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LETTER TO THE EDITOR

The new Pest Risk Analysis for *Tilletia indica*, the cause of Karnal bunt of wheat, continues to support the quarantine status of the pathogen in Europe

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Tilletia indica, the fungus that causes Karnal bunt of wheat, is listed as a I/AI quarantine pest for the European Union (EU) (Anon., 2000a). This means that it is considered absent from the EU, is potentially damaging and so its entry into the EU is banned. This listing was the result of a Pest Risk Analysis (PRA) (Sansford, 1996, 1998), which considered the potential for the pathogen to enter, establish and cause unacceptable impacts in the UK/EU following the first reports of the disease in the USA in 1996 (Ykema *et al*., 1996). This PRA was updated in 2004 (Sansford, 2004) and recently fully revised (Sansford *et al*., 2006) for the EU. This new EU-PRA continues to support the view that *T. indica* has the potential to enter, establish and cause unacceptable economic impacts throughout much of the wheat-growing area of the EU.

Two recent papers (Jones, 2007a,b) have challenged these tenets and the new EU-PRA.

In this letter, we summarise the work of a 4-year collaborative study and address the key points made by Jones (2007a,b) and refute his conclusions, particularly regarding (i) past opportunities for entry to Europe; (ii) the climatic requirements for the completion of the lifecycle of *T. indica*, leading to the disease Karnal bunt; (iii) inoculum thresholds; (iv) the potential economic damage caused by the disease in Europe; and (v) the cost of control. The full findings of the study can be accessed online (http://karnalpublic.pestrisk.net/) together with published accounts of teliospore survival and germination in Europe (Inman *et al*., 2008) and susceptibility of European wheat cultivars to *T. indica* (Riccioni *et al*., 2008).

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The pathogen and the disease

Tilletia indica is a floret-infecting smut pathogen of wheat (*Triticum aestivum* and *T. durum*) and triticale (×*Triticosecale*) causing the disease Karnal or partial bunt. Records on triticale are rare. The pathogen has not been reported in Europe.

The full life cycle of the pathogen is reviewed in Carris *et al*. (2006). Key aspects of the lifecycle, which are relevant to the EU-PRA, include the following:

- In areas where *T. indica* is established, the pathogen survives as teliospores in the soil for up to 5 years (Agarwal *et al*., 1993).
- Fresh teliospores typically have a period of dormancy before they will germinate; only those on or very near to the soil surface break dormancy (Nagarajan *et al*., 1997).
- Germination of teliospores leads to an epiphytic phase involving the cycling of sporidia.
- Infective sporidia deposited on the flag leaf of wheat and washed into the boot cavity, or deposited directly on the emerging ear of wheat plants at the susceptible stage for infection, can under suitable climatic conditions infect the developing grain through the glumes. Warham (1986) indicated that low (moderate) temperatures and high humidity are necessary for infection to occur, while dry weather, high temperatures and bright sunshine are unfavourable. This is supported by studies in India (e.g. Nagarajan *et al*., 1997; Singh *et al*., 2003; Duhan *et al*., 2004; Kaur *et al*., 2005).
- Successful infection of wheat leads to production of teliospores which are restricted to the pericarp layers of the seed (Goates, 1988).

Infected seeds are usually only partially colonized and, within a wheat ear, not all grains necessarily become

infected; hence the alternative disease name of partial bunt. At harvest, teliospores of *T. indica* are usually dispersed locally from bunted grain by the mechanical action of harvesting. Long-distance dispersal occurs through the movement in trade of infected or contaminated seed for sowing or grain for consumption/processing.

Entry pathways and Pest Risk Analyses

The finding of *T. indica*–infected crops in the USA in 1996 (Ykema *et al*., 1996) established a significant new origin on an existing trade pathway for entry into Europe. Wheat had been imported into Europe from the USA before 1996, but in the absence of the pathogen in the USA, there appeared to be no risk of entry along this pathway. Since 1996, there have been several reported or suspected interceptions of *T. indica* on wheat imported from India into Poland and the UK, from Mexico into Italy and from the USA into Greece (Sansford *et al*., 2006).

The establishment of the significant new trade pathway for *T. indica* from the USA into Europe initiated a UK PRA (Sansford, 1996, 1998). The UK-PRA findings suggested that the pathogen could enter, establish and cause economic damage in the UK/EU. As a result, *T. indica* was added as a quarantine pest to the European Commission (EC) Plant Health Directive 77/93/EEC (now 2000/29/EC) in 1997 (Anon., 2000a). It was the first fungal pathogen and the only pathogen of cereals to have been added to the list since the Directive came into force. The EC Directive applies minimal quarantine requirements to seed and grain of *Triticum*, *Secale* and ×*Triticosecale* from countries where *T. indica* is known to occur (currently listed in the EC Directive as Afghanistan, India, Iran, Iraq, Mexico, Nepal, Pakistan, South Africa and the USA). The EC requirements are for area freedom for exports of seed from countries where *T. indica* occurs and area or place of production freedom for grain, the latter being based upon crop inspection and sampling and testing of grain for *T. indica* at harvest and pre-export. Rye (*Secale cereale*) is to be considered for deletion from the EC Plant Health Directive because it is no longer considered to be a natural host.

Because data were not available on the behaviour of the pathogen in the UK/European Union (EU), such as teliospore survival, dormancy, germination and host susceptibility, the earlier UK-PRAs (Sansford, 1996, 1998) formulated conclusions from the existing biological information in the available literature.

Subsequently, there has been much international debate as to whether *T. indica* poses a risk to wheat production and whether it should be listed as a quarantine organism by any country or Regional Plant Protection Organisation (e.g. Malik & Mathre, 1998). Some (e.g. Rush *et al*., 2005) contend that *T. indica* does not have significant effects on yield or quality and that its main effect is in the loss of export markets for countries where it occurs. In addition some scientists think *T. indica* has low invasive potential (e.g. Garrett & Bowden, 2002). If these opinions were correct *T. indica* might not be considered to fulfil the definition of a quarantine organism and in such circumstances would not be regulated.

The EU-PRA for *T. indica*

The prediction of the likelihood of *T. indica* completing its life cycle and establishing in wheat crops in the EU, as determined in the EU-PRA (Sansford *et al*., 2006), depends on four stages:

- A suitable pathway between origin and destination.
- The presence of susceptible host crops at the destination.
- The ability of the pathogen to survive between crops.
- The ability of the over-wintering phase of the pathogen (teliospores) to produce infective sporidia at the vulnerable growth stages for infection and under appropriate conditions to infect and cause disease.

For the first stage, records of cereal imports from countries where *T. indica* occurs were examined, together with evidence of interceptions of *T. indica* over the past 10 years. The other three stages were investigated by experimental work and a review of the literature. Together, and through a multidisciplinary approach, an EU-PRA was produced (Sansford *et al*., 2006).

The risk of establishment of *T. indica* in the wheat-growing areas of the EU has been estimated and mapped (Ewert *et al*., 2002; Baker *et al*., 2004, 2005) by combining crop phenology models (Porter *et al*., 2002) for bread and durum wheat with a disease model, the Humid Thermal Index (HTI) (Jhorar *et al*., 1992). The outputs were interpreted in light of the results of studies on teliospore survival and germination under European conditions (Peterson *et al*., 2006; Inman *et al*., 2008) and an evaluation of the susceptibility of European wheat cultivars (Riccioni *et al*., 2008).

In addition, the potential socio-economic impact of *T. indica* in the EU arising from a small and a large outbreak scenario in a wheat-growing area of the UK was determined (Brennan *et al*., 2004a,b).

The EU-PRA accounted for the results of these studies and concluded that *T. indica* should continue to be listed as a I/AI quarantine pest for the EU.

A recent challenge to the EU-PRA

Two recent papers (Jones, 2007a,b) have challenged the findings of the EU-PRA. In the first the author suggests that there is a low risk of establishment of *T. indica* for Europe, contrary to the view of the EU-PRA. Furthermore, Jones (2007b) concludes that *T. indica* does not warrant the status of a quarantine pest for Europe and considers that there should be "A reappraisal of the quarantine significance of *T. indica* by world plant health authorities ...". The following points are not a full review of these papers nor do they represent the full scope of the collaborative work undertaken to develop the EU-PRA, but they address the key points made by Jones (2007a,b).

Risk of establishment

Past opportunities for entry – is this evidence valid and is it a strong basis for supporting a low risk of establishment in the EU?

Jones (2007a) surmises that wheat seed contaminated with teliospores of *T. indica* has been sent into Europe over many years and therefore in the absence of any outbreaks of Karnal bunt there is a low risk of establishment. Jones (2007a) takes this as evidence that conditions are unsuitable. However, there are no specific data or evidence supporting the contention that there have been "numerous" opportunities for *T. indica* to enter or spread in Europe. Jones (2007a) presented new information (a personal communication from CIMMYT – the International Maize and Wheat Improvement Centre, Mexico) on shipments of wheat seed from CIMMYT for use in nursery trials in "24 European countries" (naming only England, France, Greece, Italy, Spain and Wales) but with no data from the receiving organisations, or on the health status of the seed, assuming that this seed was contaminated with teliospores. No evidence was given in Jones (2007a) as to whether the receiving organisations planted the seed in the field or the glasshouse. Nevertheless, Jones (2007a) uses this to express a view that the risk of establishment in EU countries is low, rather than high.

Assumptions about climatic requirements

Jones (2007a) proposes that Karnal bunt occurs only where the climate is solely "hot arid" or "semi-arid". This view seems to arise from the Indian usage of 'semi-arid', which is applied in India with annual rainfall of 400–800 mm. For example, Chausaria *et al.* (1991) described wheat production in Ludhiana, a "semi-arid" area of the Punjab, and noted that the average annual rainfall is 700 mm a year, with temperatures in the growing season (November to April) of 20 to 30°C (day) and 6 to 18°C (night). The general meteorological usage of 'semi-arid' refers to areas where there is some moisture stress to plants during the year and so it depends on rainfall distribution relative to evaporation (American Meteorological Society, undated), or more simply to areas with annual rainfall of 250–500 mm (Anon., 2006a).

Areas of India with high incidence of Karnal bunt (Gill *et al.*, 1993; Sharma *et al.*, 2004) have annual rainfall of 500–1000 mm, while the incidence declines as the rainfall falls below 500 mm and seasonal temperatures are higher. An up-to-date review of the disease prevalence and severity in India by state is provided in Sansford *et al.* (2006). Europe has many areas with similar annual rainfall to India (Encarta, undated), although the seasonal pattern of rainfall varies (USDA, undated).

Wheat needs moisture in order to grow, whether this comes from rainfall or irrigation or a combination of both. Parts of India produce wheat under irrigation, with the amount used, as with other wheat-growing areas of the world, varying with the available moisture during the growing season. Jones (2007a) does not mention that in the areas of India where Karnal bunt occurs, wheat is frequently grown in rotation with summer-irrigated crops such as rice and soybeans (G. Murray, unpublished data). Teliospores of the pathogen survive, ungerminated, in these wet soils between wheat crops, facilitating infection in the following wheat crop. Teliospores have also survived in summer-irrigated alfalfa fields in Arizona for at least 4 years (G. Peterson, personal communication). Survival for at least 3 years was recorded in wet European soils (Inman *et al.*, 2008). Thus, rather than surviving just in dry soils, the teliospores also survive in wet soils in a range of locations.

Jones (2007a) states that Karnal bunt is not found in (hot, dry) southern areas of India, but it would not be found in these areas as wheat is not grown there (Anon., 2000b).

Jones (2007a) does not elaborate on the finding in Brazil even though it occurred in an area where the climate was neither hot nor semi-arid. Da Luz *et al.* (1993) reported that *T. indica* was found in two seed lines and one cultivar that were harvested in 1989 from the south of the state of Rio Grande do Sul. According to the USDA (Anon., undated), the states of Parana and Rio Grande do Sul are responsible for most of Brazil's wheat production and these states have a large number of rainfed or irrigated fields. The location of Cruz Alta, Rio Grande do Sul, Brazil, has a ME3 moisture regime according to CIMMYT (Rajaram *et al.*, undated) of high rainfall (> 500 mm of precipitation) with a temperate climate.

Jones (2007b) suggests that in Texas, disease outbreaks are sporadic and unpredictable. This is because the level of disease varies with the favourability of the climate on an annual basis, not because the environment may be marginal for establishment, as the author suggests.

Synchronicity of the host-pathogen relationship

For infection of wheat to occur, the timing of teliospore germination and subsequent development of the infective sporidia must be in synchrony with the phenologically susceptible stage of the crop. Magnus *et al.* (2004) investigated the timing of this susceptible stage for European spring, winter and durum wheat cultivars. They confirmed other reports (e.g. Nagarajan *et al.*, 1997) which suggest that the susceptible infection period is from the 'boot' stage to the end of anthesis, i.e. Zadoks growth stages (GS) (43) – 45 – 61 – (69), depending on the cultivar. Teliospore germination to produce basidiospores (primary sporidia) must occur earlier, perhaps at or before flag leaf emergence (GS 37) for the infective secondary sporidia to be available at the susceptible period. It is potentially possible for teliospores to germinate earlier than this and for the sporidial stage to continue to cycle within the crop canopy. Laboratory work published by Goates (2005) suggests that sporidia might be more robust than previously thought, thus allowing teliospores that germinate ahead of the critical period for infection to produce sporidia which could cycle epiphytically and still be available at the susceptible stage to infect the host.

Use of a disease model and the experimental work

Jones (2007a) is critical of the HTI disease model to predict the likelihood of establishment of *T. indica* in Europe,

and appears to assume (on occasion) that it has been used in isolation for this purpose in developing the current EU-PRA. However, the prediction of the risk of establishment in the EU-PRA was primarily based upon an examination of specific parts of the pathogen's lifecycle in relation to its principal host, wheat. Thus, data were generated on: European wheat cultivar susceptibility (most were susceptible and some highly susceptible) (Riccioni *et al*., 2008); teliospore survival under containment in the field in Italy, Norway and the UK for at least 3 years (recovery and germination rates were 61 and 31%, respectively, in the UK) (Inman *et al*., 2008); and a study of the time at which teliospores located on the soil surface would germinate under simulated European climatic conditions to produce the infective sporidia. This last experiment (Peterson *et al*., 2006) was conducted from the simulated time of wheat sowing to anthesis using weekly mean min/max temperature profiles over 5 years for selected wheat growing regions in Mexico (comparative control), Norway, Hungary, United Kingdom and Italy. The start dates represented winter wheat sowing dates for the European sites and spring wheat (not requiring vernalization) sown in the autumn for a location in Mexico where the disease occurs. Jones (2007a) does not refer to this latter critical study. It was done with one soil type (a sandy clay loam) with one of three of the moisture levels tested (10% w/w, 25% w/w and fluctuating between the two over a 7-day period) being at a level (25% w/w) which was in excess of field capacity (16·9% w/w) for this soil and known to be optimum for germination of teliospores. One soil type was used because preliminary experiments showed that soil type had no effect on the germinability of teliospores. The teliospores that were used were 1-year-old and so a proportion would have lost their dormancy. Even at 25% w/w continuous moisture, which was highly favourable for germination, a proportion of teliospores of *T. indica* remained ungerminated until the critical period for infection. Because such continuously favourable soil moisture conditions are unlikely to occur in wheat growing areas of Europe, an even larger number of teliospores would be expected to remain viable. Indeed, in some years, soil moisture deficits occur in Europe (Anon., 2006b) making early germination of teliospores in a wheat crop even less likely. Many EU regions face problems of water scarcity, making drier soils more likely than wet, and several rely on irrigation for agricultural production (Baldock *et al*., 2000).

The HTI model was combined with crop phenology models for bread wheat (*T. aestivum*) (Porter, 1984) and durum wheat (*T. durum*) (Miglietta, 1989). Crop phenology modelling was deployed in formulating the EU-PRA to identify if overlap occurs between the susceptible phenological stage of wheat and the meteorological conditions, within the range predicted by the HTI, to be suitable for *T. indica* infection and disease development. Full details of the experimental work and results are reported in Porter *et al*. (2002) and Baker *et al*. (2004); a summary of the work is provisionally reported in Ewert *et al*. (2002) and later in Baker *et al*. (2005). Jones (2007a) does not refer to this use of crop phenology models which have helped refine the prediction of the risk of establishment of *T. indica* in Europe.

Jones (2007a) refers to two of the three other disease models: the Geophytopathology Index (Diekmann, 1993) and the Rainfall-Temperature Model (Smiley, 1997), but not the Rainfall Model of Nagarajan *et al*. (1997) that has been used by other authors to predict the risk of establishment of *T. indica*. Murray (2004) reviewed all of the models and found the HTI to be the most suitable for helping to predict the risk of establishment in Europe for the last stage of the life cycle that cannot be investigated under field conditions, i.e. covering the period from sporidial release from germinating teliospores from GS 37 to infection and the start of teliospore formation (production of sporogoneus hyphae) up until GS 75. Murray (2004) found that the Geophytopathology Index and the Rainfall-Temperature Model were invalid, while the Rainfall Model was site-specific. Prior to this, Murray & Brennan (1998), in reviewing the use of all the available models for predicting the risk of establishment of *T. indica* in Australia, had concluded that the use of the HTI as deployed by Sansford (1996, 1998) was the most appropriate for their intended purpose. However, Jones (2007a) does not mention the comparative study of Murray (2004).

The HTI should normally be computed for the time of the year when wheat is between flag leaf emergence (GS 37) through heading/flowering until mid-milk (GS 75). This time will vary with seasonal conditions and to some extent with wheat maturation types and sowing date. In formulating the EU-PRA, the HTI was run from GS 37 (flag leaf just visible) to 65 (flowering half completed) and not, as stated by Jones (2007a), "a few weeks prior to wheat anthesis".

Bread and durum wheat phenology models were used to predict the timing of such stages, based upon climatic data information across wheat cultivation basins in Europe. Models were run for several years (the time period varied with the location) to encompass high climatic inter-annual variability.

With the majority of European wheat cultivars being susceptible (Riccioni *et al*., 2008), the period of susceptibility determined (GS 43–69) (Magnus *et al*., 2004) and used in these predictions fits within and goes slightly beyond the period over which the HTI was run (GS 37–65).

The HTI model assumes that teliospores germinate inside the 'window' required for successful infection of wheat. Jones (2007a) correctly cites Sharma & Nanda (2003) stating that when conditions in India favour early germination of teliospores, such that no infective sporidia are available at the critical period for infection, then the HTI model does not work. However, this only occurred once from 1973 to 1999 (Sharma & Nanda, 2003). Jones (2007a) does not refer to evidence provided by Peterson *et al*. (2006), which showed that, even under high soil moisture conditions favourable for early germination of surface-borne teliospores, some remained available and capable of germination at the critical time for infection under a range of European field temperature conditions.

Consequently, Jones (2007a) overlooks the critical work on the timing of teliospore germination in relation to wheat crop phenology and misrepresents how the HTI was used in determining the risk of establishment of *T. indica* in Europe.

Is there an inoculum threshold for establishment?
Jones (2007a) assumes that there is a threshold number of teliospores needed, below which infection and disease will not occur, but does not present data to support a threshold. There are none in the literature. This assumption has been made by others (Garrett & Bowden, 2002) and has been extensively reviewed (Sansford *et al*., 2006) and tested (Murray & Sansford, 2005) and is not supported by the evidence.

Spread of the pathogen
Jones (2007a) correctly states that there has not been a significant increase in the incidence and distribution of Karnal bunt in the USA in recent years and that the disease has spread very little since it was first reported in 1996. However, this does not mean that teliospores of the pathogen have not been spread. Stein *et al*. (2005) conducted soil sampling in wheat fields in Texas, USA in June 2002 to determine the distribution of teliospores of *T. indica* in both regulated (15 fields) and non-regulated areas (1 field). Within the regulated area teliospores were found in 14 of the fields sampled including fields that had never shown detectable levels of disease. Since 1997, USDA surveys for *T. indica* in wheat grain outside of the regulated areas have been based upon symptomatology of Karnal bunt in small samples, but not by looking for the pathogen *T. indica*. Marshall *et al*. (2003) analysed the USDA/APHIS Port Information Network from 1984 to 2000 to determine likely pathways of introduction into the USA based upon *T. indica*-interception records. All 995 interceptions were made on wheat originating in Mexico and 98·8% were intercepted at land border crossings, mainly in Texas and Arizona and mainly in automobiles and road and railfreight vehicles. They surmise that despite having the opportunity to enter the USA since at least 1984 if not earlier, the first official report of a disease outbreak in the USA in 1996 (Ykema *et al*., 1996) and the recognition of known specific requirements for infection and disease development suggests a long period of "latent survival" between initial arrival and becoming a "thriving, established disease". Rush *et al*. (2005) state that following the first report of *T. indica* in the USA, reference samples of wheat maintained by the Arizona Department of Agriculture were tested for Karnal bunt and teliospores of *T. indica* were found in grain harvested in 1993, indicating that the disease had been in Arizona since at least 1992 (the disease is present in Arizona, California and Texas). In Texas, it was presumed to be spreading northwards (Anon., 2001), although this may also reflect past sowings of contaminated wheat seed in combination with favourable climatic conditions rather than natural spread. Based upon these findings it seems apparent that because of the ability of the pathogen to survive long periods as teliospores before germinating and infecting the wheat crop to produce detectable levels of disease, it is not possible to say where it has spread to within the USA. It is also likely that early establishment in Europe will be highly localized and unlikely to be detected until the pathogen becomes more widespread (Murray & Sansford, 2005).

Although the rate of movement of *T. indica* from northern India where it was first described in 1930 (Mitra, 1931) has been slow, the pathogen has moved to new continents from its apparent origin in Asia. The time between arrival and detection is likely to have been lengthy (Murray & Sansford, 2005). Thus, it was first reported as being detected in Mexico during the 1969–1970 crop season (Fuentes-Dávila, 1996); in Brazil where it has been present since at least 1989 (Da Luz *et al*., 1993); in the USA in March 1996 (Ykema *et al*., 1996); and in South Africa in December 2000 (Crous *et al*., 2001), where despite being subject to eradication it spread to new areas in the country between 2002 and 2004 (Naudé, 2002; Anon., 2004).

Risk of establishment – transparency of uncertainty in the EU Project
Areas of uncertainty in the prediction of the risk of establishment were highlighted in Sansford *et al*. (2006) and in particular the epiphytic stage of the fungus (sporidial production and dispersal) prior to infection of the host. This has never been reproduced in a growing wheat crop under controlled conditions and because of quarantine restrictions has not been attempted in the field.

Determination of quarantine pest status by the process of PRA

Jones (2007b) argues that *T. indica* does not qualify for the status of a quarantine pest either in the EU or the EPPO (European and Mediterranean Plant Protection Organisation) region. This is the opposite view of the EU-PRA and is not the view of either the EC or EPPO, who list it as a I/AI and A1 (absent from the EPPO region and recommended for regulation by member countries) pest, respectively.

The WTO-SPS Agreement (WTO, 1995) requires an evidence-based PRA before any phytosanitary measures are set, such as the listing of *T. indica* as a regulated quarantine pest for the EU and its assignment as a quarantine pest by EPPO. In contrast, Jones (2007b) states (i) "... there is strong evidence to suggest that *T. indica* may not even establish in Europe", and (ii) "... yield losses caused by the pathogen are insignificant and disease levels are rarely high enough to cause serious quality problems". This is followed by "... the sole reason for its designation as an important quarantine pest would seem to lie in the serious implications for trade should the pathogen be detected in an exporting country". The latter is only one of several serious economic effects that are considered in the EU-PRA; however, it is still sufficient justification for defining *T. indica* as a quarantine pest (Sansford *et al*., 2006).

By definition (FAO, 2006) a quarantine pest is, "a pest of potential economic importance to the area endangered

thereby and not yet present there, or present but not widely distributed and being officially controlled". PRA is defined as "the process of evaluating biological or other scientific and economic evidence to determine whether a pest should be regulated and the strength of any phytosanitary measures to be taken against it". Further detail on the PRA process can be found in FAO (2004). We consider that the work of the EU Project provides the requisite scientific and economic evidence that is needed for a PRA and justifies the minimal EC measures that are aimed at preventing, or minimising the risk of entry of *T. indica* to the EU.

As indicated earlier, Jones (2007b) asserts that, "there is strong evidence to suggest that *T. indica* may not even establish in Europe" without providing such evidence or going through the rigorous process of data gathering, data generation and PRA undertaken to produce the comprehensive EU-PRA. Further comments by the author discuss the origin of the quarantine restrictions in the USA but this is not relevant to the assessment of risk to the EU and the EU/EPPO quarantine status of the pathogen which is based upon a scientific assessment of the risk to the EU/EPPO region.

Socio-economic impacts – yield, quality, loss of export markets and costs of control

In developing a PRA according to FAO (2004), consideration needs to be taken of both direct and indirect pest effects in the area potentially at risk. For *T. indica* the direct effects include the effect on yield and quality and the indirect effects include effects on export markets, changes to producer costs or input demands including control costs, changes in consumer demand, feasibility and cost of eradication and containment, resources needed for additional research and advice as well as social and other effects. All of these costs have been considered in the EU-PRA, but not fully by Jones (2007b).

Jones (2007b) assesses the economic consequences of introduction by firstly considering the levels of disease in affected countries. In contrast to Sansford *et al.* (2006) actual levels of disease incidence and severity are reported only briefly and superficially. For example, data from India are the highest reported in any of the affected countries, especially when susceptible wheat cultivars have been grown (Gill *et al.*, 1993; Sharma *et al.*, 2004; Sansford *et al.*, 2006) but are not reported by Jones (2007b).

Yield

Jones (2007b) states that Karnal bunt currently causes very little actual yield loss and only small quality losses in countries where it is endemic. The current yield losses are small in percentage terms but their financial value can be high where they occur across a large wheat production area (Sansford *et al.*, 2006).

Cultivar resistance – a control cost to minimise yield losses

The current yield losses are relatively small in countries where the pathogen is endemic because the growing of less susceptible wheat cultivars provides good control. Jones (2007b) ignores the costs of these controls: selection for resistance is labour intensive and only one generation can be screened each year (Gill *et al.*, 1993). Any addition of the requirement for resistance to *T. indica* in European breeding programmes would be costly and if made a priority, would reduce the rate of progress for the development of other genetic characters.

Jones (2007b) states that yield losses alone would seem insufficient for *T. indica* to warrant quarantine pest status in the EU. Whilst this is so in countries growing less susceptible cultivars, the lack of resistance in current European wheat cultivars (Riccioni *et al.*, 2008) suggests yield losses may be higher than where Karnal bunt is endemic (where breeding programmes are established).

Quality

Quality losses due to discolouration of grain and grain products and the presence of trimethylamine (Warham, 1986) vary according to the level of disease and the market requirements. Jones (2007b) assumes, based upon Warham (1986), that grain quality is affected only if more than 3% of grain is affected by *T. indica*. Observations in India clearly show that when susceptible wheat cultivars are grown in areas that favour disease development, the level of grain infection frequently exceeds 3% and severely affects grain quality (Gill *et al.*, 1993; Sharma *et al.*, 2004; Sansford *et al.*, 2006). Under experimental conditions, susceptible European wheat cultivars developed levels of disease exceeding 3% infection (Riccioni *et al.*, 2008). Furthermore, the quality requirements for wheat in European countries are different to countries such as India, and in Europe there is zero tolerance of bunt affected, or contaminated grain grown under Quality Assurance (QA) schemes such as those in the UK (NABIM, 2005).

Jones (2007b) assumes that grain infected with *T. indica* can be cleaned to remove the pathogen. All grain is cleaned before it is milled. Under European QA schemes, grain which is known to be contaminated with *T. indica* is more likely to be rejected and downgraded to animal feed. The presence of bunted grain and teliospores of *T. indica* in European crops would require phytosanitary measures. This would incur a cost in addition to the loss of income from downgrading milling wheat to animal feed.

Socio-economic impact

A detailed analysis of the socio-economic impact if an outbreak of Karnal bunt occurred in the EU was undertaken to support the EU-PRA (Brennan *et al.*, 2004a,b). Jones (2007b) does not refer to the detailed analysis, presenting only gross overall figures, and does not mention that the costings include the costs of implementing a draft contingency plan to prevent the predicted spread of *T. indica* resulting from a hypothetical outbreak. Furthermore, the author comments on the fact that reaction and control

costs were estimated to account for over 99·5% of the total economic effect of an outbreak in the UK. A breakdown of what is included in those costs is shown below and gives a clearer picture of how these costs were justified. Breeding for resistance is not included in these costs but as outlined above, would add significantly to overall control costs should *T. indica* become established in Europe.

Direct costs include yield losses and downgrading affected milling wheat to feed. Reaction costs include downgrading unaffected milling wheat to feed, price and export effects, seed industry costs and quality assurance costs. Costs associated with control include: surveillance and testing; administration and compliance procedures; income loss from cropping restrictions; yield reduction from growing tolerant cultivars (if permitted); additional fungicide inputs; value of standing crops destroyed; costs of destroying growing crops; value of affected grain destroyed; costs of destroying affected grain; treatment of mill by-products; grain processing costs (heat treatment); livestock industry costs; machinery cleaning costs and facility cleaning costs.

A later economic analysis (F. Thorne, unpublished data; Anon., 2006c), considered a reduction in phytosanitary controls with resulting spread of the pathogen. Consequently the direct and reaction costs increased proportionately. This illustrates the benefit of controls on imports as much as controlling any outbreak of disease. Thus, a single 50 000 ha outbreak with phytosanitary controls was estimated to cost €454 million over 10 years from the time of detection. With less official control being implemented and national spread, the ultimate cost was €548 million. If the spread were across the EU then the cost would be 10 times full control, i.e. €4540 million over 10 years. Thus, control costs are large but may be worthwhile if the controls prevent *T. indica* spreading nationally or across the EU. However, it is more cost-effective to keep the pathogen out, which is the aim of the EC phytosanitary measures.

Jones (2007b) considers that "Reaction and control management costs ... are artificial in that they are a result of the current undeserved international status of the pathogen as a significant quarantine pest". This is not supported by the EU-PRA. The pathogen is a quarantine pest like any other quarantine pest by definition (FAO, 2006); such pests are never classified as either significant or insignificant. The original categorization of *T. indica* as a quarantine pest for Europe was based upon the best data available in 1996 and has been reinforced by the work done to complete the EU-PRA of 2006.

Phytosanitary measures – are there trade implications?

Jones (2007b) considers that the EC Phytosanitary Requirements for *T. indica* place a burden on an exporting country, thereby allowing wheat-producing countries without Karnal bunt a distinct advantage over those that have the disease. However, the EC requirements (which are for *T. indica*, not Karnal bunt) are not prescriptive in terms of the method by which the requirements are met and do not place any more of a burden than is required for any other quarantine pest with EC phytosanitary requirements. Countries with *T. indica* have not been prevented from exporting their wheat to the EU under this EC regime. The existing EC measures are minimal. In parts, Jones (2007b) describes the actions of other countries in defining areas that are free from the disease Karnal bunt, and has not clarified that the EC requirement is for freedom from the pathogen, *T. indica*.

Jones (2007b) comments on restrictions placed upon grain for human consumption in particular as being in need of revision; however, grain carrying teliospores of *T. indica* poses a significant risk since it arrives in larger quantities than seed wheat and teliospores of *T. indica* have the potential to escape into wheat production areas *en route* to its destination. Jones (2007b) comments that '....a general lowering of its quarantine significance to allow grain to be imported more easily into countries with environments where *T. indica* is unlikely to establish would reduce problems associated with trade'. However, there is no evidence of problems with trade into Europe, nor are there data to support a view that there are wheat-growing countries in Europe with environments where *T. indica* is unlikely to establish.

Jones (2007b) also comments that, "... trade cannot be restricted for unimportant issues". This does not reflect the fact that there have been no trade restrictions to date in Europe on wheat or either of the other two listed hosts exported from countries where *T. indica* occurs, nor why it is unimportant.

In conclusion, it is our contention that the EU-PRA for *T. indica* (Sansford *et al*., 2006) continues to support the view that the pathogen has the potential to enter, establish and cause unacceptable impacts in the wheat-growing areas of Europe. This maintains the status of *T. indica* as a quarantine pest for Europe and justifies the minimal phytosanitary requirements that are in place in European countries and which are aimed at preventing entry.

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