirect contact with an infested premises or infected plants. Contact premises may be determined through tracking movement of materials from the property that may provide a viable pathway for spread of the disease. Pathways to be considered are:  
|         | Items of equipment and machinery which have been shared between properties including bins, containers, irrigation lines, vehicles and equipment  
|         | The producer and retailer of infected material if this is suspected to be the source of the outbreak  
|         | Labour and other personnel that have moved from infected, contact and suspect premises to unaffected properties (other growers, tradesmen, visitors, salesmen, crop scouts, harvesters and possibly beekeepers)  
|         | Movement of plant material and growing media/soil from controlled and restricted areas  
|         | Storm and rain events and the direction of prevailing winds that result in air-borne dispersal of the pathogen during these weather events |
| Phase 5 | Surveillance of production and retail nurseries, gardens and public land where plants known to be hosts of pathogen are being grown |
| Phase 6 | Agreed area freedom maintenance, pest control and containment |
8.5.3 Post-containment surveillance

The period of pest freedom sufficient to indicate that containment of the pest has been achieved will be determined by a number of factors, including cropping conditions, the level of infection and the control measures applied. As a guide, the following activities should be carried out following the containment of the pest:

- Establishment of sentinel plants around the site of infection but outside the containment zone
- Sentinel plants should remain in place and inspected on a fortnightly basis for a further 6 weeks and then on a monthly basis
- Surveys comprising plant sampling for and testing for Ug99 to be undertaken for a minimum of 12 months after containment has been achieved


Atkinson J (1826). An account of agriculture and grazing in New South Wales. London (cited from McAlpine D (1906)).


10 Appendices

10.1 Appendix 1. Standard diagnostic protocols

For a range of specifically designed procedures for the emergency response to a pest incursion refer to Plant Health Australia’s PLANTPLAN.

10.2 Appendix 2. Experts, resources and facilities

The Plant Breeding Institute, University of Sydney, is the only facility with the diagnostic capability for Pgt pathotype Ug99 (and its derivatives) in Australia. Contact details are shown in Table 10.

<table>
<thead>
<tr>
<th>Expert</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prof Robert Park</td>
<td>GRDC Chair of Cereal Rust Research</td>
</tr>
<tr>
<td></td>
<td>Plant Breeding Institute, University of Sydney</td>
</tr>
<tr>
<td></td>
<td>107 Cobbitty Rd, Cobbitty NSW 2570 Australia</td>
</tr>
<tr>
<td></td>
<td>Ph: 02 9351 8806</td>
</tr>
<tr>
<td></td>
<td>Fax  02 9351 8874</td>
</tr>
</tbody>
</table>

10.3 Appendix 3. Communications strategy

A general Communications Strategy is provided in PLANTPLAN (Plant Health Australia, 2008)

10.4 Appendix 4. Market access impacts

Within the AQIS PHYTO database, no countries appear to have a specific statement regarding area freedom from *P. graminis* f. sp. *tritici*, including pathotype Ug99 (April 2009). Should Pgt pathotype Ug99 be detected or become established in Australia, some countries may require specific declaration. Latest information can be found within PHYTO, using an Advanced search “Search all text” for *Puccinia graminis* f. sp. *tritici*. 