Efficacy of systemic fungicides against Sclerotium rolfsii causing chickpea collar rot

AS Rothe, VG Mulekar, PN Kadam, KL Jaiswal and PA Shinde

Abstract
Collar rot of chickpea caused by Sclerotium rolfsii has a major constraint and potential threat to successful chickpea cultivation. Therefore efforts were made to evaluate the different systemic fungicides in vitro condition against Sclerotium rolfsii. Seven systemic fungicides were evaluated (at 500, 1000 and 1500 ppm) in vitro against S. rolfsii by poison food technique. However, 100% mycelial inhibition was recorded with difenconazole 25% EC, hexaconazole 5% EC, propiconazole 25% EC, tebuconazole 25.9 EC. These were followed by pyraclostrobin 20 WG (76.05%), carbendazim 50% WP (56.13%) and the fungicide thiophanate methyl 70% WP was found comparatively less effective with 18.49% per cent inhibition, of the test pathogen.

Keywords: Chickpea, systemic fungicides, poison food technique, Sclerotium rolfsii

Introduction
Chickpea (Cicer arietinum L.) is the world’s third most important pulse crop after dry beans (Phaseolus vulgaris L.) and dry peas (Pisum sativum L.) (Vishwa Dhar and Gurha, 1998) [1]. Chickpea is a vital source of plant derived edible protein in many countries. It has advantages in the management of soil fertility, particularly in dry lands and the semi-arid tropics. Indian subcontinent accounts for 90% of the total world chickpea production (Jalan et al., 2000) [2]. Chickpea is contributing nearly 42 to 47 per cent of total pulse production in India. Nearly 90% of the area and production is from six states viz., Madhya Pradesh, Rajasthan, Maharashtra, Uttar Pradesh, Karnataka and Andhra Pradesh (Arunodhayam et al., 2014) [3]. In India, Chickpea is grown for dal making, culinary and for table purpose. It constitutes the main source of protein and several amino acids therefore, it is useful diet for human being. A legume crop fixes atmospheric nitrogen and thus helps to improve the soil fertility and has significant role in crop rotation. There are many reasons for low yield of chickpea such as lack of appropriate technology, use of local varieties, absence of irrigation facilities, growing of chickpea crop on marginal lands and pests and disease problems etc. Amongst them, diseases play an important role in reducing the yield potential of chickpea. Out of total diseases, collar rot causing a serious loss in chickpea production. Yield losses caused by collar rot of chickpea is 14-74% (Muthusamy and Mariappan, 1999) [4]. Chickpea collar rot is most serious and challenging disease which causes severe yield losses i.e. 60-70% under favourable conditions (Nene, 1985). S. rolfsii is soil borne pathogen and survives in soil for many years (Allce, 1984) [5]. Considering these issues, present study was planned and conducted with the aim to evaluate the different systemic fungicides in vitro condition against Sclerotium rolfsii causing collar rot of chickpea.

Material and Methods
Efficacy of seven systemic fungicides were evaluated under in vitro conditions by applying poison food technique (Nene and Thapliyal, 1993) [6]. The required quantity of respective fungicide was incorporated in 100 ml of PDA in 250 ml flasks. The medium was shaken well to give uniform dispersal of the fungicides. Seven systemic fungicides carbendazim, hexaconazole, difenconazole, propiconazole, tebuconazole, pyraclostrobin and thiophanate methyl, were evaluated at concentration of 500, 1000 and 1500 ppm against S. rolfsii. Twenty ml medium was poured separately into each sterilized Petri plates, replicated three times and centrally inoculated with 5 mm mycelial disc of the pathogen and incubated at 28±2°C for seven days. A suitable control was maintained by growing the pathogen on fungicides free PDA medium. Observation on radial mycelial growth / colony diameter of the S. rolfsii was
recorded at an interval of 24 hours and continued till untreated control plates were fully covered with mycelial growth. Per cent mycelial growth inhibition of the pathogen with the test fungicides over the untreated control was calculated by using the formula (Vincent, 1927) \[ \text{Per cent inhibition} = \frac{C - T}{C} \times 100 \]

Where,
- \( C \): Growth of the test fungus in untreated (control) plates.
- \( T \): Growth of the test fungus in treated plates.

### Results and Discussion

All seven systemic fungicides evaluated \textit{in vitro} (each @ 500, 1000 and 1500 ppm) were found to influence significantly mycelial growth and its corresponding inhibition of \( S. \) \textit{rolfsii}. (Plate I). Further, mycelial growth and its inhibition were found inversely and directly proportional, respectively to concentrations of the fungicides tested.

### Radial mycelial growth

Results (Plate I, Table 1) revealed that the systemic fungicides exhibited a wide range of radial mycelial growth of \( S. \) \textit{rolfsii}, and it was found to be decreased steadily with increase in concentrations of the test fungicides. However, difenconazole 25% EC, hexaconazole 5% EC, propiconazole 25% EC, and tebuconazole 25.9% EC at all three test concentrations of 500, 1000 and 1500 ppm showed none of mycelial growth of the test pathogen. This was followed by pyraclostrobin 20 WG (27.66 mm, 23.9 mm and 13.08 mm), carbendazim 50% WP (59.33 mm, 34.41 mm and 30.66 mm), and thiophanate methyl 70% WP (83.58 mm, 73.33 mm and 63.16 mm), respectively @ 500, 1000 and 1500 ppm, as against maximum mycelial growth (90.00 mm) with untreated control. Average radial mycelial growth recorded with the test systemic fungicides ranged from 0.00 mm to 73.35 mm. However, it was nil with fungicides difenconazole 25% EC, hexaconazole 5% EC, propiconazole 25% EC, hexaconazole 5% EC, propiconazole 25% EC and tebuconazole 25.9% EC. These were followed by pyraclostrobin 20 WG (21.54 mm), carbendazim (41.46 mm) and thiophanate methyl 70% WP (73.35 mm) as against 90.00 mm with untreated control.

### Mycelial growth inhibition

Results (Plate I, Table 1) revealed that the systemic fungicides tested (each @ 500, 1000 and 1500 ppm) significantly inhibited mycelial growth of \( S. \) \textit{rolfsii}, over untreated control and it was directly proportional to concentrations of the test fungicides. However, mycelial growth inhibition was cent per cent (100%) with difenconazole 25% EC, hexaconazole 5% EC, propiconazole 25% EC, and tebuconazole 25.9 EC at all three concentrations of 500, 1000 and 1500 ppm. This was followed by pyraclostrobin 20 WG (69.26%, 73.74% and 85.46%), carbendazim 50% WP (40.7%, 61.76% and 65.93%), and thiophanate methyl 70% WP (71.3%, 18.52% and 29.82%), respectively @ 500, 1000 and 1500 ppm, as against minimum mycelial inhibition (0.00 mm) with untreated control.

### Table 1: \textit{In vitro} efficacy of systemic fungicides against \( S. \) \textit{rolfsii} causing collar rot of chickpea

<table>
<thead>
<tr>
<th>Tr. No</th>
<th>Treatments</th>
<th>Col. Dia.* (mm) at ppm</th>
<th>Av. (mm)</th>
<th>% Inhibition* at ppm</th>
<th>Av. Inhib. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1</td>
<td>Carbendazim 50 WP</td>
<td>59.33 34.41 30.66</td>
<td>41.46</td>
<td>40.71(39.64) 61.76(51.80) 65.93(54.28)</td>
<td>56.13(48.52)</td>
</tr>
<tr>
<td>T2</td>
<td>Difenconazole 25 EC</td>
<td>0.00 0.00 0.00</td>
<td>0.00</td>
<td>100(0.00) 100(0.00) 100(0.00)</td>
<td>100(0.00)</td>
</tr>
<tr>
<td>T3</td>
<td>Hexaconazole 5 EC</td>
<td>0.00 0.00 0.00</td>
<td>0.00</td>
<td>100(0.00) 100(0.00) 100(0.00)</td>
<td>100(0.00)</td>
</tr>
<tr>
<td>T4</td>
<td>Propiconazole 25 EC</td>
<td>0.00 0.00 0.00</td>
<td>0.00</td>
<td>100(0.00) 100(0.00) 100(0.00)</td>
<td>100(0.00)</td>
</tr>
<tr>
<td>T5</td>
<td>Tebuconazole 25.9 EC</td>
<td>0.00 0.00 0.00</td>
<td>0.00</td>
<td>100(0.00) 100(0.00) 100(0.00)</td>
<td>100(0.00)</td>
</tr>
<tr>
<td>T6</td>
<td>Pyraclostrobin 20 WG</td>
<td>27.66 23.9 13.08</td>
<td>21.54</td>
<td>69.26(56.32) 73.44(58.97) 85.46(67.58)</td>
<td>76.05(60.69)</td>
</tr>
<tr>
<td>T7</td>
<td>Thiophanate methyl 70 WP</td>
<td>83.58 73.33 63.16</td>
<td>73.35</td>
<td>07.13(15.48) 18.52(25.48) 29.82(33.09)</td>
<td>18.49(25.46)</td>
</tr>
<tr>
<td>T8</td>
<td>Control (untreated)</td>
<td>90.00 90.00 90.00</td>
<td>90.00</td>
<td>0.00(0.00) 0.00(0.00) 0.00(0.00)</td>
<td>0.00(0.00)</td>
</tr>
</tbody>
</table>

SE ± 0.37 0.27 0.21 - 0.47 0.28 0.33 -
CD (P=0.01) 1.09 0.78 0.63 - 1.40 0.82 0.96 -

*- Mean of three replications, Figures in parentheses are arcsine transformed values

Average mycelial growth inhibition recorded with the systemic fungicides ranged from 18.49 to 100.00 per cent. However, it was significantly highest and cent per cent (100%) with difenconazole 25% EC, hexaconazole 5% EC, propiconazole 25% EC, tebuconazole 25.9 EC, followed by pyraclostrobin 20 WG (76.05%), carbendazim (56.13%) and thiophanate methyl 70% WP (18.49%) as against (0.00) mm with untreated control.
Thus, all of the seven systemic fungicides tested were found fungistatic against *S. rolfsii* and significantly inhibited its mycelial growth, over untreated control. However, fungicides found most effective in their order of merit were difenconazole 25% EC, hexaconazole 5% EC, propiconazole 25% EC, tebuconazole 25.9 EC, pyraclostrobin 20% WG, carbendazim 50% WP, and thiophanate methyl 70% WP. Similar fungistatic effects of systemic fungicides against *S. rolfsii* were reported by Salvi *et al.* (2017) and they revealed that complete inhibition of *S. rolfsii* with hexaconazole, propiconazole and difenconazole. These results are also in conformity to the reports of several earlier workers (Kuldhar and Suryawanshi, 2017; Wavare *et al.*., 2017 and Archana *et al.*., 2018).

References