Yield loss assessment of Sclerotinia stem rot of canola in Iran

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Abstract: Sclerotinia stem rot (SSR), caused by Sclerotinia sclerotiorum, is one of the most important diseases of canola (Brassica napus) in Golestan province, the leading canola producer in Iran. In order to assess the yield loss of canola caused by SSR, 80 fields were surveyed in four different regions of the province (Gorgan, Ali Abad, Kalaleh and Gonbad) during 2006-2007, and SSR intensity was recorded weekly in the fields. Study of yield loss-SSR severity relationships by linear, nonlinear and multiple regression analyses with final intensity ($S_f$), time to initial symptoms ($t_{is}$), Gompertz rate of disease progress ($r_G$), and standardized area under disease progress curve (SAUDPC) as independent variables indicate that single point and integral models were significant ($P < 0.05$) only in three cases. Results of multiple point models which were performed using weekly recorded SSR intensities ($S_1, S_2, \ldots$), were significant in two cases and a general model for 2007 survey was developed using $S_3$ to $S_6$. Eventually, response surface models were developed for each region by integrating $t_{is}$ with SSR intensity variables ($S_f$ or SAUDPC).

Keywords: Brassica napus; crop loss assessment; response surface models; Sclerotinia sclerotiorum

Introduction

Nearly 40% of the canola (Brassica napus L.) planted in Iran is located in Golestan province, in north of the country. In 2006, approximately 62,000 ha were planted in this state, with a production of 119,000 tons and a market value of over US$48030.91** (website of ministry of Jehad-e-Agriculture).

Sclerotinia stem rot (SSR), caused by Sclerotinia sclerotiorum (Lib.) de Bary, is the most important disease affecting canola production in Golestan province. SSR is endemic in the province, with an average incidence of 11.1% (ranging 1-81.5) and 17.2% (ranging 3-78.3) during 2006 and 2007 seasons, respectively (Aghajani et al., 2008b). In spite of well-documented history and importance of the disease in Iran, the relationship between its incidence and yield of canola plants has not been characterized yet. The only published estimates available belong to SSR diseases on canola and other hosts in other countries.

del Rio et al., (2007) studied the impact of SSR on yield of canola in North Dakota and Minnesota and found that 0.5% of the potential yield (equivalent to 12.75 kg/ha) was lost for every unit percentage of SSR incidence (range of 0.18 to 0.96%). Considering the cost of fungicide applications and the market value of canola, a 17% SSR incidence was defined by them as economic damage threshold (EDT) of disease. Koch et al., (2007) in Germany developed a forecasting model to provide decision support for the fungicide spray of canola against SSR (named as SkleroPro) at flowering stage using four weather variables.

Handling Editor: Dr. Vahe Minassian

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Received: 22 May 2012; Accepted: 4 March 2013

**. Based on US$1 = 900 tomans
They explained EDT of SSR as 13 to 25% disease incidence, corresponding to yield levels from 5 to 3 tons/ha, respectively.

Loss is the measurable reduction in quantity and/or quality of yield. In order to reduce losses to the acceptable level, we must first know how much loss occurs (Campbell and Madden, 1990). Data collection for study of disease intensity-yield loss relationship can be carried out by conventional field experiments, survey of natural epidemics, and expert opinion (Madden et al., 2007). Empirical models for estimating yield loss caused by a single disease were categorized into the following types: single point (SP), multiple point (MP), integral, or AUDPC and response surface. SP models utilize one independent variable to estimate loss; this variable has to reflect the entire epidemic. This type of models are also named as “critical point” (CP) models. MP models estimate yield loss from several disease assessments made during growing season. Integral models predict loss from input variables that represent disease for a defined epidemic duration, such as AUDPC (Campbell and Madden, 1990; Nutter, 2001; Teng and Johnson, 1985). Response surface models estimate yield loss from two different types of input variables derived from the epidemics or host (Campbell and Madden, 1990; Teng and Johnson, 1985). Teng and Gaunt (1981) presented a conceptual model for predicting yield loss ($Y$) from disease intensity ($X$) and crop growth stage ($T$), which pictorially may be represented as a three-dimensional response surface with $Y$ as the vertical axis and $XT$ as the two horizontal axes.

An estimation of the relationship between SSR intensity and yield of canola would not only result in a more accurate assessment of the economic impact of this important disease but also would help growers determine the necessity for fungicide applications. Thus, the objective of this study was to estimate the relationship between SSR intensity and yield of canola under growing conditions in Golestan province and determine EDT in order to manage this disease in different regions of the province.

Materials and Methods

To study canola SSR loss in Golestan province, 4 circle-shaped areas with 10 km diameter were considered in different parts of the province (Gorgan, Ali Abad, Gonbad and Kalaleh). During two consecutive cultivation years (2006-2007), 10 fields (cv. Hyola 401) were selected in each area (40 fields per year). After flowering (during March), the fields were surveyed in a regular program (every week) and amount of disease was recorded. For each recording, 500-600 plants were randomly observed in each field and disease severity was determined based on the scale of (Bradley et al., 2006) (0: no disease, 1: small branch infected, 2: large branch infected, 3: stem at least 50% girdled, 4: plant dead, good yield, 5: plant dead, poor yield). By incorporating the values of disease incidence (percent of diseased plants = $I$) and severity of diseased plants; the mean severity ($S$) of disease was calculated for the fields (as the percent values), which is the best estimate of disease intensity (McRoberts et al., 2003).

Yield loss-disease intensity relationships were determined by regression analysis with the four disease progress curves-associated variables as independent variables and yield loss as dependent variable. The independent variables were: (i) $t_s$ = the time in days after sowing to initial symptoms; (ii) $S_f$ = final disease severity; (iii) SAUDPC = standardized area under disease progress curve, calculated by trapezoidal integration method standardized by epidemic duration in days; and (iv) $r_G$ = rate of disease increase based on Gompertz model, fitness of which has been proven in temporal analyses (Aghajani et al., 2008c). Yield loss data were expressed as percent yield loss, which were calculated as follows: (attainable yield – yield of the fields/ attainable yield) X 100 (Ali et al., 1987). Yield of fields with disease intensity lower than 5% in each region, was used as attainable yield.

These analyses were performed as SP ($t_s$ and $S_f$ as independent variables), MP (disease intensities recorded weekly during the epidemic
as independent variables) and integral (SAUDPC and \( r_G \) as independent variables) models for different regions of the study. Data for SP and integral models were analyzed with the Simple Regression procedure of StatGraphics Centurion XV version 15.2.05 (StatPoint, Inc.). MP models were developed with Multiple Regression procedure. First series of analyses were performed with all recorded data, but for simplifying the final model, second series of analyses were performed with “Regression Model Selection” procedure and the best fit model was selected based on adjusted coefficient of determination (\( R_a^2 \)), which is a good statistic for comparing models with different number of independent variables (Madden, 1983). After these analyses, relationships between yield loss and four independent variables were studied by multiple regression analyses and with regard to appropriateness of the results, their relationships were investigated in response surface models with Nonlinear Regression analyses. The overall status of response surface model was as follows:

\[
L = (a + bX_1)(c + dX_2) \tag{1}
\]

in which \( L \) is the yield loss, \( a, b, c \) and \( d \) are parameters, \( X_1 \) is \( t_{is} \), and \( X_2 \) is \( S_f, \) SAUDPC or \( r_G \). Coefficient of determination (\( R^2 \)), \( R_a^2 \), the mean square error (MSE) or standard deviation of the estimates (SEEy), and the pattern of the standardized residuals plotted against either predicted values or the independent variable were used to evaluate the appropriateness of a model to describe the data (Campbell and Madden, 1990; Navas-Cortes et al., 2000).

**Results**

Results of regression analyses showed that yield loss (\( L \)) of canola was significantly (\( P < 0.05 \)) correlated with SSR amount (as its different quantities such as \( S_f, \) SAUDPC and \( r_G \)) (Table 1). \( L \) increased in a field when disease amount (\( S_f, \) SAUDPC, or \( r_G \)) was increased (Figure 1a), but it decreased when the \( t_{is} \) increased i.e. when disease onset was delayed (Figure 1b). Results of SP and integral models were presented in Table 1. Only three cases of these analyses were significant (\( P < 0.05 \)), plot of which is presented in Figure 2. The only significant SP model belonged to Gorgan (2006) based on \( t_{is} \) which describes nearly 80 percent of variability in \( L \). Two significant integral models belonging to Ali Abad (2006) and Kalaleh (2007), were developed based on \( r_G \) (\( R^2 = 0.65 \)) and SAUDPC (\( R^2 = 0.84 \)), respectively.

**Figure 1** Relationships between yield of canola and SSR severity (a) and time (days) to initial symptoms (b) in Golestan province, Iran.
### Table 1 Relationship between yield loss of canola and disease progress curve-associated variables of Sclerotinia stem rot epidemics based on linear regression of data collected from the fields of Golestan province, Iran.

<table>
<thead>
<tr>
<th>Region</th>
<th>Year</th>
<th>Disease progress curve-associated variables</th>
<th>$R^2$</th>
<th>MSE</th>
<th>$R^2$</th>
<th>MSE</th>
<th>$R^2$</th>
<th>MSE</th>
<th>$R^2$</th>
<th>MSE</th>
<th>$R^2$</th>
<th>MSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gorgan</td>
<td>2006</td>
<td>$t_{is}$</td>
<td>80 *</td>
<td>79.1</td>
<td>26</td>
<td>294.8</td>
<td>21</td>
<td>312.9</td>
<td>2</td>
<td>388.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2007</td>
<td>$S_f$</td>
<td>16</td>
<td>253874</td>
<td>12</td>
<td>266714</td>
<td>19</td>
<td>247037</td>
<td>4</td>
<td>291127</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2006</td>
<td>$SAUDPC$</td>
<td>1</td>
<td>373.4</td>
<td>30</td>
<td>262</td>
<td>31</td>
<td>264.5</td>
<td>65 *</td>
<td>131.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2007</td>
<td>$r_G$</td>
<td>19</td>
<td>415.2</td>
<td>31</td>
<td>353</td>
<td>23</td>
<td>397.4</td>
<td>2</td>
<td>504.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2006</td>
<td>$SAUDPC$</td>
<td>9</td>
<td>602.3</td>
<td>79</td>
<td>135.6</td>
<td>84 *</td>
<td>104.8</td>
<td>80</td>
<td>128.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2007</td>
<td>$r_G$</td>
<td>0</td>
<td>229.1</td>
<td>02</td>
<td>224.4</td>
<td>2</td>
<td>225.9</td>
<td>12</td>
<td>201.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2006</td>
<td>$SAUDPC$</td>
<td>2</td>
<td>183.4</td>
<td>5</td>
<td>176.3</td>
<td>5</td>
<td>176.9</td>
<td>10</td>
<td>166.7</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1. Independent variables were: $t_{is}$ (the time in days after sowing to initial symptoms), $S_f$ (final disease severity), $SAUDPC$ (standardized area under disease progress curve), and $r_G$ (rate of disease increase based on Gompertz model).
2. Statistics used in determination of goodness of fit of the models were: $R^2$ (coefficient of determination) and MSE (mean square error).
3. Data of Kalaleh in the first year were not sufficient for developing a model.
4. For each case, * indicates the significance of the developed model ($P < 0.05$).

### Table 2 Relationship between yield loss of canola and disease intensity of Sclerotinia stem rot epidemics based on multiple regression of data collected from the fields of Golestan province, Iran.

<table>
<thead>
<tr>
<th>Region</th>
<th>Year</th>
<th>All recorded data</th>
<th>Selected data</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>$R^2$</td>
<td>MSE</td>
</tr>
<tr>
<td>Gorgan</td>
<td>2006</td>
<td>87.1</td>
<td>127.6</td>
</tr>
<tr>
<td></td>
<td>2007</td>
<td>67.9</td>
<td>284.3</td>
</tr>
<tr>
<td></td>
<td>2006</td>
<td>36.3</td>
<td>654.8</td>
</tr>
<tr>
<td></td>
<td>2007</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>AliAbad</td>
<td>2006</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>2007</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>2006</td>
<td>68.8</td>
<td>114.6</td>
</tr>
<tr>
<td></td>
<td>2007</td>
<td>80</td>
<td>99</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>2006</td>
<td>26.3</td>
</tr>
<tr>
<td></td>
<td>2007</td>
<td>54.8 *</td>
<td>146.2</td>
</tr>
</tbody>
</table>

1. Statistics used in determination of goodness of fit of the models were: $R^2$ (coefficient of determination) and MSE (mean square error).
2. Data of the cases with symbol “-“ were not sufficient for developing a model.
3. For each cases, * indicates the statistical significance of the developed model ($P < 0.05$).
Results of multiple regression analyses showed that most of the developed models were not statistically significant (Table 2). In Table 2, two multiple regression models were developed for all regions in the second year which utilized 6 and 4 independent variables. The second model which used all of the recorded disease intensities, except the data of first and second weeks after disease onset, was a more reliable and simpler model than the first one. The equations of significant MP models are presented in Table 3.

Based on the relationships between yield loss and two independent variables, \( t_{is} \) (Figures 1b and 2a) and disease progress curve-associated variables (Figures 1a, 2b and 2c), their relationship was studied in a single equation with Nonlinear Regression procedure. The result was a response surface model that included a dependent variable and two independent variables. Based on collected data from all of the fields during two years, the equation of final model was obtained as:

\[
L = (20.9 - 0.072 t_{is}) (3.75 + 0.04 S_f) \quad (2)
\]

These analyses were performed using different independent variables (\( S_f \), \( r_G \) and SAUDPC, in addition to \( t_{is} \)) for data collected from four regions (Table 4).

Based on the analyses statistics, final response surface model for Gorgan was developed with \( t_{is} \) and SAUDPC as independent variables, and with \( t_{is} \) and \( S_f \) for other three regions (Figure 3).

Response surface models for explaining the relationships between SSR intensity and yield of canola showed that one percent increase of disease severity causes 0.52 percent decrease in yield. This percent of loss in a field with potential yield of 2 tons/ha corresponds to 10.4 kg canola seeds. At the current market price of US$0.68 per kilogram of canola, each percent of SSR intensity represents a loss of approximately US$7.0/ha. If we consider that the cost of a fungicide application is approximately US$57.8 per ha, EDT of SSR would be equivalent to 8.2% disease intensity. With regard to I-S relationships for SSR (Aghajani et al., 2008a), EDT of this disease in a field with 2 ton/ha potential yield in Gorgan and Gonbad is 10.1 and 17.2 percent of SSR incidence, respectively.

![Figure 2](image-url)  
**Figure 2** Single point (A) and integral (B and C) models for yield loss assessment of Sclerotinia stem rot of canola in Gorgan (2006), Ali Abad (2006) and Kalaleh (2007), respectively.
Table 3 Equations of statistically significant ($P < 0.05$) multiple regression models for describing the relationship between yield loss of canola and disease intensity of Sclerotinia stem rot epidemics in Golestan province, Iran.

<table>
<thead>
<tr>
<th>Region</th>
<th>Year</th>
<th>Equation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gorgan</td>
<td>2006</td>
<td>$L = 39.1 - 4623.6 ,(S1) + 5180.7 ,(S3) - 2355.1 ,(S4)$</td>
</tr>
<tr>
<td>Gonbad</td>
<td>2006</td>
<td>$L = 25.3 + 3551.5 ,(S2) - 1008 ,(S3) - 284 ,(S4)$</td>
</tr>
<tr>
<td>Total</td>
<td>2007</td>
<td>$L = 30.9 + 2125.9 ,(S3) - 3184.3 ,(S4) + 644.6 ,(S5) + 372.3 ,(S6)$</td>
</tr>
</tbody>
</table>

1. $S_1$ to $S_6$ represent the disease intensity of Sclerotinia stem rot of canola in the first to sixth weeks.

Figure 3 Response surfaces as a function of time to initial symptoms (based on days after sowing) and standardized area under disease progress curve (SAUDPC) or disease intensity (percent) of Sclerotinia stem rot for the yield loss (percent) of canola in four region of Golestan province, Iran: Gorgan (a), Ali Abad (b), Kalaleh (c), and Gonbad (d).
Table 4 Relationship between yield loss of canola and disease progress curve-associated variables of Sclerotinia stem rot epidemics based on response surface models for data collected from the fields of Golestan province, Iran.

<table>
<thead>
<tr>
<th>Region</th>
<th>Statistics</th>
<th>Model’s components</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$R^2$</td>
<td>$t_{ls}$</td>
</tr>
<tr>
<td>Gorgan</td>
<td>33</td>
<td>3</td>
</tr>
<tr>
<td>SEE</td>
<td>22.5</td>
<td>27.1</td>
</tr>
<tr>
<td>Ali Abad</td>
<td>8</td>
<td>74</td>
</tr>
<tr>
<td>SEE</td>
<td>12.6</td>
<td>14.4</td>
</tr>
<tr>
<td>Kalaleh</td>
<td>66</td>
<td>65</td>
</tr>
<tr>
<td>SEE</td>
<td>20.3</td>
<td>23.5</td>
</tr>
<tr>
<td>Gonbad</td>
<td>91</td>
<td>8</td>
</tr>
<tr>
<td>SEE</td>
<td>5.7</td>
<td>8.5</td>
</tr>
<tr>
<td>Residuals</td>
<td>64</td>
<td>21</td>
</tr>
</tbody>
</table>

1. Independent variables were: $t$ (the time in days after sowing to initial symptoms), $S_f$ (final disease intensity), SAUDPC (standardized area under disease progress curve), and $r_G$ (rate of disease increase based on Gompertz model).
2. Statistics used in determination of goodness of fit of the models were: $R^2$ (coefficient of determination), $R_a^2$ (adjusted coefficient of determination based on degrees of freedom) and MSE (mean square error).

Discussion

In this study yield loss modeling for Sclerotinia stem rot of canola was carried out for the first time in Iran. There was a negative statistically significant relationship between the seed yield of canola and disease intensity in the fields, i.e. yield loss decreased in a field when disease intensity increased. Similar relationships were found in case of other Sclerotinia diseases of common bean (del Rio et al., 2004), soybean (Danielson et al., 2004; Yang et al., 1999) and canola (del Rio et al., 2007). del Rio et al., (2007) showed that 0.5% of the potential yield was lost for every unit percentage of SSR incidence, but in the current study it was concluded that 0.52% of the field yield was lost for every unit percentage of disease severity (not incidence). With regard to $I$-$S$ relationships for this pathosystem in this area (Aghajani et al., 2008a), 0.25 and 0.4% of potential yield was lost for unit percentage of SSR incidence in Gonbad and the other three regions, respectively. The cause of this obvious difference between the regions is discussed in Aghajani et al., (2008a), but it is mainly due to hot and dry weather conditions in Gonbad region.

For many crops, plant growth and yield are dramatically affected by the time of infection by pathogens, because the sensitivity of a crop to injury varies throughout its growing season. This is especially true for diseases caused by viruses, systemic fungi and bacteria (Madden and Nutter, 1995; Madden et al., 2000; Zadocks, 1985). Effect of infection time (or appearance of symptoms which is more applicable) on the yield can be shown by different models whose common point is a negative correlation of time of symptom appearance with yield loss (Madden and Nutter, 1995). Shtienberg et al., (1990) studied the effects of stripe rust, leaf rust and Septoria blotch on wheat yield in a critical point model and explained that loss is a function of plant growth stage and that the more delayed infections cause lower losses. We found a similar relationship between $t_{ls}$ and yield loss (Figure 1A), although its slope varied in different regions. Yang et al., (1999) in the study of SSR of soybean concluded that different intensity of disease in different regions for a specific variety may be due to difference in environmental conditions, genetic variability of the pathogen, and time of infection. Variation in the slope of regression models of yield loss could be attributed to difference in the infection time.

Our SP models, except in three cases (Figure 2), were not statistically acceptable (Table 1). It is probably due to large variation of collected data from different regions. This type of yield loss models are appropriate for the cases in which a host plant has a specific susceptibility to a pathogen in a specific point (or growth stage) during the growing season. For example, a model was developed for assessing yield loss of rice due to neck blast, in which independent variable was the percent of diseased necks 30 days after heading (Teng and James, 2002).
Results of response surface model developing revealed that canola yield was affected by SSR intensity ($S_f$) in the field, but effects of symptoms appearance time ($t_a$) was stronger than $S_f$ (Table 1 and equation 2), therefore a SP model based on $S_f$ could not reliably describe the yield loss-disease intensity relationships.

The required precision for loss estimates will be one of the major factors governing choice of model. MP and AUDPC models require more inputs of disease assessments than the SP, and consequently they are more precise. The MP model provides the maximum flexibility and accuracy in dealing with situations where the onset, rate of infection and level of infection may vary (Teng and James, 2002). It is revealed in this study that MP models have better fit with the collected data than SP and integral models, but their development needs a hard work and huge data collection. A MP model was developed for all of the regions in the second year with 6 independent variables (recorded disease intensity in 1 to 6 weeks after symptoms appearance). Regression Model Selection is a useful statistical procedure in StatGraphics that decreases the number of input variables. In the mentioned MP model, this procedure decreased the number of variables from 6 to 4 (omitting $S_1$ and $S_2$) (Table 3).

The relationship between disease and loss is inherently a nonlinear one, even though the majority of empirical disease-loss models have been developed using linear regression (Teng and Johnson, 1985). Therefore yield loss models developed by nonlinear regression, are usually more reliable. In this study, relationships between disease and epidemic-derived variables was modeled by nonlinear regression analyses as response surface models, which had higher fit with the data (Table 4). Calpouzos et al., (1976) predicted the yield loss of wheat caused by stem rust based on the slope of epidemic line (infection rate) and growth stage of the host at time of epidemic onset. Navas-Cortes et al., (2000) developed a similar model for Fusarium wilt of chickpea using rate of disease progress and time of symptoms appearance. El Yousfi and Ezzahiri (2002) developed a response surface model based on grain yield of barley, AUDPC of net blotch epidemic and crop growth stage, which explained most of the yield variability ($R^2 = 0.94$). In this study, we also developed response surface models based on time of symptoms appearance ($t_a$) and three other disease progress curve-derived variables ($S_f$, $r_G$, and SAUDPC) via nonlinear regression analyses (Figure 3). Our final response surface models were developed using $t_a$ and $S_f$ (and in case of Gorgan using SAUDPC), which are similar conceptually, to the above mentioned researcher’s models and may help extension service stations to predict seed yield production of canola fields from any disease reading made at a known time (in days after sowing) and consequently, forecast yield with minimum risk.

Models represented by a response surface provide a conceptual framework based on knowledge of disease epidemiology and crop physiology for modeling disease-loss systems (Navas-Cortes et al., 2000).

SSR of canola is a host growth stage-dependent disease, because disease onset occurs by falling colonized petals on the stem, branches or leaves of the plants (Abawi and Grogan, 1979) Therefore, disease cannot start before the time of petal fall, which occurs in 20-30% flowering stage (= growth stages 62-63 BBCH, Thomas, 2008). Little disease may occur by myceliogenic germination of sclerotia in the soil prior to this growth stage. (Morall and Dueck 1982). In Golestan province, petal falling starts at the end of March and our surveys were conducted after this time. In fact, our response surface models have a conceptual, not practical, importance and they were developed mainly as more accurate models for assessing yield loss, comparing with other types of loss models, and for precisely determining the EDT of the disease. This is because any control measures must be done before disease onset, since after symptom appearance, control of SSR is almost impossible. On the other hand, SSR of canola does not occur in a long duration of host growth stage and it is limited to a short time at the end of the season, whereas canola...
yield is accumulated during a long period of growing season (Thomas, 2008). Because of the short duration of epidemic and strict dependence of disease onset to a specific growth stage of the canola, developing a yield loss model based on host growth stage, seemed to not have a good applicability. In contrast, in cereals, most of the foliar diseases occur along a wide range of growth stages and it is possible for the diseases to start from the early stages of growth. Therefore developing response surface models for relating observed disease intensity to the growth stage is logical and applicable (Calpouzos et al., 1976; El Yousfi and Ezzahiri, 2002). Based on the developed models, it is possible to propose a change in sowing date, so that epidemic occurs at or near the end of the growing season and thereby minimize overlapping period of flowering stage and ascospore discharge of *S. sclerotiorum*. In alfalfa, Sclerotinia crown and stem rot was controlled by early sowing so that plants will be at least 10 weeks old at the time of apothecia appearance (Sulc and Rhodes, 1997).

EDT of disease in this study, based on disease incidence, is less than other studies (10.5% compared with 17%) and this is mainly because of different canola prices in Iran and other countries, so that in 2007, price of canola seeds was US$0.68, whereas in USA it was US$0.24 (del Rio et al., 2007; Koch et al., 2007). Each percent of SSR intensity in a field with potential yield of 2 tons/ha in the province (except Gonbad) represents a loss of approximately US$7.12/ha, which is more than twice that of SSR loss (in price) in USA (del Rio et al., 2007).

In most of the yield loss assessments of Sclerotinia diseases of crops, disease incidence was used as the quantity of disease intensity, while in many cases, such as foliar diseases, severity is mostly utilized (Campbell and Madden, 1990). del Rio et al., (2007) believed that SSR primarily affects stem and branches of canola plants. As a consequence, plant parts above the infected tissues wilt or die prematurely, a symptom that resembles more the effect of some vascular pathogens or stem canker pathogens. In this sense, a more accurate estimation of SSR intensity could be achieved if the overall impact of the disease on the plant is evaluated, instead of just measuring lesion expansion rates, with multiple readings instead of single observations. Our results of incidence-severity relationships (Aghajani et al., 2008a) showed that a unit of *I* means just “a diseased plant”, without regard to disease severity on the plant, and this is while in horizontal spread of SSR in the field (Morall et al., 1982), especially in a field with dense canopy, disease intensity in many of the plants is limited to infection of lateral branches which does not seem to affect the yield. Therefore, disease incidence cannot explain the actual amount of SSR in the field. This problem is more obvious in Gonbad region where the recorded disease intensity was nearly half that of disease incidence. As a consequence, in order to use the incidence data for yield loss assessment, it would be necessary to study *I*-*S* relationships in the region, and convert *I* values to *S* ones, based on the relationships.

However, the response surface models and EDTs should be utilized in a decision support system for proposing control measures before the onset of epidemic, because a yield loss model and a threshold model are two major components of a decision making model for the management of a moderately destructive pathogen, such as *S. sclerotiorum*, in an extensively grown crop, i.e. canola (Shtienberg, 2000).

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ارزیابی خسارت بیماری پوسیدگی اسکلروتیپای ساقه کلزا در ایران

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دریافت: ۲ خرداد ۱۳۹۱؛ پذیرش: ۱۴ اسفند ۱۳۹۱

چکیده: پوسیدگی اسکلروتیپای ساقه (SSR) ناشی از فارج Sclerotinia sclerotiorum در گلستان، استان چهارمی به عامل موجب کلزا در برخی مزارع در چهار شهرستان مختلف استان گلستان (قومرگان، علی آباد کلاته و گنبد) طی دو سال زراعی ۸۵-۸۶ و ۸۶-۸۷ مورد بررسی قرار گرفت و مقادیر بیماری در آنها به صورت هفتگی ثبت شد. بررسی روابط بین کاهش اعمای بیماری و شدت SSR از طریق آنالیزهای رگرسیون خطی، غیرخطی و چندتراکمی با استفاده از نرم‌افزار SPSS آنالیز شد. نتایج نشان داد که بیماری SSR در سطح هفتگی و محتوای سطحی در زمان شکوفا بوده و در واقع می‌تواند به عنوان یکی از عوامل اصلی کاهش غلظت این بیماری در زمینه شکوفا در این استان، اثربخش شود.

واژگان کلیدی: کلزا، پوسیدگی اسکلروتیپای ساقه، ارزیابی خسارت، مدل‌های سطح پایخ