UK Pest Risk Analysis for *Tilletia indica*

**STAGE 1: PRA INITIATION**

1. **What is the name of the pathogen?**
   *Tilletia indica* Mitra
   Synonym: *Neovossia indica* (Mitra) Mundkur
   Fungus: Basidiomycete; Ustilaginales
   Common name: Karnal or partial bunt of wheat and other cereals.

2. **What is the reason for the PRA?**
   This PRA is the fourth revision of the first UK PRA (Sansford, 1996, *unpublished*; Sansford, 1998) which was originally prepared as a result of the first report of *T. indica* in the USA in 1996 (Ykema *et al*., 1996).

   This first PRA addressed a new risk to the UK, EU and EPPO\(^1\) region arising from an existing trade pathway i.e. on wheat grain exported from the USA to Europe. During and prior to 1996 the published trade figures suggested that no pathway of entry appeared to exist from countries where *T. indica* was known to occur (i.e. no imports of cereal grain or seed from these countries were recorded in the trade figures available at the time). The UK PRA predicted a risk of entry based principally on trade pathways, a risk of establishment in Europe and the potential for economic damage through quality losses (downgrading of milling wheat to feed wheat) as well as losses in export markets. To manage the risk, *T. indica* was listed as a I/AI quarantine pest by the EC in 1997. Many cereal-importing countries outside of the EU also imposed phytosanitary regulations against the pathogen in response to the US finding.

   The potential risk of entry needed to be reassessed and appears to have increased since 1997 in particular because of changes to United States Department of Agriculture (USDA) policy in the methods by which affected areas and articles are regulated. Also, interceptions have occurred in several European countries subsequent to the implementation of legislation against *T. indica*. Three updated PRAs (two drafts, one final) were prepared in 2003 in response to a request from Defra Plant Health Division and the Plant Health and Seeds Inspectorate to support the development of a draft contingency plan (Sansford *et al*., 2004 (latest version), *unpublished*) which has been prepared to prevent or manage interceptions of *T. indica* in trade and manage potential outbreaks of Karnal bunt in the UK and which will be the subject of public consultation.

   This fourth revision of the PRA updates the science and the status of the pathogen but gives key references only. However, the main scientific and socio-economic findings from an EU Fifth Framework Research Project ‘*Karnal bunt risks*’ are summarised, pending full publication of results by the Project Partners (see Acknowledgement). The principal objective of the Project is to produce a further and fully revised PRA by the end of 2004 (see Anon., 2004).

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\(^1\) The European and Mediterranean Plant Protection Organisation, the Regional Plant Protection Organisation for 45 countries including the Member States of the EU
3. **What is the PRA area?**  
The PRA area is the UK and includes analysis where possible for the EU and EPPO region.

**STAGE 2: PEST RISK ASSESSMENT**

4. **Is the pest established in the PRA area or does it maintain transient populations?**  
No, it is not established and does not maintain transient populations.

5. **Is there any other reason to suspect that the pest is already established in the PRA area?**  
No.

6. **What is the pest’s EPPO status?** *(A1/A2 quarantine pest or Alert List?)*  
*T. indica* is in the EPPO A1 category of organisms recommended for quarantine listing.

7. **What is the pest’s EC Plant Health Directive status?**  
*T. indica* is an EC I/AI listed quarantine pest (Anon., 2000 as amended). Measures are in place in individual Member States including the UK aimed at preventing entry. EC requirements are for seeds of *Triticum* (wheat), *Secale* (rye) and *x Triticosecale* (triticale) imported from countries where the pathogen occurs to originate in an area where *T. indica* is known not to occur. For grain there are two options: 1. To originate in an area where *T. indica* is known not to occur, or 2. For place of production freedom based upon inspection for symptoms of *T. indica* during the growing season and testing of the grain for freedom from the pathogen at harvest and testing for freedom from the pathogen again before shipment.

8. **What are its host plants?**  
The natural host plants of *T. indica* are bread and durum wheat (*Triticum aestivum* and *Triticum durum*) as well as triticale (*x Triticosecale*). Despite being currently regulated (see 7.), rye (*Secale cereale*) is no longer considered to be a host.

9. **What hosts are of economic and/or environmental importance in the PRA area?**

   9.1 **Economic**  
   Bread wheat, durum wheat and triticale are of economic importance in the UK, EU and EPPO region.

   9.2 **Environmental**  
   There are no hosts of environmental importance in the PRA area.

10. **If the pest needs a vector, is the vector present in the PRA area?**  
No vector is required.

11. **What is the pest’s present geographical distribution?**  
The pathogen was first found in the Karnal district of northern India in 1930 (Mitra, 1931). All countries for which there is a record of the pathogen are given below. In addition to
Mexico, some detail of the distribution of *T. indica* is given for the countries where the pathogen has been reported for the first time since the mid-1990’s.

<table>
<thead>
<tr>
<th>Region</th>
<th>Country(s)</th>
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<tbody>
<tr>
<td>North America</td>
<td>USA – present since 1996 in the states of Arizona, California and Texas; few records.</td>
</tr>
<tr>
<td>Central America</td>
<td>Mexico – present since 1969-70; limited distribution confined to the states of Sonora and Sinaloa.</td>
</tr>
<tr>
<td>South America</td>
<td>Brazil – present since at least 1990 in the southern part of the Rio Grande do Sul; few records.</td>
</tr>
<tr>
<td>Caribbean</td>
<td>Absent - no records.</td>
</tr>
<tr>
<td>Europe</td>
<td>Absent - no records.</td>
</tr>
<tr>
<td>Africa</td>
<td>South Africa – present since 2000 in Douglas, Northern State Province; few records.</td>
</tr>
<tr>
<td>Asia</td>
<td>Afghanistan - present, no details.</td>
</tr>
<tr>
<td></td>
<td>India - present and widespread.</td>
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<tr>
<td></td>
<td>Iran - present since 1996, limited distribution.</td>
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<tr>
<td></td>
<td>Iraq - present, no details.</td>
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<td></td>
<td>Nepal - present, no details.</td>
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<td></td>
<td>Pakistan - present, limited distribution.</td>
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<tr>
<td>Australasia</td>
<td>Absent – no records.</td>
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</table>

12. **Could the pest enter the PRA area?**

Yes. The most likely pathway for *T. indica* to enter is via international trade in seed and grain of wheat or triticale that has been infected or contaminated with *T. indica*.

At low levels the disease is difficult to detect in the growing crop; symptoms affect the ear at heading time and can be hard to find. If crops grown in areas where the pathogen is known to occur are not tested for the pathogen itself (i.e. laboratory tests for teliospores), then it is possible that *T. indica* may be present in the harvested grain or seed and, if destined for export, then the pathogen could be moved in trade.

In 1996, *T. indica* was intercepted in Poland in grain of *T. aestivum* from India. Despite the existence of EC legislation (see 7.) and exporting country requirements since 1997, several interceptions of *T. indica* have been reported subsequently in the PRA area. These were in Italy in 1998 on two consignments of grain of *T. durum* from Mexico; in the UK in 2003 and again in 2004 in grain of *Triticum* sp. and *T. durum* respectively from India; and possibly in Greece in 1996 on grain of *T. durum* and also possibly on *T. aestivum* from the USA.

Regarding the risk of entry of *T. indica* from imports of wheat originating in the USA, the USDA Animal and Plant Health Inspection Service (APHIS) adopted an interim rule in May 1997 that there would be a requirement for a ‘bunted kernel’ (a wheat grain with visible disease symptoms) to be ‘*found in or associated with a field within an area, before the area be designated as regulated*’. This rule (adopted as a final rule in 1999) could allow low levels of infection to go undetected and for teliospores of the pathogen to potentially be present in grain shipments derived from infested crops originating in the USA. This method of regulating areas does not meet with the EC phytosanitary requirement for freedom from the pathogen *T. indica*; all it seeks to guarantee is freedom from symptoms of Karnal bunt. US wheat seed for sowing is still tested for the presence of the pathogen itself prior to export. In July 2003 USDA APHIS made an announcement of a proposed
revision to domestic regulations in the USA for Karnal bunt (USDA, 2003); the revisions became final in 2004 (USDA, 2004). With few exceptions, the main premise of the revision is the requirement for bunted kernels to be found in most circumstances before any official action is taken, rather than sampling and testing for teliospores of *T. indica*. Seed is still tested for freedom from the pathogen. Although this revision of the regulations may alleviate pressure on farmers and the officials involved in inspection, the potential result is for further spread of the pathogen in the USA. The USA exports wheat to many EU Member States (see 16.1, Table 2). The potential risk of entry to the PRA area may therefore have increased. European Commission Plant health officials are in correspondence with their counterparts in the United States to clarify the basis on which US exports of wheat are certified as meeting the EU’s requirements with regard to *Tilletia indica*.

The pathway for the pathogen to enter via seed is clear since planting infected or contaminated seed in arable land will allow the pathogen to enter the PRA area. The pathway for the pathogen to enter via grain will depend upon the location of the port of entry and the route that the consignment takes post-entry as well as its final destination and intended use. Infected or contaminated grain destined for transport through, or for processing in areas where wheat is grown, poses the highest risk, since teliospores can be released during transportation and by handling operations. In addition to seed and grain there are other potential routes of entry including imports and post-entry movement of agricultural machinery and equipment that have been contaminated with spores.

With respect to other potential sources of entry, until the late 1990’s India and Pakistan were principally wheat importers. Huge harvests in the region thereafter led both nations to become wheat exporters (see FAOSTAT, 2004b). Although the majority of the wheat grown in this region is destined for other Asian countries the potential for dissemination of *T. indica* from this part of the world has increased.

Specific and recent details of imports to the UK from countries where *T. indica* occurs (HGCA, 2004) show that between July 2002 and June 2003, 91,708 tonnes of wheat (‘wheat’ not defined; i.e. not broken-down into grain or seed, or bread or durum wheat) were brought in from the USA, 2,803 tonnes from Brazil and 26 tonnes from India. Between July 2003 and June 2004, 24,165 tonnes were imported from the USA and 57 tonnes from India. For other countries in the PRA area, import figures are only available to June 2001 from FAOSTAT (2004b); between July 2000 and June 2001 the only country where *T. indica* occurs that appears to have exported wheat was the USA, with Italy being the recipient of the largest amount (1,071,291 tonnes) of all the countries in the PRA area (see 16.1, Table 2).

**13. Could the pest establish outdoors in the PRA area?**

Yes. Pathway(s) of entry into the PRA area exist and the UK, EU and EPPO region produce all of the susceptible host crops (bread wheat, durum wheat and triticale).

The assessment of the risk of establishment depends upon a combination of several other factors which relate to the potential lifecycle and behaviour of the pathogen in relation to European meteorological and soil factors and the phenology and susceptibility of European cultivars of its main host, wheat (*T. aestivum* and *T. durum*) in the PRA area. The full life-cycle of the pathogen has been described in many publications and in earlier versions of the PRA and therefore is only briefly described here:
13.1 Brief description of the life-cycle of \textit{T. indica}

Karnal bunt of wheat is a monocyclic disease. The pathogen is soil-borne and seed-borne in the form of teliospores but it is not directly transmitted from the seed to the plant. The pathogen is airborne in the form of sporidia or teliospores. The life-cycle commences when teliospores, present on or very near to the soil surface, germinate and produce primary sporidia. Primary sporidia germinate to produce mycelium on plant or other surfaces, which in turn produces secondary sporidia. Secondary sporidia are splashed by rain or blown by wind. If these are deposited onto the flag leaf or ear of wheat plants at the susceptible stage for infection (broadly defined as at the ‘heading’ stage here for simplicity), under suitable climatic conditions, the pathogen can infect the developing grain through the glumes. The disease typically develops in only some of the grains in the ear, and each grain usually only becomes partially bunted. At harvest, teliospores are usually dispersed locally from bunted grain by the mechanical action of harvesting; bunted grain itself will also act as a vehicle for long-distance dispersal of inoculum in trade. The teliospores or bunted grain become deposited in the soil, in the harvesting machinery, and, teliospores can adhere to the surface of healthy grains as an external contaminant. Inoculum can be further transported in soil or in/on grain or seed on farm machinery and vehicles used for grain or seed transportation, or even by wind. The ingestion of infected or contaminated grain by livestock can also lead to the dispersal of teliospore inoculum in animal faeces. Animals can act as a vehicle for surface contamination by teliospores, facilitating spread. According to the scientific literature from India, Mexico and the USA, teliospores can survive in the soil for up to five years.

13.2 Potential for the establishment of \textit{T. indica} in Europe based upon the results of the EU Fifth Framework RTD Project, ‘Karnal bunt risks’ (Anon., 2004)

The epidemiology of Karnal bunt was discussed in the first UK PRA in 1996. It was concluded at that time that there were no limiting factors preventing the establishment of the pathogen in the PRA area. In support of the development of the assessment of establishment risk for Europe, experimental results from the current EU Project (Anon., 2004) described below, show that there remains a risk of establishment of \textit{T. indica} should it enter the PRA area. The Project has assessed the ability of the pathogen to survive between crops, the ability of the over-wintering phase of the pathogen (the teliospores) to germinate to produce infective spores at the time when the host is susceptible to infection and the occurrence of appropriate climatic conditions for infection of the crop and subsequent disease development. The susceptibility of European wheat cultivars was also assessed.

13.2.1 Teliopore survival under European conditions

Results from experimental work conducted in Italy, Norway and the UK investigating teliospore survival in Europe, showed that teliospores survived at all depths (5, 10 and 20 cm) after a range of 1-, 2- and 3-year burial periods in European soils in the field under quarantine containment. (Buried in 2000, 2001 and 2002 at approximately wheat harvest time and sampled at yearly intervals between 2001 and 2003). The risk of establishment remains as the pathogen can survive for at least one year; the data suggests that survival may in fact significantly exceed three years. Some of the results from this part of the EU Project were published in 2002 (Valvassori \textit{et al.}, 2002).
Experimental results also showed that a proportion of teliospores (ca. up to 50%) could be capable of germination under suitable conditions when brought to the soil surface after 1–3 years of burial. The timing of germination of viable teliospores in the remaining portion in relation to the important wheat phenological growth stages for infection (see 13.2.2) is critical for potential establishment and the development of disease and is discussed below (see 13.2.3).

The results of this European study agree broadly with those reported in India and USA studies in that teliospore survival can occur over several years. As reported in a recently-reported study under quarantine containment in Montana, USA (Babadoost et al., 2004), there was evidence for a decline in teliospore numbers within the first year of burial in European soil conditions, but then very little decline over subsequent years. The data suggest that teliospores which survive for one year are likely to survive significantly beyond three years. In contrast, Indian studies predict a steady decline in teliospore survival over time when buried in soil, and that maximum survival is unlikely to exceed 3–4 years under Indian soil conditions.

13.2.2 Timing of the susceptible growth stage of wheat and European wheat cultivar susceptibility

Further unpublished data from the EU Project using European winter, spring and durum wheat cultivars and the known susceptible Indian spring wheat cultivar WL-711 as the control, as well as the existing scientific literature (e.g. Nagarajan et al., 1997), suggest that the susceptible period for infection of wheat is growth stage (GS) 43 (boot just visibly swollen) to GS69 (anthesis complete). However, the most likely timing in the field is likely to be from GS47/49 (flag sheath opening to first awns visible) to GS55 (half of inflorescence emerged) depending upon environmental conditions. The potential window of growth stages which are susceptible to infection may be longer for some cultivars than others. Almost all European winter, spring and durum wheat cultivars tested were shown to be physiologically susceptible to T. indica by artificial inoculation of the boot cavity. The range of susceptibilities was similar to cultivars grown in countries such as India and Mexico where T. indica is established. Many European winter, spring and durum wheat cultivars were shown to be morphologically susceptible to infection (field susceptibility determined by spray inoculation of the ear) both in the EU Project and for European winter wheat cultivars in other similar work in the UK and the US (A. Inman, CSL, UK and G. Peterson, USDA Agricultural Research Service (ARS); 2004, personal communications).

13.2.3 Timing of teliospore germination relative to the susceptible growth stage of wheat

Detailed investigations have been undertaken in the EU Project to determine the timing of teliospore germination during the wheat growing period in a range of European climates. Results suggest that teliospores would be likely to germinate in most soil types and under a range of soil moistures and temperatures in European countries.

Utilising five years of available soil surface and air temperature data from the time of autumn planting of wheat until anthesis, obtained from wheat growing regions of
Sonora, a Karnal bunt area in Mexico, as well as the UK, Norway, Italy and Hungary, investigations were conducted in containment growth rooms into the likely timing of teliospore germination under minimum, optimum and fluctuating soil moisture conditions. Results for treatments where soil moisture was held at a constant 25 % (optimum) showed that there was a significant drop in the number of teliospores available for infection at the susceptible period of infection of wheat due to early germination, except in Norway, where sub-freezing temperatures maintained a large proportion of the teliospores that were still viable at heading time, and available for germination to produce infective sporidia. Results also showed however, that in all locations, despite the presence of conducive germination conditions, a small proportion of teliospores continued to germinate through the simulated flowering stage of wheat, demonstrating a form of physiological dormancy within the population. At all locations, teliospores held at the minimum soil moisture level for germination (10%) showed that a significant number of teliospores remained ungerminated throughout the length of the profile and would be available to germinate and produce infective sporidia at the time of anthesis. Teliospores subjected to weekly periods of drying under profiles similar to UK, Hungarian and Italian climatic conditions showed a high level of teliospore recovery throughout the experiment but a significant drop in viability over time. Those exposed to Mexican and Norwegian profiles also showed a high teliospore recovery rate throughout the experiment, but a significant level of spore viability during the entire length of the experiment. In this study, the data also indicated that, when compared to those results obtained from the Mexican profiles, Italy would be an area with the greatest establishment potential for *Tilletia indica*.

The data obtained from these studies highlights two factors that can result in early decline of potential inoculum at the soil surface: either the occurrence of soil moisture conditions conducive to early teliospore germination and/or a decline in teliospore viability observed when soil moisture conditions fluctuate over the season. Results suggest that in most regions of the EU, a significant number of viable teliospores on the soil surface would be eliminated prior to the window of infection, however, some level of inoculum would remain and would be available at the susceptible period of infection for the wheat crop, thus supporting the potential establishment of *Tilletia indica* in the PRA area.

### 13.2.4 Prediction of the risk of establishment based upon climate

With respect to the suitability of the climate in the PRA area, Warham (1986) (in a review of the literature) concluded that low temperatures and high humidity are necessary at wheat anthesis for infection to occur, whilst dry weather, high temperatures and bright sunlight are unfavourable conditions for the infection process. Rainfall is necessary but rainfall on its own at flowering is not enough to cause infection, suggesting that a specific combination of climatic factors is required. Crop irrigation is an additional factor favouring disease. Taking these factors into account, the first UK PRA in 1996 showed a clear risk of establishment in the PRA area based upon this information as part of a review of the literature, as well as the use of the Humid Thermal Index (HTI) of Jhorar *et al.*, (1992) as described below.

Jhorar *et al.*, (1992) modelled the disease, Karnal bunt, in the central Punjab, India, by an empirical method. A study of the relationships between ‘plant disease intensity’ (not defined) of Karnal bunt in wheat and meteorological factors, using
historical meteorological data and data pertaining to disease intensity for Ludhiana district, in the central plain of the Punjab, was made for the reproductive stage of the crop. The period studied corresponded to flag leaf emergence (starting 12 February in Ludhiana) and subsequent stages (ending on 18 March). This corresponded to the most important period for the pathogen, when teliospores that geminated at that time could lead to the production of infective sporidia that survived to infect the host and for Karnal bunt to develop.

During the period that meteorological factors were analysed, the relationships between these and plant disease intensity were only found to be strong, during the third, fourth and fifth week. Of all the factors, the most significant relationship was found between disease intensity and evening relative humidity (A), maximum temperature (B), and the ‘Humid Thermal Index’ (A/B). A ‘best-fit’ model was developed for forecasting the severity of Karnal bunt in the central Punjab thus:

1. \[ DI = -0.8 + 1.5 \text{HTI} \]
2. \[ \text{HTI} = \frac{\text{ERH}}{\text{TMX}} \]

Where DI = Disease index, HTI = Humid Thermal Index, ERH = ‘Evening’ relative humidity recorded at 14.30 hrs (average of the third, fourth, and fifth reproductive weeks), TMX = Maximum temperature (average of third, fourth, and fifth reproductive weeks).

Jhorar et al., (1992) found that the HTI during this part of the growing season varied between 1 and 5; the lowest values occurring in extremely dry and warm conditions, and the highest values representing extremely humid and cold weather, neither of which favour the disease. An HTI of 2.2 - 3.3 during the third and fourth week of the study period favoured the disease. These conditions result from frequent cloudiness and intermittent showers which can be predicted, thus allowing a disease forecast to be made.

In order to predict the likely risk of the pathogen establishing in the PRA area, the 1996 UK PRA applied the HTI used climatic data from individual meteorological stations in the UK and showed that conditions during the ‘heading’ period (broadly speaking May and June) were favourable for infection and disease development, i.e. the majority of calculated HTI values fell within 2.2 and 3.3. Kehlenbeck et al., (1997) also calculated the HTI for the wheat-growing areas of Germany and found that some of the southern areas had HTI values which fell within the optimum range for disease development. Murray and Brennan (1998) also used this methodology for Australia and found that of 122 sites tested within the Australian wheat belt, 67 had HTIs favourable for disease development.

The UK, German and Australian studies were conducted using individual weather station data. To progress this work further, Baker et al., (2000) undertook provisional climatic mapping using interpolated meteorological data (1961-90) with adjustments in the calculation to allow for the lack of mid-afternoon (‘evening’) RH measurements. The results showed that for June, an area covering much of central and southern England had HTIs falling between 2.2 and 3.3 which was therefore considered, using this factor alone, favourable for disease development in wheat crops should they be phenologically susceptible at that time.
The timing of the life-cycle of the pathogen will differ from country to country because the key susceptible growth stage of the wheat crop varies with time between countries. In order to conduct a more detailed assessment as to the extent to which the pathogen could establish itself in the PRA area, it was deemed necessary to compare more accurately the climatic conditions at key stages in the life-cycle of the pathogen and the crop, on a country-by-country basis.

In the current EU Project the potential distribution of Karnal bunt in Europe was predicted using the HTI and two wheat phenology models. This work successfully combined: (a) experimental results on the developmental stages of wheat which are vulnerable to *T. indica* (see 13.2.2 above); (b) bread and durum wheat phenology models to predict the timing of these developmental stages; (c) the potential for *Tilletia indica* infection based on the HTI during this period.

HTI values were obtained for large numbers of weather stations and interpolated at low resolution for Europe and at high resolution for England, Wales, Denmark and Italy (Tuscany, Marche, Puglia and Basilicata). Maps summarising the results were produced with a Geographic Information System.

Based on predictions of the areas where the HTIs fell between 2.2 and 3.3 during the development stages when wheat is vulnerable to infection, there is a very high probability of conditions being suitable for bread wheat infection by *T. indica* in arable areas of western and central Europe. More northerly regions were predicted to have suitable HTI values in about one third of the sowing dates and years studied.

The northern Italian plain and the important Italian pasta regions of Tuscany and Marche are most suitable for durum wheat infection. The durum wheat producing areas of southern Italy, France, Spain and eastern Europe are less suitable, primarily because the vulnerable wheat developmental stages occur during hotter, drier weather conditions.

For both bread and durum wheat, sowing date and the choice of cultivars based upon their phenology (maturity class) had little effect on their potential vulnerability to infection.

Survival studies (see 13.2.1 above) have shown that teliospores can survive for up to at least three years in European soils but possibly longer. An analysis of the number of consecutive years during which the HTI was outside the critical 2.2 – 3.3 range showed that, in northern Europe (Denmark (82%), England and Wales (64%)), significant areas had gaps of at least four years, whereas, overall, only 21% of European arable areas had gaps of over four years. Where wheat is grown in short-rotation or in continuous cropping these gaps are not significant.

This work supports the findings of the first PRA for *T. indica* and shows that the pathogen has the potential to establish in bread and durum wheat growing areas of Europe should it enter.
14. **Could the pest establish in protected environments in the PRA area?**
Crop hosts are only grown under protection for breeding purposes. It is possible for the pathogen to infect and cause disease under protected conditions but the disease should be detected under these circumstances.

15. **How quickly could the pest spread within the PRA area?**
The rate of spread would depend firstly upon the behaviour of the pathogen, the distribution of the hosts and the climate in the local area, secondly on how rapidly any outbreak is detected and reported to the National Plant Protection Organisation, and finally what action is taken as a result. *Tilletia indica* is spread efficiently for both long and short distances through infected or contaminated seed and grain, contaminated farm machinery (etc) as well as through the movement of contaminated soil be it through cultivation practices or as a contaminant on any surface. Local spread can also be through rain and air currents. Animals can carry teliospores as a surface contaminant or through ingestion of teliospores. These survive the digestion process and are spread through contaminated faeces.

Because the pathogen typically causes symptoms on only parts of individual grains this makes it difficult to detect in the first year of any outbreak. Low levels of disease may go undetected. A slow but steady build up of the pathogen could occur in continuous wheat cropping situations if favoured by local climatic factors. Because of the longevity of teliospores, once present in the soil, these could be present for considerably longer than one cropping season and available to infect over a number of years during favourable climatic conditions.

In crops grown commercially for seed, following an initial outbreak, detection should be feasible post-harvest as the harvested seed will be inspected for certification purposes. In crops grown for grain, if the crop is to be managed under recognised Quality Assurance schemes in countries including the UK, infection may be detected post-harvest since the pathogen reduces the quality of grain by the production of malodorous trimethylamine (a rotten fish smell). However, home-saved seed from infected crops may be a route by which spread occurs in the absence of any official inspection or testing.

Murray and Brennan (1998) concluded for Australia that the entry of a small number of teliospores may give rise to a small risk of infection since there is no reported minimum threshold number of spores required for this to occur. Slow spread occurred in Mexico after the disease was first found in 1969-70. Murray and Brennan concluded that outbreaks could occur following the entry of a small number of teliospores which would take several years to reach detectable levels, thus facilitating spread.

Regarding the current situation in Texas, USA, the apparent movement 200km northwards in 2001 within the state (Anon., 2001; EPPO, 2001), may have been due to one of three possible scenarios: either (i) the pathogen was spread beyond the regulated area because of the ineffective 'bunted kernel' inspection policy which would allow teliospores to go undetected in any survey (see 12.) or, (ii) the pathogen was already present in that part of the state and causing low but undetectable levels of disease for some time, or, (iii) the pathogen was already present in that part of the state and causing disease for the first time in that year. However, since the introduction of the USDA APHIS requirement for a 'bunted kernel' to be present before any field is regulated it is not possible to reliably determine the extent to which the pathogen may have spread within Texas or beyond.
In conclusion, national spread within the PRA area from an initial undetected outbreak could be slow but steady.

16. What is the pest’s potential to cause economic, environmental or social impacts in the PRA area?
*Tilletia indica* presents a major economic threat to bread wheat, durum wheat and possibly triticale production and trade in areas with favourable climatic conditions for germination, infection and disease development.

16.1 Crop production and export figures
To illustrate crop production and export value, the most recently available relevant data on production, imports and exports of wheat and triticale (production only) were obtained via the FAOSTAT database collections (FAOSTAT, 2004a and b) and the UK Home Grown Cereals Authority (HGCA, 2004).

Wheat (not broken down into wheat type; i.e., grain or seed, or, bread or durum) and triticale production in 2003 for the fifteen EU Member States and ten EU Accession countries which joined the EU on May 1st 2004 (referred to collectively as the ‘EU region’ in this section) and the USA are presented in Table 1.

Table 1: Wheat and triticale production (tonnes) in descending order of productivity by EU Member State and Accession Countries in 2003 compared to the USA

<table>
<thead>
<tr>
<th>Country*</th>
<th>Wheat Production (tonnes)</th>
<th>Country</th>
<th>Triticale Production (tonnes)</th>
</tr>
</thead>
<tbody>
<tr>
<td>France</td>
<td>30,582,000</td>
<td>Poland</td>
<td>2,811,596</td>
</tr>
<tr>
<td>Germany</td>
<td>19,296,100</td>
<td>Germany</td>
<td>2,508,876</td>
</tr>
<tr>
<td>United Kingdom</td>
<td>14,288,000</td>
<td>France</td>
<td>1,291,000</td>
</tr>
<tr>
<td>Poland</td>
<td>7,858,160</td>
<td>Hungary</td>
<td>278,000</td>
</tr>
<tr>
<td>Spain</td>
<td>6,290,100</td>
<td>Lithuania</td>
<td>214,200</td>
</tr>
<tr>
<td>Italy</td>
<td>6,243,390</td>
<td>Sweden</td>
<td>205,300</td>
</tr>
<tr>
<td>Denmark</td>
<td>4,699,000</td>
<td>Austria</td>
<td>168,637</td>
</tr>
<tr>
<td>Hungary</td>
<td>2,919,000</td>
<td>Czech Republic</td>
<td>165,297</td>
</tr>
<tr>
<td>Czech Republic</td>
<td>2,637,890</td>
<td>Denmark</td>
<td>120,000</td>
</tr>
<tr>
<td>Sweden</td>
<td>2,285,100</td>
<td>Spain</td>
<td>87,600</td>
</tr>
<tr>
<td>Belgium</td>
<td>1,640,364</td>
<td>United Kingdom</td>
<td>61,000</td>
</tr>
<tr>
<td>Greece</td>
<td>1,631,700</td>
<td>Belgium</td>
<td>46,840</td>
</tr>
<tr>
<td>Netherlands</td>
<td>1,228,300</td>
<td>Latvia</td>
<td>40,000</td>
</tr>
<tr>
<td>Lithuania</td>
<td>1,204,100</td>
<td>Netherlands</td>
<td>31,600</td>
</tr>
<tr>
<td>Austria</td>
<td>1,191,380</td>
<td>Slovakia</td>
<td>31,136</td>
</tr>
<tr>
<td>Slovakia</td>
<td>930,363</td>
<td>Luxembourg</td>
<td>19,992</td>
</tr>
<tr>
<td>Ireland</td>
<td>750,000</td>
<td>Portugal</td>
<td>16,000</td>
</tr>
<tr>
<td>Finland</td>
<td>679,000</td>
<td>Estonia</td>
<td>12,900</td>
</tr>
<tr>
<td>Latvia</td>
<td>525,000</td>
<td>Slovenia</td>
<td>5,000</td>
</tr>
<tr>
<td>Portugal</td>
<td>226,000</td>
<td>Italy</td>
<td>0</td>
</tr>
<tr>
<td>Estonia</td>
<td>150,000</td>
<td>Cyprus</td>
<td>-</td>
</tr>
</tbody>
</table>
As an exporter the former EU 15 Member States exported a total of 2,136,493 tonnes of wheat between July 2003 and June 2004; the countries of destination are unavailable (HGCA, 2004). The UK exported a total of 2,200,309 tonnes of wheat between July 2003 and June 2004 to a range of countries in the EU region and to 4 non-EU region countries (Table 2, HGCA 2004). The USA exported a total of 25,608,962 tonnes to the world between July 2000 and June 2001 (FAOSTAT 2004b) (these are the most recent figures), and within the EU region (where data is available) to 13 countries with Italy, Spain, Belgium-Luxembourg and the UK being the recipients of the highest volumes of wheat (Table 2).

Table 2: Exports of UK wheat to individual countries - July 2003 to June 2004 (Source: HGCA, 2004) and exports of US wheat to the current EU Member States – July 2000 to June 2001 (Source: FAOSTAT, 2004b)

<table>
<thead>
<tr>
<th>Destination of UK wheat exports</th>
<th>Tonnes</th>
<th>Destination of US wheat exports within EU Member States</th>
<th>Tonnes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spain</td>
<td>803,603</td>
<td>Italy</td>
<td>1,071,291</td>
</tr>
<tr>
<td>Italy</td>
<td>336,579</td>
<td>Spain</td>
<td>395,188</td>
</tr>
<tr>
<td>Eire</td>
<td>264,493</td>
<td>Belgium-Luxembourg</td>
<td>175,929</td>
</tr>
<tr>
<td>Portugal</td>
<td>230,849</td>
<td>United Kingdom</td>
<td>108,455</td>
</tr>
<tr>
<td>France</td>
<td>218,392</td>
<td>Netherlands</td>
<td>99,243</td>
</tr>
<tr>
<td>Netherlands</td>
<td>160,824</td>
<td>Cyprus</td>
<td>72,222</td>
</tr>
<tr>
<td>Germany</td>
<td>48,353</td>
<td>Malta</td>
<td>38,157</td>
</tr>
<tr>
<td>Greece</td>
<td>40,187</td>
<td>Germany</td>
<td>28,893</td>
</tr>
<tr>
<td>Belgium-Luxembourg</td>
<td>31,052</td>
<td>Portugal</td>
<td>17,771</td>
</tr>
<tr>
<td>South Africa</td>
<td>22,500</td>
<td>Greece</td>
<td>15,905</td>
</tr>
<tr>
<td>Nigeria</td>
<td>22,003</td>
<td>Sweden</td>
<td>2,000</td>
</tr>
<tr>
<td>Eritrea</td>
<td>15,600</td>
<td>France</td>
<td>19</td>
</tr>
<tr>
<td>Faro Islands</td>
<td>3,692</td>
<td>Finland</td>
<td>0</td>
</tr>
<tr>
<td>Finland</td>
<td>2,023</td>
<td>Austria</td>
<td>-</td>
</tr>
<tr>
<td>Denmark</td>
<td>130</td>
<td>Czech Republic</td>
<td>-</td>
</tr>
<tr>
<td>Poland</td>
<td>9</td>
<td>Denmark</td>
<td>-</td>
</tr>
<tr>
<td>Sweden</td>
<td>7</td>
<td>Estonia</td>
<td>-</td>
</tr>
<tr>
<td>Lithuania</td>
<td>7</td>
<td>Hungary</td>
<td>-</td>
</tr>
<tr>
<td>Norway</td>
<td>4</td>
<td>Ireland</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Latvia</td>
<td>-</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>2,200,309</strong></td>
<td><strong>Total</strong></td>
<td><strong>2,025,073</strong></td>
</tr>
</tbody>
</table>

*Accession countries which joined the EU on May 1st 2004 are italicised
- = No data
16.2 Likely impact of *T. indica* based including the summary of results of the EU Fifth Framework Project, ‘Karnal bunt risks’ (Anon., 2004)

In terms of the direct effect that *T. indica* has on wheat, the pathogen is known to affect yield and grain quality as well as seed germination. Reports of the yield and quality effects of *T. indica* were summarised by Warham (1986) who stated that several workers found that the weight of infected grains was directly related to the severity of the disease in the growing crop (with an increase in infection there is a corresponding decrease in grain weight). According to this review, grain yield losses of up to 20% have been reported from a number of infected crops, but generally yield losses are relatively small. However, no precise data on the relationship between the severity or incidence of disease and yield were quoted. The overriding feature of this pathogen is the effect it has on the quality of infected grain, since, according to Warham (1986), when more than 3% of grains are affected the grain is no longer accepted for processing and is declared unfit for human consumption. The National Association of British and Irish Millers (NABIM) would most likely reject any grain found to be affected by Karnal bunt irrespective of the percentage affected, since flour made from infected grain is discoloured, baking quality is impaired and palatability is reduced due to the fishy odour of trimethylamine which the fungus produces. European bread and durum wheat cultivars have not been bred for resistance to *T. indica* and are now known to be susceptible (Porta-Puglia et al., 2003 and 13.2.2 above). It could be that under climatic conditions which favour disease development, yield and quality losses could be significant. *T. indica* is also known to affect seed viability. Affected grains can produce abnormal or non-vigorous seedlings. Seed crops are therefore directly at risk from this pathogen but infected or contaminated seed would not be suitable for planting, because this would introduce the fungus to new areas or perpetuate it in affected areas.

The first UK PRA for this pathogen in 1996 made no specific calculations of the likely economic effect of the introduction of *T. indica* into the UK since the effect on UK-grown cultivars of wheat (which have not been bred for resistance to *T. indica*) or indeed cultivars grown in other countries in the PRA area was unknown. In addition there was no consensus regarding the relationship between disease incidence, severity and yield loss. It was assumed, based upon the available literature, that if *T. indica* were introduced there would be some loss in yield, greater loss in quality and a direct reduction in the germination potential of seed crops. The potential loss of export markets arising as a result of the pathogen’s quarantine status in countries where *T. indica* does not occur, represented a substantial cost as did the costs of disease control and of seed certification.

Subsequent to the first UK PRA, Kehlenbeck et al., (1997) repeated the PRA exercise for Germany. Again it proved difficult to estimate the true losses, given the array of data in the literature. However, considering only the direct losses in yield, given a conservative estimate of loss of 0.5% across the whole of the wheat production area of Germany, the annual losses would amount to DM 15 million (US $8.3 million based upon the exchange rate at the time of publication). For the ‘best case’ scenario the losses would have been DM 5 million (US $2.8 million). (Euros were not national currency at the time of this publication).
Australia has also assessed the economic implications associated with the introduction of *T. indica* (Murray *et al.*, 1996). The estimated overall costs of Karnal bunt being found in 'a region' in Australia amounted to Aus. $55 per tonne (US $36 based upon the exchange rate at the time of publication). The smallest element of this was yield losses at Aus. <$1 per tonne, but losses from quality amounted to 20% of the milling wheat price, based on downgrading to stock feed, estimated at Aus. $14 per tonne. The total overall losses per tonne amounted to Aus. $55 of which Aus. $26 resulted from loss of export sales for uninfected grain. Such a loss seems disproportionate, but will of course depend upon the attitude of the trading partners.

It is now known that UK and European wheat cultivars are susceptible to infection and support disease development under experimental conditions. It can be assumed that where the disease occurs there will be some loss in yield of harvestable grain as well as a definite loss in quality. Under UK and other European Quality Assurance schemes this will result in downgrading of wheat destined for human consumption to feed wheat, particularly as the practice of blending-out a contaminant such as *T. indica* is unacceptable to the National Association of British and Irish Millers (NABIM, A. Waugh, undated personal communication). This will lead to a direct loss in value.

A detailed analysis of the socio-economic impact that this pathogen could have if an outbreak occurred in the PRA area was undertaken within the current EU Project (Brennan *et al.*, 2004, *unpublished report*) and provisional findings (which have been subsequently updated) were published in 2004 (see Brennan *et al.*, 2004a and Thorne *et al.*, 2004).

To assess the likely impacts, an examination was made of the policies and arrangements that are in place in countries where Karnal bunt occurs. The cost components associated with a Karnal bunt outbreak and occurrence were identified, and the cost components were classified as:

- Direct costs;
- Reaction costs;
- Control costs.

The direct costs are the yield and quality losses in crops affected with Karnal bunt.

In addition to the specific direct costs associated with a Karnal bunt outbreak, there are also reaction costs which must be taken into consideration. These costs arise as a result of the market reacting to the fact that *T. indica* has been detected in a particular region (including the categorisation of the pathogen as a quarantine pest in countries where it does not occur). These reaction costs include indirect quality losses, loss of exports and seed industry costs.

In addition to direct and reaction costs there could also be control costs associated with an outbreak of Karnal bunt. These are costs associated with any efforts that occur in an attempt to control and/or eradicate the pathogen. The specific control costs considered for this analysis include the containment costs, eradication costs
and surveillance and testing costs based upon the management of two different sized outbreak scenarios.

The main scenario assessed in this study is a ‘large’ outbreak in a region in the UK initially affecting 50,000 ha of wheat. As a contrast, a smaller outbreak in a region in the UK affecting 1,000 ha of wheat was also assessed. In both cases the scenario assumes a policy of implementing measures aimed firstly at containment and ultimately at eradication of *T. indica*. In each case, the outbreak is detected in mid-harvest, with the pathogen being found in grain being delivered to a silo. This means that in the first year, the only impact is on the harvesting, processing and storage of the existing grain. In subsequent years, affected farmers would have to leave affected fields as bare fallow or they could be grassed-down for a minimum of 5 years with a buffer zone of 3 km around the affected fields of non-susceptible hosts. Fields left bare fallow would have to have herbicides applied to them to control volunteer cereals and weeds. In the remainder of the affected region, only non-host crops could be grown, so that there would be no wheat grown for at least five years. Thus the costs differ between years both because of the likely levels of the pathogen and because of official controls which will be imposed.

The results of the ‘large outbreak’ scenario indicate that an outbreak affecting 50,000 ha of wheat would have very large economic costs for the affected region. The disruption to production, the inability to export wheat from the region and the wide range of control measures introduced would impose costs of €454 million (approximately £309 million at an exchange rate of Euro 1: £0.68) on the region over a ten-year period. While there would be some economic consequences for those outside the affected region, and even some gain in economic welfare, those consequences are small compared to those within the region. In the first year, the cost to the affected region is equivalent to €159 (£108) per ha of wheat, increasing to €237 (£161) per ha by the following year. At the same time, in the rest of the country and in the rest of the EU there would be small net gains equivalent to less than €1 (£0.68) per ha. The details of the components of costs in the affected region for a large outbreak affecting 50,000 ha in the UK managed according to the scenario are provided in Table 3.

Table 3: Components of Costs in Affected Region, for management of a 50,000 ha Karnal bunt outbreak, Years 1 to 10 (from Brennan *et al.*, 2004)

<table>
<thead>
<tr>
<th>Year</th>
<th>Direct Costs</th>
<th>Reaction Costs</th>
<th>Control Costs</th>
<th>Total Costs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Year 1</td>
<td>(€ m)</td>
<td>1.70</td>
<td>23.88</td>
<td>17.55</td>
</tr>
<tr>
<td>Year 2</td>
<td>(€ m)</td>
<td>0.00</td>
<td>0.00</td>
<td>64.43</td>
</tr>
<tr>
<td>Year 3</td>
<td>(€ m)</td>
<td>0.00</td>
<td>0.00</td>
<td>64.43</td>
</tr>
<tr>
<td>Year 4</td>
<td>(€ m)</td>
<td>0.00</td>
<td>0.00</td>
<td>64.43</td>
</tr>
<tr>
<td>Year 5</td>
<td>(€ m)</td>
<td>0.00</td>
<td>0.00</td>
<td>64.43</td>
</tr>
<tr>
<td>Year 6</td>
<td>(€ m)</td>
<td>0.00</td>
<td>0.00</td>
<td>64.43</td>
</tr>
<tr>
<td>Year 7</td>
<td>(€ m)</td>
<td>0.00</td>
<td>0.00</td>
<td>47.38</td>
</tr>
<tr>
<td>Year 8</td>
<td>(€ m)</td>
<td>0.00</td>
<td>0.00</td>
<td>47.38</td>
</tr>
<tr>
<td>Year 9</td>
<td>(€ m)</td>
<td>0.00</td>
<td>0.00</td>
<td>47.38</td>
</tr>
<tr>
<td>Year 10</td>
<td>(€ m)</td>
<td>0.00</td>
<td>0.00</td>
<td>47.38</td>
</tr>
</tbody>
</table>
Brennan et al., (2004) consider that the direct yield and quality effects of the disease are relatively small, and on their own do not justify substantial control measures being implemented or substantial efforts to exclude the pathogen from the EU. However, the reaction costs, where the market’s response to the outbreak and the presence of the pathogen is reflected, are substantial in Year 1, and would increase markedly if the controls were not put in place. In trying to minimise the direct and reaction costs, and especially in trying to prevent the spread of the pathogen to other parts of the EU, considerable control costs are defensible. Nevertheless, on the basis of these estimates of the costs associated with the baseline scenario, control costs constitute the overwhelming majority of the economic costs borne by the industry within the affected region.

Thus, the producers in the affected region can pay a high price for the controls that are put in place to prevent it spreading elsewhere. The impacts of an outbreak of Karnal bunt are likely to be felt unevenly across the wheat industry and the wider economy. Even within the affected region, there can be large differences in outcomes for individuals under the scenario. Farmers with Karnal bunt on their farms would suffer severe economic losses, particularly if crops and/or harvested grain were destroyed. Farmers within the affected region but not having a crop affected by the pathogen would also suffer considerable economic losses where they are prevented from growing any wheat in the following years. Farmers outside the affected region will not incur significant costs, and may even gain from the outbreak, as long as it does not spread to their own region.

In the alternative scenario of a ‘small’ outbreak, the costs of containment and eradication would be generally proportionally smaller, since most of them are based on the area affected or the amount of grain produced in the affected area. The cost items that are not proportional to the size of the production affected are the surveillance and testing, identification, administration and compliance costs. These costs represent a higher proportion of total costs in the event of a smaller outbreak.

In addition, there are likely to be significant social consequences if there were to be an outbreak of Karnal bunt in the EU. There would be social disruption for the
farmers, particularly (but not only) those with affected crops, as there are likely to be significant impacts on many aspects of their production, including which crop to grow, the seed that can be used, crop management practices, where and how the grain can be marketed (etc.) There would also be social disruption for those involved in supplying inputs and processing the outputs of the grains industry. These social effects are likely to affect the broader community across the region, as multiplier effects occur and quarantine and other control measures are imposed. These impacts could extend beyond the agricultural sector in the event of a major outbreak.

Brennan et al., (2004) conclude that an outbreak of Karnal bunt could have serious impacts in the affected region within the PRA area. If the outbreak were large, and the affected region correspondingly large, the aggregate costs would be very significant. Given the likely response of markets to the outbreak, the costs are likely to be substantial, and considerable efforts are warranted to prevent such an outbreak occurring. The costs of doing nothing have not been evaluated, however, the risk is that T. indica would establish and spread leading to ongoing costs in perpetuity, based upon the longevity of the pathogen especially in areas where wheat is grown in short-rotation or is continuously cropped.

STAGE 3: PEST RISK MANAGEMENT

17. What are the prospects for continued exclusion from the PRA area?
Exclusion has worked well to date. The outcome of the first UK/EU PRA was that T. indica became listed as a I/A1 quarantine pest by the EC. The measures to be taken against the organism are minimal and appropriate to the level of risk associated with importing contaminated/infected grain or seed from countries where the organism is known to occur. Seed of wheat, rye (now not considered to be a natural host) and triticale is acceptable for import provided it can be shown that the commodity originates from an area where T. indica is proven not to occur. With respect to grain, the EC requirement for imported material is either area freedom based upon the absence of the pathogen, or, place of production freedom based on visual crop inspection for symptoms of T. indica during the growing season and testing of the grain for T. indica at harvest and testing for T. indica again before export. These measures became incorporated into EC legislation (Anon., 2000, as amended) and into the domestic legislation of the EU Member States in 1997. Despite these requirements, several interceptions of T. indica have been reported by European countries. It should therefore be assumed that there is potential for the pathogen to enter the PRA area from any country where it occurs.

The UK Plant Health Service (PHS) has a target to sample and test all consignments of wheat (and triticale) grain and seed which have arisen in countries where T. indica occurs. This resulted in the identification of T. indica in two consignments of grain from India (one in 2003 and one in 2004). These were managed post-entry according to the draft contingency plan. The UK PHS is aware of all consignments arriving directly in UK ports from countries where T. indica occurs. There may be problems however in identifying consignments which have arrived in another EU Member State before being transported onwards to the UK, since these will have been cleared for movement within the EU by the official body in the country in which these first arrive.

Regarding the risk of entry of T. indica from imports of wheat originating in the USA, the USDA APHIS ‘bunted kernel’ policy, if applied to exports, may allow low levels of infection
to go undetected and for teliospores of the pathogen to be present in grain shipments derived from infested crops originating in the USA. This method of regulating areas does not meet with the EC phytosanitary requirement for freedom from the pathogen *T. indica*; all it seeks to guarantee is freedom from symptoms of Karnal bunt. US wheat seed for sowing is still tested for the presence of the pathogen itself prior to export.

The potential for continued exclusion is possible, but will rely on the exporting countries where *T. indica* occurs complying with the current UK/EC legislation, and on early detection of the pathogen on imported contaminated or infected material. Increasing the number of inspections and testing of grain and seed imported from countries where *T. indica* occurs will help exclude the pathogen from the PRA area. The EU *Tilletia indica* Diagnostic Protocol (Inman *et al.*, 2003) is in current use and could be deployed in further helping to exclude the entry of *T. indica* to the PRA area whenever import inspections are increased.

### 18. If the pest enters or has entered the PRA area what are the prospects of eradication?

In theory, poor. There has been no clear, published evidence from those countries in which the pathogen is established that an eradication policy has succeeded based upon crop rotation, chemical control, host plant resistance, seed certification or other quarantine measures. However, the options for attempting eradication are outlined briefly below.

### 19. What management options are available?

Management options have been reviewed within the EU Project and are not described in detail here. The draft contingency plan for *T. indica* broadly outlines the available options for controlling the pathogen when intercepted on imported grain or seed (processing under official control, or safe disposal, or re-export); as well as when outbreaks occur as part of a containment and eradication plan.

In general terms, eradication of a highly localised outbreak may be possible (although probably quite difficult), if detected in the first year of infection, through timely application of quarantine controls once trace-back and trace-forward activities to determine the extent of the problem have been completed. Measures would include decontamination of equipment, safe destruction of infected seed lots and grain, fumigation, long-term fallowing or grassing-down of infected land, and a prohibition on growing susceptible crops in the affected fields and in a buffer zone around the affected fields. If the pathogen was introduced over a wide area, eradication would be unlikely to be either feasible or cost-effective. Containment aimed at the eventual demise of the pathogen in the absence of its host (not guaranteed) would be the most likely option required for delimited outbreaks. Affected fields identified by trace back and trace forward activities would be managed by keeping them under bare fallow or grassing-down. Measures would have to be implemented for at least 5 years if not longer because of the longevity of the pathogen in the soil. The period of implementation would have to be determined by surveys and testing for the pathogen within the affected fields and in a 3km buffer zone delimiting the area, as well as in susceptible crops grown around the outside of the buffer zone, until such time as the affected fields could be declared pathogen-free. National surveys would be implemented to identify pest-free areas and pest-free places of production to facilitate exports of grain and seed to countries which categorise *T. indica* as a quarantine pest.

If a decision was taken to ‘live with the pest’ rather than to manage it as a quarantine organism, control would still be difficult because of the longevity of its teliospores and the
fact that it can be carried both as a surface contaminant of seed and as inoculum within the seed. Unlike other bunts and smuts of wheat it is difficult to control by seed treatments as it does not infect at the seedling stage (this is discussed under 19.3 below). However, several options for disease control are available, which, if used in an integrated manner, might reduce the incidence and severity of the disease as outlined below:

19.1 The development of resistant cultivars
Extensive research has been carried-out to breed resistant cultivars of wheat, but only in countries outside of the PRA area. This work has occurred mainly in Mexico at CIMMYT, (the International Maize and Wheat Improvement Centre), as well as in India. The use or development of resistant cultivars is not likely to be a practical option for management of outbreaks of T. indica in the PRA area. This strategy is more likely to be considered long-term by plant breeders, should the disease establish in the area, after taking into account other agronomic and market requirements.

Unpublished results from the EU Project indicate that European winter, spring and durum wheat cultivars have a range of susceptibilities that is similar to cultivars grown in Karnal bunt affected countries (Porta-Puglia et al., 2003; Anon., 2004). However, for all three types of wheat there was at least one cultivar tested that showed a very low susceptibility rating. The resistance mechanisms for these could be investigated further if necessary for long-term control strategies for T. indica.

Similar findings have been made in other unpublished studies for European winter wheat cultivars (A. Inman, CSL, UK, 2004, and G. Peterson, USDA ARS, 2004; personal communications). European wheat cultivars with resistance to T. indica have not been developed as the pathogen does not occur here.

19.2 Use of pathogen-free seed
The use of pathogen-free seed is especially important in a seed multiplication programme. Many seed testing methods have been developed for T. indica. EPPO (1997) advise that seeds from countries where Karnal bunt occurs should be tested. They advise that crops for seed should be inspected during the growing season between heading and harvest with any bunted seeds being sampled for laboratory analysis. EPPO advise that direct visual inspection of dry seed is insufficient for quarantine purposes since low levels of infection might pass undetected. Recently an EU Diagnostic Protocol for the detection and identification of T. indica teliospores in seed (or grain) has been developed (Inman et al., 2003) which is to be adopted by EPPO (EPPO, 2004, in press) and could be used as part of a seed testing programme.

19.3 Seed treatment
According to Agarwal et al., (1993), numerous chemical compounds have activity against this pathogen acting by reducing the germination of teliospores. However, EPPO (1997) suggest that chemical seed treatments have proved ineffective in killing teliospores with the exception of mercurial compounds which are banned in most countries. Seed treatment does little to eliminate soil-borne inoculum. Karnal bunt is difficult to control chemically. Teliospores resist physical and chemical treatments and this is likely to contribute to spread of the pathogen (Smilanick et al., 1985, 1988; Rivera-Castaneda et al., 2001). Treatments such as hot water or sodium hypochlorite can be used to disinfest seed but result in seed destruction
Fungicidal seed treatments can have efficacy against surface contamination by *T. indica* but teliospores below the pericarp may resist treatment (e.g. Aujla *et al.*, 1989; Bryson *et al.*, 2002). Because teliospores of *T. indica* can be a contaminant of seed and soil, and because infection does not occur at the seedling stage but at ‘heading’, seed treatment alone is likely to be ineffective.

### 19.4 Foliar sprays

Sporidia released from germinating teliospores land on leaf surfaces and can germinate to produce epiphytic colonies which go on to produce further airborne sporidia capable of infecting florets. Agarwal *et al.*, (1993) state that a range of fungicides give effective control of sporidia on wheat foliage if applied at the ‘early heading’ stage. These include mancozeb, carbendazim, fentin hydroxide, bitertanol and propiconazole. Unpublished findings from the current EU Research Project (Anon., 2004) show that some active ingredients, particularly azoxystrobin, as well as (e.g.) propiconazole are effective both pre- and post-infection (by *T. indica*) when applied as a single spray treatment at GS39 (flag leaf ligule just visible), 49 (first awns visible), 65 (mid-anthesis) or 71 (caryopsis watery ripe).

### CONCLUSION OF THE PEST RISK ANALYSIS

*Tilletia indica* is a I/A1 pathogen for the EC (since 1997) and an A1 pest for EPPO. The pathogen has the potential to enter, establish and spread in the PRA area and to cause damage. The most likely means of introduction to the UK/EU EPPO region would be via infected and/or contaminated wheat seed as well as through grain.

The main route (in terms of volume alone imported from affected countries) for wheat entering the PRA area from countries where the pathogen occurs is from the USA. However, interceptions of *T. indica* have occurred in the PRA area (including the UK) on wheat exported from India, Mexico and possibly the USA. Based upon one years data, Italy seems to be a major importer of wheat from the USA and depending upon the area from which this wheat originates, this country may be at significant risk from *T. indica*.

The pathogen can be detected in seed and grain, but inadequate sampling procedures or only looking for symptoms of the disease and not the pathogen itself in the country of export, will not detect low levels of the pathogen.

The current USDA policy of regulating areas for wheat grain production based upon a voluntary survey and looking for the presence of a ‘bunted kernel’, rather than testing for the pathogen itself, does not comply with the EC phytosanitary requirement for freedom from *T. indica* in grain destined for export to EU Member States. Unless supported by other measures applied specifically for exports, such a policy may allow low levels of the pathogen to go undetected in grain harvested from infested crops and is likely to have increased the risk of entry of *T. indica* from the USA to the PRA area.

The UK is inspecting and testing all consignments of wheat grain known to have been exported from countries where *T. indica* occurs. The situation in other countries in the PRA area is unknown, but if not being undertaken, an increase in inspections and testing of grain imported from countries where the pathogen occurs will help prevent entry of *T. indica* to the PRA area.
The final research findings from the current EU Project ‘Karnal bunt risks’ (Anon., 2004) have been used in this fourth revision of the UK PRA. The Project has demonstrated that teliospores of *T. indica* can survive for significant periods (at least 3 years) in European soils, that some teliospores will be available and capable of germinating to produce infective sporidia during the known window of infection in the now known to be susceptible European wheat crops and that suitable environmental conditions exist for infection and disease development in the PRA area. The results of this work continue to support the view that the pathogen has the potential to establish in the PRA area. The pathogen has the potential to cause small losses in wheat yield and more significant losses in quality, making it unfit for human consumption. The downgrading of infected wheat intended for human consumption to feed wheat would result in a loss of value to the farmer. Because of the quarantine status of *T. indica* in many countries around the world, the potential for loss of export markets remains and the Project has shown that this is a significant element in the impact that the pathogen would have if it was detected in crops grown in the PRA area. The Project has concluded that considerable effort is warranted to prevent the pathogen entering the PRA area; or if it enters, in implementing measures aimed at preventing further spread.

Management options for interceptions and potential outbreaks include several strategies which could be developed within the draft contingency plan to reduce damage. However, the efficacy of these measures under European conditions has not been fully-assessed. A key factor in the success of outbreak management is their early detection. This could only be improved by stratified sampling and testing of grain harvested in the PRA area. Eradication of the pathogen, once introduced, is considered to be potentially difficult to achieve given the longevity of the teliospores of the fungus. The most likely strategy to be adopted in the PRA area following an outbreak would be one of containment. However, by adopting a policy which avoids planting susceptible hosts in the affected area it is possible, but not guaranteed, that the pathogen may eventually disappear i.e. eradication may be deemed to have occurred.

In conclusion, there remains a risk of entry, establishment and socio-economic impact for the UK, EU and EPPO region justifying the existing minimal requirements in EC and domestic legislation. The pathogen should remain listed as a I/A1 pest for the EC and in the A1 category for EPPO.

As of 1 January 2005, implementation of a further amendment to the EC Plant Health Directive will require that specified Additional Declarations (ADs) be stated on all Phytosanitary Certificates (PCs). This will mean that ADs on all PCs accompanying seed or grain of wheat, rye or triticale from countries where *T. indica* occurs will need to be completed to show freedom from *T. indica*. Rye is no longer considered to be a host and can be deleted from UK and EC legislation. To reduce the risk of entry still further, it is suggested that a proposal that EC and domestic legislation in Member States be amended, with more precise requirements for exporting countries where the pathogen occurs to determine pest-free areas and pest-free places of production for exports of wheat and triticale, be considered. Should an outbreak occur in the UK long-term measures aimed at containment and eradication have significant cost implications for the farmers whose land is affected.
**Acknowledgement**

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**UNCERTAINTIES AND FURTHER WORK**

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<tr>
<th>Section of PRA</th>
<th>Major uncertainties</th>
<th>Work required to improve the PRA</th>
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<tbody>
<tr>
<td>Taxonomy</td>
<td>None.</td>
<td>None.</td>
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<tr>
<td>Pathway</td>
<td>Infected or contaminated seed or grain are the main routes through which the pathogen could enter the PRA area. Seed is a direct route of entry. Grain transported through the cereal producing areas of the PRA area also has the potential to disseminate teliospores of the fungus. Export data from countries where <em>T. indica</em> occurs is needed; currently FAOSTAT has been used and it is thought that small quantities from India (for</td>
<td>A study of the routes taken within the PRA area by consignments from countries where the pathogen occurs would improve the pathway analysis. More information on export trade from countries where the pathogen occurs would improve this aspect of the PRA.</td>
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<tr>
<td><strong>Distribution</strong></td>
<td>There may be other countries where the pathogen occurs, but is not yet discovered or reported.</td>
<td>Only the provision of survey data could improve the PRA.</td>
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<tr>
<td><strong>Hosts</strong></td>
<td>None.</td>
<td>None.</td>
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<tr>
<td><strong>Establishment</strong></td>
<td>Data are experimental only. There is still possible uncertainty with respect to sporidial production and dispersal within EU crops (not studied). One of only a handful of models has been used to aid in the prediction of establishment but other published models have been evaluated by partners in the EU Project and found unsuitable for this purpose.</td>
<td>There is always scope for further investigation but while the pathogen is a quarantine listed organism investigation in the field without the use of quarantine containment is impossible. There is always scope for further investigation but while the pathogen is a quarantine listed organism investigation in the field without the use of quarantine containment is impossible. One of the Project partners is developing a germination model which may assist further in the prediction of the risk of establishment.</td>
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<tr>
<td><strong>Spread</strong></td>
<td>Rate of spread if moved by air currents or in trade. More information is needed from countries where <em>T. indica</em> occurs. This is likely to be anecdotal however and all that can be assumed is that teliospores can move both in air currents and in trade. The rate of spread is difficult to determine.</td>
<td>Evaluation of the economic consequences of not taking action for interceptions and outbreaks could be undertaken.</td>
</tr>
<tr>
<td><strong>Impact</strong></td>
<td>The economic consequences of not taking action for interceptions and outbreaks has not been evaluated.</td>
<td>Evaluation of the economic consequences of not taking action for interceptions and outbreaks could be undertaken.</td>
</tr>
<tr>
<td><strong>Management</strong></td>
<td>The efficacy of some of the proposed measures in the draft contingency plan for controlling <em>T. indica</em> (should outbreaks occur) is not known.</td>
<td>As part of the draft contingency plan, monitoring the efficacy of the measures is proposed and this includes pathogen monitoring for at least 5 years within the affected areas and in a buffer zone and in any susceptible crops grown within a specified distance around the buffer zone, until pathogen-freedom is confirmed.</td>
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SELECTED REFERENCES


