YIELD LOSS ASSESSMENT OF SCLEROTINIA STEM ROT OF CANOLA IN IRAN

REZAPOUR MAHDI ALAMDARLOO [1] & MOHAMMAD ALI AGHAJANI[2*]

1Department of Plant Protection, University of Agriculture, Sari, Iran

2Department of Plant Protection Research, Agricultural and Natural Resources Research Center of Golestan Province, Gorgan, Iran

*Corresponding author: M. A. Aghajani, E Mail: maaghajanina@yahoo.com

ABSTRACT

Sclerotinia stem rot (SSR), caused by *Sclerotinia sclerotiorum*, is the most important disease of canola (*Brassica napus*) in Mazandaran and Golestan provinces, 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 (Galogah, Ali Abad, Dashte Naz and Gonbad) during 2010-2011, 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 3 cases. Results of multiple point models which performed using weekly recorded SSR intensities (S_1, S_2, …), were significant in 2 cases and a general model for 2011 survey was developed using S_3 to S_6. At last, response surface models were developed for each region by integrating t_{is} with SSR intensity variables (S_f or SAUDPC).

Keywords*: Brassica napus,* response surface models, *Sclerotinia sclerotiorum,* yield loss assessment

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[1] (website of ministry of Jehad-e-Agriculture).

Sclerotinia stem rot (SSR), caused by *Sclerotinia sclerotiorum* (Lib.) de Bary, is the most important diseases affecting canola production in Mazandaran and Golestan provinces. SSR is endemic in the province, with an average incidence 0f 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 SSR history and importance in Iran, the relationship between its incidence and yield of canola plants has not been characterized recently. The only published estimates available were belonged to SSR diseases on canola and other hosts in the other countries. del Rio et al. (2007) with the study of impact of SSR on yield of canola in North Dakota and Minnesota 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 explained as economic damage threshold (EDT) of disease. Koch et al. (2007) 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 in Germany. 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). Collection data 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 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

---

[1]. Based on US$1 = 900 tomans

estimate yield loss from two different types of input variables derived from the epidemic 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 $X.T$ as the two horizontal axes.

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

MATERIAL AND METHODS

In order to study of canola SSR in Golestan province, 4 circle-shaped areas with 10 km diameter were considered in different parts of the province (Galogah, Ali Abad, Gonbad and Dashte Naz). During two consecutive cultivation year (2010-2011), 10 fields (cv. Hyola 401) were selected in each area (40 fields in each year). After flowering (during

March), the fields were surveyed in a regular program (every week) and amount of disease was recorded. In each record, 500-600 plants randomly observed in each field and disease severity was determined based on the scale (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) (Bradley et al., 2006). With incorporating the values of disease incidence (percent of diseased plants = I) and severity of diseased plants (X), mean severity (S) of disease was calculated for the fields, 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. Independent variables were: (i) t_{is} = the time in days after sowing to initial symptoms; (ii) S_f = final disease intensity; (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, which its fitness proved in temporal analyses (Aghajani et al., 2008c). Yield loss data were expressed as percent yield loss, which was calculated as

follows: attainable yield – yield of the fields/ yield of control fields X 100 (Ali et al., 1987). Yield of fields with disease intensity lower 5 % in each region, were used as attainable yield.

These analyses were performed as four SP (t_{is} 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 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 studied by multiple regression analyses and with regard to appropriateness of the results, their relationships investigated in response surface models with Nonlinear Regression analyses. The overall status of response surface model was as follows:

$$L = (a + bX_1)(c + dX_2) \quad (1)$$

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 ($P< 0.05$), that their plot is presented in Figure 2. The only significant SP model belonged to Galogah (2010) based

on t_{is}, which describe nearly 80 percent of variability in *L*. Two significant integral models belonged to Ali Abad (2010) and Dashte Naz (2011), which developed based on r_G ($R^2 = 0.65$) and SAUDPC ($R^2 = 0.84$), respectively.

Results of multiple regression analyses showed that most of the developed models were not statistically significant (Table 2). In this table, 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 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 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, equation of final model was 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 Galogah were developed with t_{is} and SAUDPC as independent variables, and with t_{is} and S_f for other three regions (Figure 3).

Linear and response surface models for explain the relationships between SSR intensity and yield of canola showed that one percent increase of disease intensity causes 0.52 percent decrease in yield. This percent of loss in a field with potential yield of 2 tons/ha corresponds with 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 Galogah and Gonbad is 10.1 and 17.2 percent of SSR incidence, respectively.

DISCUSSION

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 intensity (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 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 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 showed by different models which their 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 more delayed infections cause lower losses. We found a similar relationship between t_{is} 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 3 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 was appropriate for the cases in which a host plant had 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 affected by SSR intensity (S_f) in the field, but effects of symptoms appearance time (t_{is}) 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 to deal 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 to a hard work and collection of high quantity of data. 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 decrease 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_{is}) 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_{is} and S_f (and in case of Galogah using SAUDPC). It which is similar, in conception, to the abovementioned researcher's models, may help extension services 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, branch or leaf of the plants (Abawi and Grogan, 1979) Therefore, disease cannot be started before the time of petal fall, which occur in 20-30% flowering stage (= growth stages 62-63 BBCH, Thomas, 2008). Few disease amounts in the canola fields before this time, caused by myceliogenic germination of sclerotia in the soil (Morall et al., 1982). In Mazandaran and Golestan provinces, petal falling is started 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, and 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 in the end of the season, whereas canola yield is accumulated during a long period of growing season (Thomas, 2008). Because of this short duration of epidemic and strict dependence of disease start 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 stage and it is possible starting the diseases from the primary 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 changing sowing date, so that epidemic occurs at the end of the growing season and minimize simultaneous period between flowering stage of canola and ascospores discharge of *S. sclerotiorum*. In alfalfa, Sclerotinia root and stem rot was controlled by changing sowing date, so that due to early sowing, plants will be at least 10-week in 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 2011, price of canola seeds was US$0.68, whereas in USA 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 of SSR loss (in price) in USA (del Rio et al., 2007).

In most of the yield loss assessment of Sclerotinia diseases of crops, disease incidence was used as the quantity of disease intensity, while in many cases, such as foliar diseases, severity 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 as a diseased plant, without regard to disease severity on the plant, and this is while in horizontal progress 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 that disease intensity was nearly half of disease incidence. As a consequent, in order to use of incidence data for yield loss assessment, it is necessary to study *I-S* relationships in that 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 starting epidemic, because a yield loss model and a threshold model are two major components of a decision making model for themanagement of a moderately destructive pathogen, e.g. *S. sclerotiorum,* in an extensive crop, e.g. canola (Shtienberg, 2000).

REFERENCES:

[1] Abawi GS, and Grogan, R. G. (1979) Epidemiology of diseases caused by *Sclerotinia* species. Phytopathology 69:899-904.

[2] Aghajani MA, Safaei N and Alizadeh A (2008a) Incidence-severity relationships for Sclerotinia stem rot of canola. Page 53 in: Proceeding of the Iranian 18th Plant Protection Congress, Hamedan, Iran.

[3] Aghajani MA, Safaei N and Alizadeh A (2008b) Sclerotinia infection situation of canola in Golestan province. Page 52 in: Proceeding of the Iranian 18th Plant Protection Congress, Hamedan, Iran.

[4] Aghajani MA, Safaei N and Alizadeh A (2008c) Temporal study of sclerotinia stem rot of canola in Golestan province. Page 51 in:

Proceeding of the Iranian 18th Plant Protection Congress, Hamedan, Iran.

[5] Ali MEK, Warren HL, Latin RX (1987) Relationship between anthracnose leaf blight and losses in grain yield of sorghum. Plant Disease 71:803-806.

[6] Calpouzos L, Roelfs AP, Madson ME, Martin FB, Welsh JR, Wilcoxson RD (1976) A new model to measure yield losses caused by stem rust in spring wheat. Minnesota Agricultural Experiment Station Technical Bulletin 307:1-23.

[7] Campbell CL, Madden LV (1990) Introduction to Plant Disease Epidemiology. John Wiley & Sons, New York.

[8] Danielson GA, Nelson BD, Helms TC (2004) Effect of Sclerotinia stem rot on yield of soybean inoculated at different growth stages. Plant Disease 88:297-300.

[9] Del Río LE, Venette JR, Lamey HA (2004) Impact of white mold incidence on dry bean yield under nonirrigated conditions. Plant Disease 88:1352-1356.

[10] del Río LE, Bradley CA, Henson RA, Endres GJ, Hanson BK, McKay K, Halvorson M, Porter PM, Gare

DGL, Lamey HA (2007) Impact of Sclerotinia stem rot on yield of canola. Plant Disease 91:191-194.

[11] El Yousfi B, Ezzahiri B (2002) Net blotch in semi-arid regions of Morocco. II. Yield and yield-loss modeling. Field Crop Research 73:81-93.

[12] Koch S, Dunke S, Kleinhenz B, Röhrig M, Tiedemann Av (2007) A crop loss-related forecasting model for Sclerotinia stem rot in winter oilseed rape. Phytopathology 97:1186-1194.

[13] Madden LV (1983) Measuring and modeling crop losses at the field level. Phytopathology 73:1591-1596.

[14] Madden LV, Hughes G, Irwin ME (2000) Coupling disease progress-curve and time-of-infection functions for predicting yield loss of crops. Phytopathology 90:788-800.

[15] Madden LV, Hughes G, van den Bosch F (2007) The Study of Plant Disease Epidemics. American Phytopathological Society, St. Paul, Minnesota, USA.

[16] Madden LV, Nutter FW, Jr. (1995) Modeling crop losses at the field scale. Canadian Journal of Plant Pathology 17:124-137.

[17] McRoberts N, Hughes G, Madden LV (2003) The theoretical basis and practical application of relationships between different disease intensity measurements in plants. Annals of Applied Biology 142:191-211.

[18] Morrall RAA, Dueck J (1982) Epidemiology of sclerotinia stem rot of rapeseed in Saskatchewan. Canadian Journal of Plant Pathology 4:161-168.

[19] Navas-Cortés JA, Hau B, Jiménez-Díaz RM (2000) Yield loss in chickpeas in relation to development of Fusarium wilt epidemics. Phytopathology 90:1269-1278.

[20] Nutter FW, Jr. (2001) Disease losses. In: Maloy OC, Murray TD (eds) Encyclopedia of plant pathology. John Wiley & Sons, Inc., pp 340-351.

[21] Shtienberg D (2000) Modelling: the basis for rational disease management. Crop Protection 19:747-752.

[22] Shtienberg D, Dinoor A, Marani A (1990) Wheat Disease Control Advisory, a decision support system for management of foliar diseases of wheat in Israel. Canadian Journal of

Plant Pathology 12:195-203.

[23] Sulc RM, Rhodes L H (1997) Planting date, fungicide, and cultivar effects on sclerotinia crown and stem rot severity in alfalfa. Plant Disease 81:13-17.

[24] Teng PS, Gaunt RE (1981) Modeling systems of disease and yield loss in cereals. Agricultural Systems 6:131-154.

[25] Teng PS, James WC (2002) Disease and yield loss assessment. In: Waller JM, Lenné JM, Waller SJ (eds) Plant Pathologist's Pocketbook. CAB International, pp 25-38.

[26] Teng PS, Johnson KB (1985) Analysis of epidemiological components in yield loss

assessment. In: Kranz J, Rotem J (eds) Exprimental techniques in plant disease epidemiology. Springer, Germany, pp 179-189.

[27] Thomas P (2008) Canola Growers Manual. Canola Council of Canada, Winnipeg, Manitoba, Canada, Retrieved from http://www.canola-council.org/default.aspx.

[28] Yang XB, Lundeen P, Uphoff MD (1999) Soybean varietal response and yield loss caused by *Sclerotinia sclerotiorum*. Plant Disease 83:456-461.

[29] Zadoks JC (1985) On the conceptual basis of crop loss assessment: The threshold theory. Annual Review of Phytopathology 23:455-473.

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 Mazandaran and Golestan provinces, Iran.

Region	Year	Disease progress curve- associated variable[1]							
		t_{is}		S_f		SAUDPC		r_G	
		R^2[2]	MSE[2]	R^2	MSE	R^2	MSE	R^2	MSE
Galogah	**2010**	0.80 *[4]	79.1	0.26	294.8	0.21	312.9	0.02	388.2
	2011	0.16	253874	0.12	266714	0.19	247037	0.04	291127
AliAbad	**2010**	0.01	373.4	0.30	262	0.31	264.5	0.65 *	131.9
	2011	0.19	415.2	0.31	353	0.23	397.4	0.02	504.6
Dashte Naz[3]	**2010**	-	-	0.20	377.2	0.19	379.1	0.07	440
	2011	0.09	602.3	0.79	135.6	0.84 *	104.8	0.80	128.3
Gonbad	**2010**	0.00	229.1	0.02	224.4	0.02	225.9	0.12	201.2
	2011	0.02	183.4	0.05	176.3	0.05	176.9	0.10	166.7

1.   Independent variables were: *t*is (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: *R2* (coefficient of determination) and MSE (mean square error).

3.   Data of Dashte Naz in the second year were not sufficient for developing a model.

4.   For each cases, * 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.

Region	Year	All recorded data[1]		Selected data[3]	
		R^2	MSE	R^2	MSE
Galogah	2010	87.1	127.6	84.8 *	99.9
	2011	67.9	284.3	42.4	170.2
AliAbad	2010	36.3	654.8	32	420.2
	2011	-	-	-	-
Dashte	2010	-	-	-	-
Naz	2011	-	-	-	-
Gonbad	2010	68.8	114.6	68.6 *	96
	2011	80	99	79.9	74.8
Total	2010	26.3	425.7	24.8	440.3
	2011	54.8 *	146.2	51.7 *	135.2

1. Statistics used in determination of goodness of fit of the models were: $R2$ (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 statistically significance of the developed model ($P < 0.05$).

Table3. 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 Mazandaran and Golestan provinces, Iran.

Region	Year	Equation[1]
Galogah	2010	L = 39.1 - 4623.6(S1) + 5180.7(S3) - 2355.1(S4)
Gonbad	2010	L = 25.3 + 3551.5(S2) – 1008(S3) – 284(S4)
Total	2011	L = 32.1 - 1108.3(S1) - 269.2(S2) + 2517.7(S3) - 3463.2(S4) + 777.4(S5) + 349.5(S6)
	2011	L = 30.9 + 2125.9(S3) - 3184.3(S4) + 644.6(S5) + 372.3(S6)

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

Table4.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.

Region	Statistics[2]	Model's components[1]		
		$t_{is} - S_f$	$t_{is} - r_G$	t_{is} - SAUDPC
Galogah	R^2	0.33	0.03	0.97
	SEE	22.5	27.1	4.1
	R_a^2	0	0	0.88
Ali Abad	R^2	0.8	0.74	0.78
	SEE	12.6	14.4	13.4
	R_a^2	0.66	0.55	0.61
Dashte Naz	R^2	0.49	0.3	0.46
	SEE	20.3	23.5	20.6
	R_a^2	0	0	0
Gonbad	R^2	0.91	0.8	0.89
	SEE	5.7	8.5	6.4
	R_a^2	0.64	0.21	0.55

1. Independent variables were: t_{is} (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: $R2$ (coefficient of determination), * (adjusted coefficient of determination based on degrees of freedom) and MSE (mean square error).
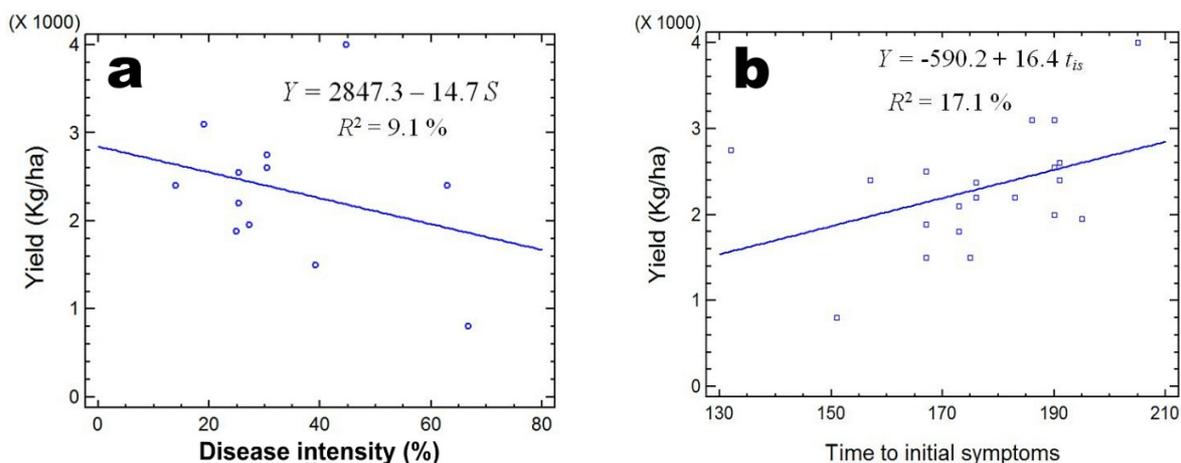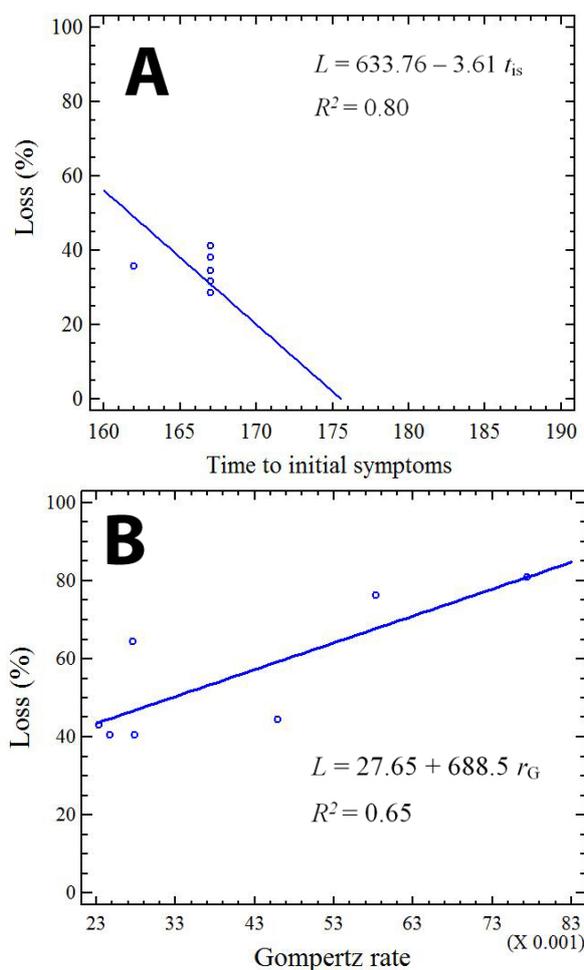
Figure 1. Relationships between yield of canola and SSR intensity (a) and time to initial symptoms (b) in Mazandaran and Golestan provinces, Iran.
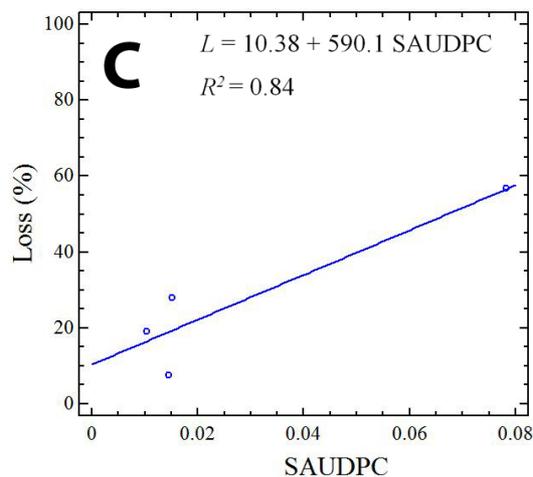
$L = 10.38 + 590.1\ SAUDPC$

$R^2 = 0.84$

Figure 2. Single point (A) and integral (B and C) models for yield loss assessment of Sclerotinia stem rot of canola in Galogah (2010), Ali Abad (2010) and Dashte Naz (2011), respectively.

a

$L = [247.97 - (1.23\ t_{is})]\ [-4.26 + (82.86\ SAUDPC)]$

$R^2 = 0.97$

b

$L = [20.1 - (0.09\ t_{is})]\ [8.36 + (0.07\ S_f)]$

$R^2 = 0.8$

c

$L = [23.06 - (0.096\ t_{is})]\ [4.3 + (0.12\ S_f)]$

$R^2 = 0.49$

d

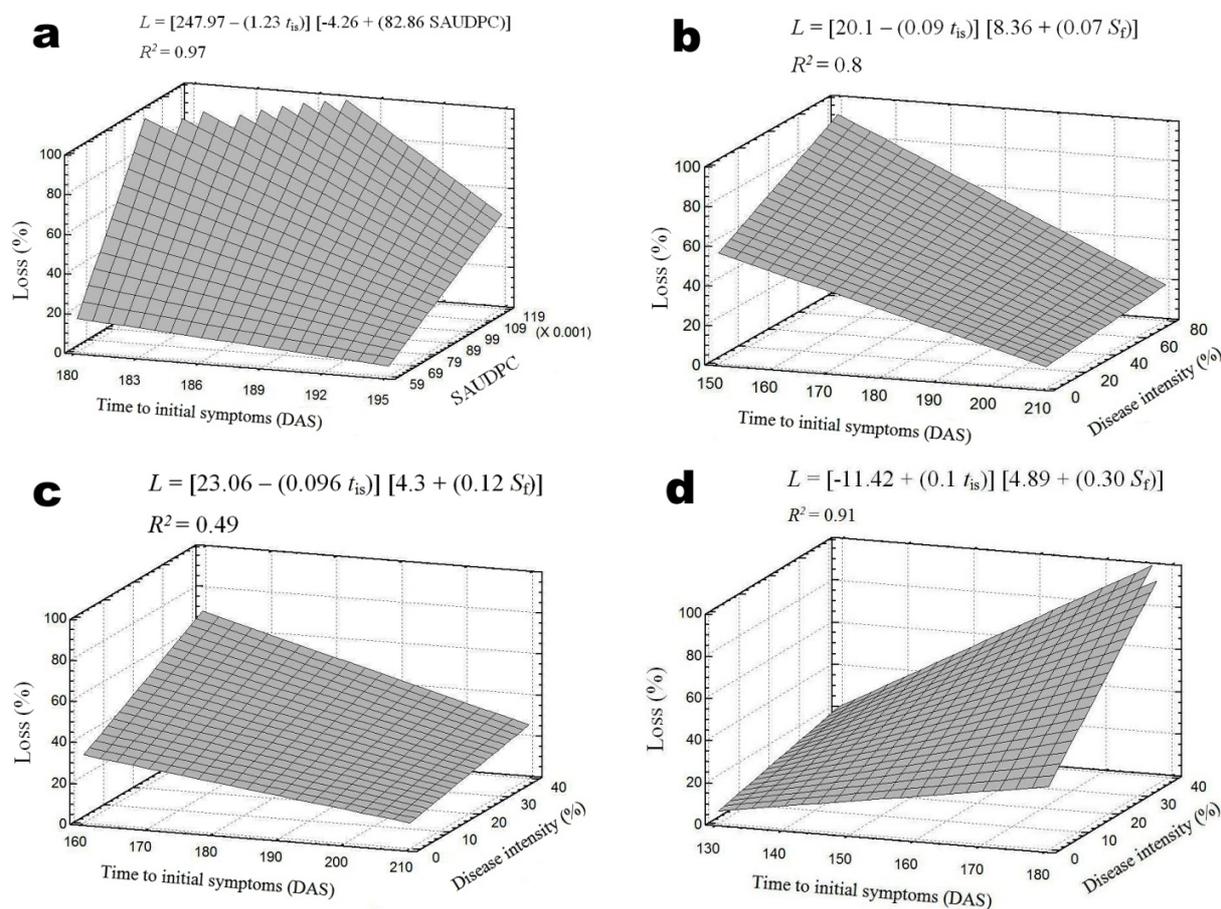$L = [-11.42 + (0.1\ t_{is})]\ [4.89 + (0.30\ S_f)]$

$R^2 = 0.91$

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 Mazandaran and Golestan provinces, Iran: Galogah (a), Ali Abad (b), Dashte Naz (c), and Gonbad (d).