Grower Summary

PE 007

Sweet pepper: aspects of the biology and control of Fusarium fruit rot

Annual 2012
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HDC is a division of the Agriculture and Horticulture Development Board.
**Project Number:** PE 007

**Project Title:** Sweet pepper: aspects of the biology and control of Fusarium fruit rot

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**Contractor(s):** ADAS
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University of Warwick, Warwick Crop Centre

**Industry Representative:** Gill Wardell, Abbey View Nurseries

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**Project Cost:** £69,449.00
Headlines

- Isolates of *Fusarium* species obtained from pepper fruit from various UK nurseries were identified as *F. lactis* (predominantly), *F. oxysporum* and *F. proliferatum*.

- Inoculation of sweet pepper flowers with spores of *Fusarium proliferatum* resulted in reduced fruit set and internal fruit rot.

Background and expected deliverables

Internal fruit rot of sweet pepper grown in glasshouses has been an increasing problem worldwide since around 2000. In the UK a survey in 2007 showed infected fruits were present in many crops at levels from 1 to 37% (PC 260). The disease causes some losses on production nurseries but more importantly *Fusarium* continues to be a frequent cause of rejection by packers and complaints by supermarkets. Losses vary greatly between crops and seasons, and growers are generally unaware a problem may be present until harvest or postharvest. The fruit rot arises through infection of flowers by spores of *Fusarium*. Several *Fusarium* species have been associated with the disease, notably *F. lactis* and *F. oxysporum*. Observations in commercial crops indicate the disease is favoured by high humidity and fluctuating temperatures. At present there is no effective method of control. This project aims to reduce losses to *Fusarium* internal fruit rot through increased knowledge of factors associated with a high incidence of the disease and use of biofungicides and fungicides to control flower infection.

Summary of the project and main conclusions

Review of overseas research on pepper *Fusarium* internal fruit rot

*Fusarium* internal fruit rot of sweet peppers has also become a significant problem in recent years in Canada, Belgium and the Netherlands. The fungi associated with the disease are variously reported as *F. lactis*, *F. oxysporum*, *F. proliferatum* and *F. solani* in Alberta, Canada; as *F. subglutinans* in British Columbia, Canada; as *F. oxysporum*, *F. proliferatum*, *F. nygamai* and *F. lactis* in Belgium, and as *F. oxysporum*, *F. proliferatum*, *F. solani* and an unknown species related to *F. lactis* in the Netherlands.

Work in Belgium showed that pepper flowers may reach dew point for several hours during the night and early morning, and it was suggested that condensation of water on petals
makes them more susceptible to infection by *Fusarium* species. It was also reported that varieties with large petals, and varieties that retain petals for longer, were more susceptible. Recent work in Canada confirmed the infection pathway of *F. lactis*. *Fusarium* spores (conidia) deposited on the stigma grew down the style and into the ovary within 5-6 days after inoculation. At 45 days after inoculation, typical internal fruit rot symptoms were observed and *F. lactis* was recovered from fruit tissue and seeds within externally symptomless fruit. It was suggested that *Fusarium* conidia were deposited on the stigma by insect pollinators or from the air.

Further work in Canada, by a different research group working with *F. subglutinans* (subsequently re-identified as *F. lactis*), identified some biological and chemical treatments that significantly reduced Fusarium internal fruit rot when applied to flowers 1 day before inoculation. Effective biological treatments included preparations of *Bacillus subtilis*, *Gliocladium catenulatum* and *Trichoderma harzianum*-T22. The fungicide Rovral (iprodione) also reduced Fusarium fruit rot.

Contact was made in 2011 with researchers in Belgium, Canada and the Netherlands. No new results were identified in Canada.

A research group in Belgium published results in early 2012 on the genetic diversity of *Fusarium lactis* species complex isolates obtained from sweet pepper. Out of 98 isolates obtained from Belgium (82), Canada (1), the Netherlands (9) and the UK (6), 74 were identified by molecular tests as *F. lactis* or *F. lactis*-like, 13 as *F. oxysporum*, nine as *F. proliferatum* and two as *F. solani*. Members of the *F. lactis* species complex showed large genetic and phenotypic diversity. It was suggested that the emergence of Fusarium internal fruit rot over the last 10 years in major sweet pepper growing regions was due to the introduction of new varieties with reduced resistance, and possibly to changes in greenhouse climate control that allowed higher relative humidity.

Work on the disease was done in the Netherlands in 2008-9 by Groen Agro Control, a private consultancy and research organisation, but their report is not in the public domain.
Molecular characterisation of *Fusarium* isolates associated with pepper *Fusarium* internal fruit rot in the UK

Out of six *Fusarium* isolates obtained from UK pepper crops and sent to Belgium for molecular characterisation, two were confirmed as *F. oxysporum*, three (peach-coloured isolates) were identified as *F. lactis*-like (but different from the type-strain), and one was identified as *F. proliferatum*.

Nineteen isolates of *Fusarium* sp. were obtained from fruit on four UK pepper nurseries and compared with reference DNA sequences of *F. andiyazi*, *F. lactis*, *F. oxysporum*, *F. proliferatum*, *F. nygamai* and *F. solani* by molecular tests at the University of Warwick. Eight isolates were found to be closely related to *F. lactis*, six to *F. oxysporum*, three to *F. proliferatum* and two to *F. solani*. An isolate obtained from a pepper stem base lesion on a fifth nursery was identified as *F. solani*.

Effect of Fusarium inoculum and flower age on infection

Two fully replicated experiments were done in commercial crops of sweet pepper in Essex. Flowers were individually inoculated and the stalks tagged so that fruits developing from them could be identified at harvest around 10 weeks later and examined for *Fusarium* internal fruit rot.

In May 2011, five methods of inoculation with an isolate of *F. proliferatum* were compared on fully open white flowers. Only 56 out of 280 inoculated flowers developed into mature fruit; 14 of these were infected with *Fusarium* internally. Inoculation of flowers with *Fusarium* spores by spraying in water, placing a small water droplet in the flower or dry spore transfer using a paintbrush, all resulted in infected fruit. Spray inoculation with a low *Fusarium* spore concentration was most successful (5 out of 8 fruit affected). None of 20 fruit developing from uninoculated flowers, or from flowers inoculated with water only, were affected by *Fusarium*.

In August 2011, a further set of flower inoculation treatments was examined using the same isolate of *F. proliferatum*. More fruit developed from flowers left untreated, inoculated with water only, or mist-inoculated with *Fusarium* spores (20-25 fruit from 60 flowers per treatment) than the other treatments (7-12 fruit from 60 flowers). *Fusarium* internal fruit rot only occurred in fruit that developed from inoculated flowers (Table 1). Inoculation of young
white flowers and old brown flowers both resulted in Fusarium fruit rot, although at a significantly higher incidence from young white flowers (50%) than old brown flowers (19%). The level of infected fruit at harvest (35-56%) varied little with spore concentration or method of applying the spores. Mist inoculation, which resulted in a similar level of fruit set to that of uninoculated flowers, and a level of Fusarium fruit rot similar to that of inoculation with a high spore concentration, will most probably be used in future work to examine varietal susceptibility and some control options.

Eight isolates of *Fusarium* sp. recovered from affected fruit were characterised by molecular tests. Three isolates were identified as *F. proliferatum*, four as *F. lactis* and one as *F. lactis*-like. *F. lactis* may have occurred through natural dual infection; occurrence of *Fusarium* within small aborted fruit from uninoculated flowers supports this explanation. Alternatively, as no internal rot occurred in fruit that developed from untreated flowers, it is possible that the original inoculum consisted of a mixture of *F. lactis* and *F. proliferatum*, rather than a pure culture of *F. proliferatum*.

**Table 1.** Effect of flower inoculation with *Fusarium proliferatum* and flower age on occurrence of Fusarium fruit rot in sweet pepper cv. Ferrari – November 2011 (Experiment 2)

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Total number fruit at harvest$^a$</th>
<th>Occurrence of Fusarium (% of fruit harvested)</th>
<th>External rot</th>
<th>Internal rot</th>
<th>On seed</th>
<th>Any symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Untreated</td>
<td>33</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2. Water control</td>
<td>27</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>3. Mist</td>
<td>26</td>
<td>12</td>
<td>31</td>
<td>19</td>
<td>35</td>
<td>33</td>
</tr>
<tr>
<td>4. Dry spore transfer</td>
<td>18</td>
<td>11</td>
<td>17</td>
<td>33</td>
<td>33</td>
<td>33</td>
</tr>
<tr>
<td>5. Spray – low concentration (5 x 10$^3$/ml)</td>
<td>16</td>
<td>44</td>
<td>56</td>
<td>38</td>
<td>38</td>
<td>56</td>
</tr>
<tr>
<td>6. Spray – medium concentration (5 x 10$^5$/ml)</td>
<td>14</td>
<td>29</td>
<td>43</td>
<td>36</td>
<td>36</td>
<td>50</td>
</tr>
<tr>
<td>7. Spray – old flowers, medium concentration (5 x 10$^5$/ml)</td>
<td>21</td>
<td>14</td>
<td>19</td>
<td>5</td>
<td>5</td>
<td>19</td>
</tr>
</tbody>
</table>

$^a$Fruit were harvested on 19 October and 2 November, 10 weeks after flower inoculation; data shown are for the combined harvests.
Financial benefits

Fusarium internal fruit rot of sweet pepper occurs in many UK sweet pepper crops, the severity varying with variety, nursery, glasshouse and time of year. The disease is more common in the spring and autumn when fruit take longer to ripen. Growers have reported that up to 20% of a day’s pick may be affected. Assuming a farm-gate-value of 50p per fruit and a harvest of [1,000] fruit/ha on a single day, this represents a loss of £100/ha/day. Additional losses arise when infected fruits are not detected at harvest or in the packhouse, but the rot develops subsequently causing supermarket rejection or customer complaint to the supermarket, both of which incur a cost for the grower. The potential financial benefits of this work are an increased proportion of harvested fruit free from *Fusarium* internal infection and reduced risks of packhouse rejection, supermarket complaints and disruption to the supply chain.

Action points for growers

None at present.