Effects of Cultivar, Leaf Wetness Duration, Temperature, and Growth Stage on Infection and Development of Ascochyta Blight of Lentil

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ABSTRACT

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Growth chamber tests were conducted on three lentil cultivars susceptible (Eston), moderately resistant (Laird), and resistant (Indianhead) to Ascochyta blight, caused by *Ascochyta fabae* f. sp. *lentis*. Leaf wetness after inoculation, temperature, and plant growth stage had significant effects on infection and disease development. The highest infection frequency occurred with a wetness period of 24 or 48 h. For all cultivars, latent periods were shortest (6–7 days) at 20 C and longest (13–14 days) at 10 C. Temperature had little effect on lesion size and number of pycnidia per lesion, but infection frequency was higher at 10 and 15 C than at 25 C. The lowest number of lesions and pycnidia and the smallest lesions occurred on Indianhead at 10, 15, and 20 C. Lesions were fewer and smaller and contained fewer pycnidia on Laird than on Eston at 15 and 20 C but not at 10 C. On stems, the highest number of lesions and pycnidia and the largest lesions were produced on Laird at all temperatures. Based on results of growth chamber and field tests, tissues below the top four or five nodes on the main stem and secondary branches were almost completely resistant in all three cultivars. The effect of this tissue-age-related resistance on disease severity was most apparent at the podding stage.

Additional keywords: components of resistance, controlled conditions, pulse crop.

Ascochyta blight, caused by *Ascochyta fabae* Speg. f. sp. *lentis* Gossen et al, is a serious disease in Canada and in many other countries that produce lentil (*Lens culinaris* Medik.). The disease is polycyclic and can occur on all aboveground parts of the lentil plant, reducing yield and seed quality (4). The pathogen survives in seed and plant residues (6,8) and is dispersed predominantly by rain splash (12).

Planting disease-free seed and providing a 3-yr break between lentil crops are recommended to help control Ascochyta blight (9). Foliar fungicides are also effective and economical. Generally, one application of chlorothalonil at early flowering provides good control of the disease, but two or more are required if conditions during flowering and podding remain cool and moist (1,2). Few studies have dealt with the effects of environmental factors on infection and disease development. Morrall (10) related weather conditions to frequency of seed transmission and subsequent epidemic development, and Gossen and Morrall (5) examined conditions that promoted seed infection after the crop was cut and placed in swaths. Other workers have made general observations relating weather to disease development (1–3,11). However, all of these reports were based on data collected from the field, where weather factors are difficult to monitor and control. Studies under controlled conditions are required to provide more specific information on the effects of environmental factors on the disease. The objective of these experiments was to examine the effects of cultivar resistance, duration of leaf wetness after inoculation, temperature, and growth stage on infection and development of Ascochyta blight of lentil under controlled conditions.

MATERIALS AND METHODS

Duration of leaf wetness. The effects of the duration of leaf wetness after inoculation were examined on three lentil cultivars using a single growth chamber. Cultivars Eston, Laird, and Indianhead, bred for western Canadian conditions, are, respectively, early-maturing and susceptible to Ascochyta blight, late-maturing and moderately resistant, and late-maturing and resistant. Seeds of Eston, Laird, or Indianhead were planted in 24 10-cm-diameter plastic pots containing a peat:vermiculite (1:1) mixture. Eston was planted in four additional pots to provide a noninoculated control. The pots were placed in a growth chamber maintained with 20/17 C day/night temperatures, 60–90% RH, and a 16-h photoperiod with a light intensity of 140 $\mu E \cdot m^{-2} \cdot s^{-1}$. Temperature and RH were monitored with a micrologger (Model CR21, Campbell Scientific Canada Corp., Edmonton, AB), using a sensor (Model 231, Campbell Scientific Canada Corp.) placed in the center of the chamber and mounted 10 cm above the surface of wetted potting mixture in a 10-cm plastic pot. All pots were fertilized weekly with a water-soluble fertilizer (20-20-20, N-P-K plus micronutrients). Shortly after emergence, the seedlings were thinned to five per pot.

A culture of *A. f. lentis* was obtained by plating infected lentil seed from a field near Zealandia, Saskatchewan, on potato-dextrose agar (PDA). Single conidia from this culture were transferred to fresh PDA plates. After 6 days of growth, one single-spore isolate, designated AFL1, was selected and a 2-mm-diameter plug of the colony was transferred to each of four 25-ml glass vials containing sterilized lentil seeds. Before inoculation, the seeds in the vials were soaked for 24 h and autoclaved twice 48 h apart, for 25 min each time. After inoculation, the vials were incubated at room temperature (20 ± 3 C) for 8 days. The vials were stored at 0 C until used. After 1 yr of storage, AFL1 was recultured on sterilized lentil seed.

To inoculate plants, cultures of AFL1 were obtained by plating the stored infected seed on PDA. After 6–8 days of growth, conidial suspensions were prepared in distilled water. The concentration of conidia in the suspensions was determined with a hemacytometer and adjusted to 2×10^5/ml. Ten-day-old seedlings were well watered, then inoculated with the suspension, using an atomizer (1 ml per seedling). After inoculation, each pot was covered with a 2-L clear-plastic chamber, which maintained leaf wetness throughout the treatment periods. Four pots of each cultivar were exposed to leaf wetness for 0, 6, 12, 24, 48, or 72 h. The temperature in the growth chamber remained unaltered. These procedures were repeated twice; thus, the experiment was a 3 × 6 factorial in a randomized complete block design with three replicates. The control seedlings were sprayed with distilled water and exposed to leaf wetness for 72 h.

After inoculation, the seedlings were examined daily and incubation and latent periods were estimated as the number of days required for development of symptoms and pycnidia, respectively, in at least two pots in a treatment. Ten days after inoculation, disease was measured on leaflets and stems separately by: 1) estimating infection frequency by counting the total number of lesions on the five seedlings in each pot and 2) measuring length and width of lesions and counting the number of pycnidia per lesion in a subsample of five to 10 randomly selected lesions per pot.

Temperature. The effects of four treatments—10, 15, 20, and 25 C—were examined on all three cultivars. Two growth chambers were used to replicate the experiment three times. Two replicates of each temperature treatment were conducted at eight different times in the first growth chamber, and one replicate was conducted at four different times in the second growth chamber. Thus, the experiment was a three-replicate, 4 × 3 factorial in a split-plot design, with the main plot treatments (temperature) randomly arranged.

Seeds of Eston, Laird, and Indianhead were planted in four pots and the seedlings were thinned to five per pot. The pot size, potting mixture, fertilization, and noninoculated control were the same as in the experiment on duration of leaf wetness. The pots were placed in a growth chamber maintained at 20 C, with 60–90% RH and a 16-h photoperiod with light intensity of 140–160 $\mu E \cdot m^{-2} \cdot s^{-1}$. Temperature and RH were monitored as indicated previously. Ten days after emergence, the seedlings were well watered and the temperature in the growth chamber was adjusted to the appropriate level. Each pot was removed and inoculated. The controls were sprayed with distilled water as described previously. All of the pots, including the one containing the sensor, were covered with plastic chambers and returned to the growth chamber. To compensate for radiant heating, the temperature in the growth chamber was reduced until the correct treatment temperature was obtained inside the plastic chamber. After 48 h of leaf wetness, the plastic chambers were removed and the temperature in the growth chamber was readjusted to the treatment temperature. Ten days after inoculation for the 15, 20, and 25 C treatments and 14 days after inoculation for the 10 C treatment, incubation and latent periods were estimated and infection frequency and disease were assessed as described previously.

Growth stage. The effects of growth stage on infection and disease development were examined in the growth chamber and in the field. In the growth chamber experiment, seeds were planted in four 13-cm-diameter plastic pots on days 1, 10, and 15 for Laird and Indianhead and on days 15, 25, and 30 for Eston. The purpose of the staggered sowing dates was to provide plants at the time of inoculation at the vegetative, flowering, and podding stages. The potting mixture and the fertilization and thinning procedures were the same as in previous experiments. After sowing, the pots were placed in a growth chamber. A second replicate was sown with a 3-day delay from the first replicate, using a different growth chamber. Both growth chambers were maintained at 20 C as described for the temperature experiment. The experiment was a 3 × 3 factorial in a two-replicate randomized complete block design.

The plants in the second replicate developed faster and reached the desired growth stages sooner than those in the first replicate. Therefore, at 62 and 67 days after the first sowing date in replicates two and one, respectively, the pots were removed from the growth chamber and entire plants in each pot were sprayed until runoff with a suspension of 2×10^5 conidia per milliliter of AFL1. The temperature in the growth chamber was reduced to 15 C, and the plants in each pot were covered with a 35 × 80 cm polyethylene bag before being placed back in the growth chamber. The bags were removed 48 h later, and disease was assessed 10–11 days after inoculation.

In the field experiment, the three cultivars were planted at Saskatoon, Saskatchewan, in 1993 on land where lentil had not been grown for at least 2 yr. Each cultivar was sown in 0.6 × 1 m plots on each of five sowing dates: 17, 27, and 28 May and 9 and 25 June for Laird and Indianhead and 1, 14, 16, and 29 June and 7 July for Eston. Row spacing was 15 cm, and the plots were arranged in a randomized complete block design with four replicates. On the evening of 10 August, plots with plants at the flowering and podding stages were sprayed with a conidial suspension of AFL1 as described previously. No plants were at the vegetative stage. After inoculation the plots were covered with polyethylene tents. The following day was cool and overcast; therefore, the tents were removed 18 h after inoculation. At 10 days after inoculation, 10 plants were collected from the center two rows of each plot and disease was assessed.

In both growth chamber and field experiments, the plants were examined daily and the incubation and latent periods were estimated as described previously. Although entire plants were examined for symptoms, disease was assessed only on the main shoot of each plant by: 1) rating disease severity on the top four fully expanded leaves, using the Horsfall-Barratt scale (7), 2) measuring the incidence of diseased leaflets among the top four leaves, 3) counting the number of lesions on the top four leaves (growth chamber experiment only), and 4) counting the number of lesions on the main stem. In the growth chamber experiment, 2 days were required to rate each replicate; therefore, one-half of the pots in each treatment were assessed on each day. Lesion

size and number of pycnidia per lesion were not estimated in either experiment because many of the lesions had coalesced.

Data analysis. The data were analyzed using the Statistical Analysis System (SAS Institute, Cary, NC) and Minitab Statistical Software (Minitab Inc., State College, PA). The effects of treatments on infection and disease were examined by analyses of variance. When quantitative factors such as leaf wetness duration and temperature were significant, trends in the data were further examined by orthogonal polynomials (14). When only qualitative factors were significant, such as cultivar and growth stage, treatment means were separated using Duncan's new multiple range test. The effect of temperature on incubation and latent periods was examined by polynomial regressions. Logarithmic or arcsine transformations were applied to some data sets to correct for high variance or nonnormality of the data, respectively. In some cases, the level of disease was too low to estimate the variables of interest adequately, so these data were excluded from analyses.

RESULTS

Duration of leaf wetness. Infection frequency in leaflets, as measured by lesion number, was significantly affected by duration of leaf wetness after inoculation and by cultivar but not by their interaction. Orthogonal polynomials showed that in all three cultivars, the response of infection frequency to increasing duration of leaf wetness was well described by a cubic function. No infection occurred with no wetness period and only a few lesions developed with a 6-h period (Fig. 1). As the duration of leaf wetness increased, so did the number of lesions, until an optimum was reached at 48 h. When the duration was extended to 72 h, the number of leaflet lesions decreased. The number of lesions was usually highest in Eston, intermediate in Laird, and lowest in Indianhead.

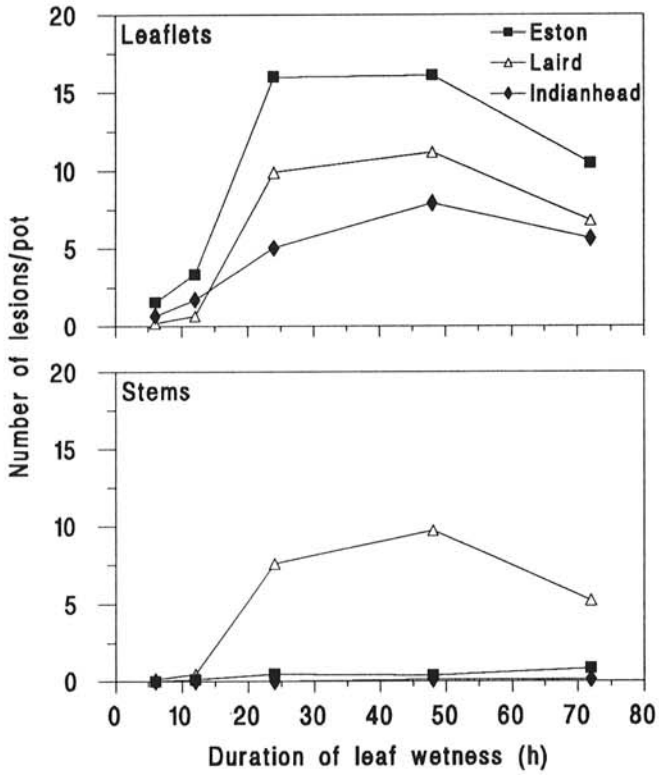

Fig. 1. Effect of duration of leaf wetness after inoculation on number of lesions of Ascochyta blight in leaflets and stems of lentil cvs. Eston, Laird, and Indianhead. Data were transformed to $x' = \log(x + 1)$ before analysis. Each point indicates the detransformed mean number of lesions averaged over three cultivars (12 pots total, with five seedlings per pot). The standard error of a difference based on the transformed data was 0.11 for leaflets and 0.13 for stems.

The incubation period was 7–8 days for all three cultivars when the leaf wetness period was at least 6 h. In all cases, the latent period was 1–2 days longer than the incubation period.

Data on stem infection in Indianhead were excluded from the analysis of variance because a low level of infection occurred only with 48 or 72 h of leaf wetness. Stem infection in Eston and Laird was significantly affected by the interaction of the duration of leaf wetness after inoculation and cultivar. The duration of leaf wetness-quadratic × cultivar interaction was also significant, indicating that the curvilinear response in Laird did not occur in Eston (Fig. 1). In Laird, a large increase in the number of stem lesions with longer periods of leaf wetness occurred up to 48 h, but a decrease in the number occurred with a 72-h period.

Lesion length and width and number of pycnidia per leaflet lesion could not be estimated for the 0-, 6-, and 12-h leaf wetness periods because too few lesions were produced. Therefore, only the data from the 24-, 48-, and 72-h wetness periods were analyzed. Development of Ascochyta blight, as measured by lesion length and width and number of pycnidia per lesion, was significantly affected by cultivar but not by the duration of leaf wetness after inoculation or by the interaction of leaf wetness and cultivar.

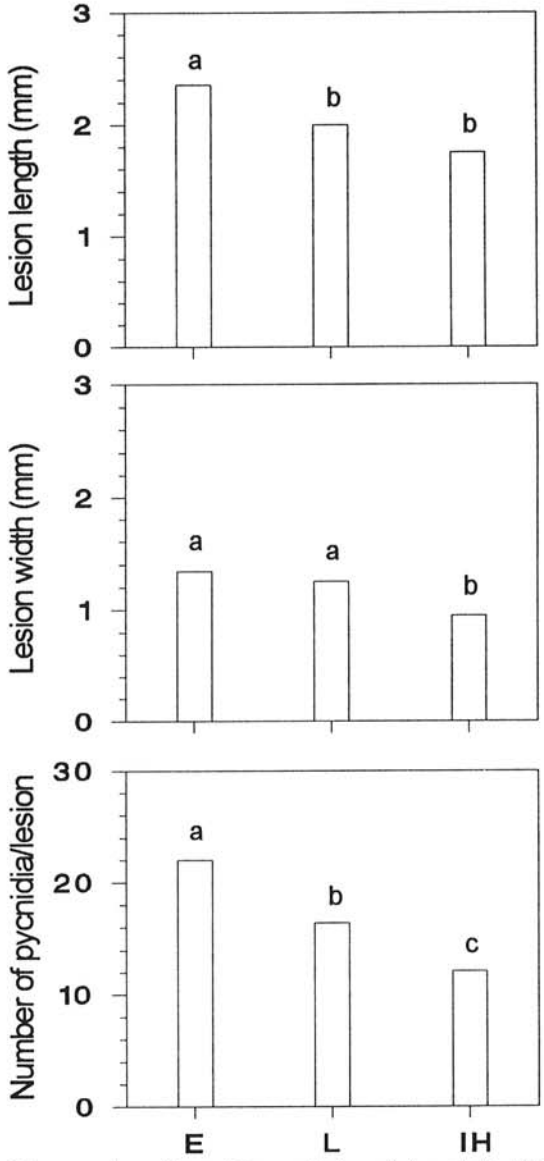

Fig. 2. Mean number of pycnidia per lesion of Ascochyta blight and mean lesion length and width in leaflets of lentil cvs. Eston (E), Laird (L), and Indianhead (IH). Each bar indicates the mean of the 24-, 48-, and 72-h treatments (36 pots total, with five seedlings per pot). Bars with the same letter are not significantly different at $P = 0.05$ according to Duncan's new multiple range test.

Overall, leaflet lesions in Eston were longer than those in Laird and Indianhead (Fig. 2). The widths of lesions in Eston and Laird were similar to each other but greater than those in Indianhead. A higher number of pycnidia per lesion was produced in Eston than in Laird, and the lowest number was produced in Indianhead.

Temperature. Temperature had substantial effects on the incubation and latent periods of all three cultivars; these effects were well described by quadratic equations (Fig. 3). Second-degree polynomial regressions (SAS) indicated that all of the estimated parameters were significant ($P < 0.05$), and in all cases the coefficient of determination (r^2) was 0.995 or higher. Plots of standardized residuals showed a random scatter. The shortest incubation periods, 5.7 days for Eston and 6.0 days for Laird and Indianhead, occurred at 20 C. The lengths of these periods increased at 25 C and were more than twice as long at 10 C. In general, the latent period was 1–2 days longer than the incubation period.

Temperature and cultivar significantly influenced infection frequency on leaflets in all three cultivars, but their interaction did not (Table 1). Only the linear component of the orthogonal polynomials was significant for temperature, indicating that the number of lesions on leaflets of all cultivars decreased linearly with increasing temperature from 10 to 25 C (Fig. 4). The highest numbers of leaflet lesions were produced in Eston at all temperatures except 10 C, where a similar number was produced in Laird and Eston. The lowest numbers of lesions were produced in Indianhead at 10, 15, and 20 C. Both temperature and cultivar had a significant effect on infection frequency on stems (Table 1). Furthermore, the temperature-quadratic × cultivar interaction was significant. Thus, the response of stem infection to temperature was quadratic in Eston and Laird, but there was no response to temperature in Indianhead (Fig. 4). The infection frequency in Eston and Laird increased from 10 to 15 C, then decreased from 15 to 25 C, but was always highest in Laird. In Indianhead, infection frequency was very low at all four temperatures.

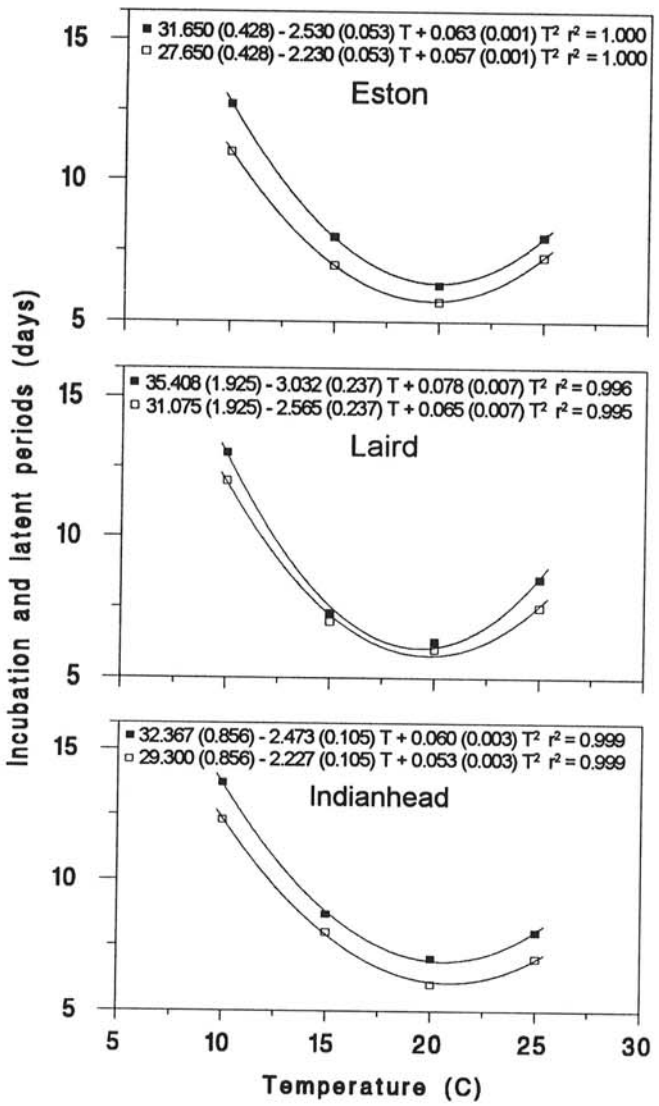

Fig. 3. Effect of temperature after inoculation on mean incubation (□) and latent (■) periods in three lentil cultivars inoculated with *Ascochyta fabae* f. sp. *lentis*. Means are based on three replicates. The polynomial regression equations were incubation or latent period = a (SE) + b_1T (SE) + b_2T^2 (SE), where incubation and latent periods are the number of days to first symptoms and first pycnidia, respectively, a is the intercept, b_1 and b_2 are regression coefficients, T is temperature (C), and SE is the standard error of the estimated parameter.

TABLE 1. Split-plot analyses[w] of variance and orthogonal polynomials for number of lesions of Ascochyta blight in leaflets and stems of three lentil cultivars incubated at four temperatures after inoculation

Source	df	Mean square (×10³) Leaflet	Stem
Temperature (T)	3	2,453**[x]	472*
T_{Linear}	1	6,000**	896*
$T_{Quadratic}$	1	1,273	499[y]
T_{Cubic}	1	49	21
Error A [Rep (T)]	8	301	113
Cultivar (C)	2	328**	1,476**
T × C	6	15	62*
$T_{Linear} \times C^z$	1	...	213**
$T_{Quadratic} \times C^z$	1	...	125*
Residual	4	...	8
Error B	16	29	16

[w] Data transformed to $x' = \log(x + 1)$ before analysis.
[x] * = Significant at $P = 0.05$, ** = significant at $P = 0.01$.
[y] Significant at $P = 0.06$.
[z] C = comparison of cvs. Eston and Laird with Indianhead.

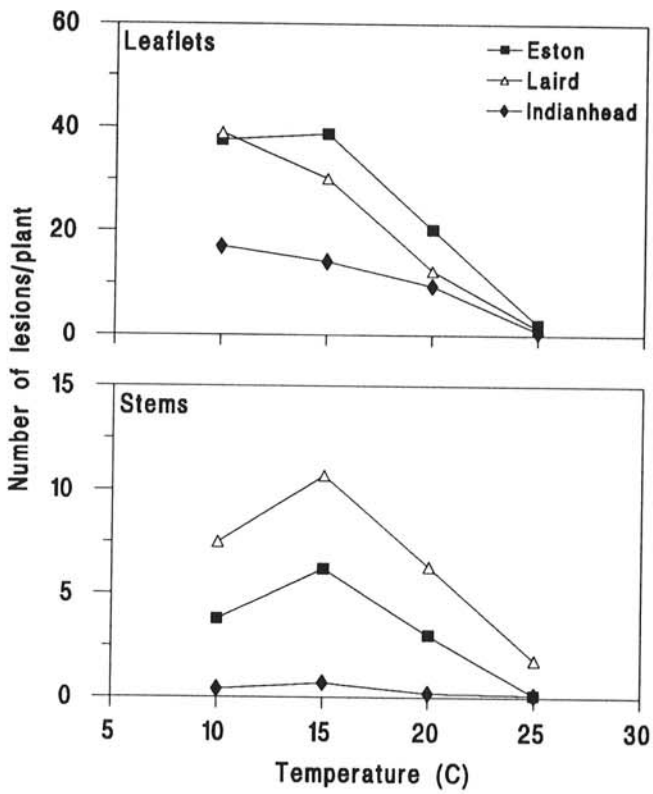

Fig. 4. Effect of temperature after inoculation on the number of lesions of Ascochyta blight in leaflets and stems of lentil cvs. Eston, Laird, and Indianhead. Data were transformed to $x' = \log(x + 1)$ before analysis. Each point indicates the detransformed mean of 60 plants.

The number of lesions on leaflets in the 25 C treatment was low on all cultivars, too low to provide adequate estimates of lesion length and width and number of pycnidia per lesion. Therefore, analyses did not include data from this temperature treatment. Temperature had no effect on length and width of leaflet lesions or on the number of pycnidia per leaflet lesion (Table 2). However, cultivar significantly affected all three variables, and a significant interaction between temperature and cultivar was observed for lesion length and number of pycnidia per lesion. The significant interactions resulted from a different response in Laird from that in Eston or Indianhead at 10 C. At 15 and 20 C, the longest lesions and highest number of pycnidia per lesion were found in Eston and the shortest lesions and lowest number of pycnidia were found in Indianhead (Fig. 5). At 10 C, the shortest lesions and lowest number of pycnidia were found in Indianhead, whereas lesion length and number of pycnidia per lesion were the same in Laird and Eston. A similar trend was observed for width of leaflet lesions, although the interaction was not significant (Table 2).

The length and width of stem lesions and the number of pycnidia per stem lesion were affected by cultivar but not by temperature or by the interaction of temperature and cultivar (Table 2). As indicated previously, data from the 25 C treatment and for Indianhead at all temperature treatments were excluded from the analyses. When averaged over the 10, 15, and 20 C treatments, stem lesions in Laird were longer and wider and had more pycnidia than those in Eston (Fig. 6).

Growth stage. In the growth chamber and field experiments, growth stage had little effect on the length of the incubation and latent periods in the three cultivars. Incubation and latent periods were 6.0–7.5 and 7.5–9.0 days, respectively, but were not consistent for a given treatment. Infection rarely occurred in the older tissues of the plants; it was almost exclusively restricted to tissues above the top four or five nodes of the main stem and secondary branches.

Growth stage, cultivar, and their interactions significantly affected disease severity on the top four nodes of the main stem in both experiments. Disease severity was higher at either the vegetative or flowering stages than at the podding stage for Eston and Laird (Table 3). A decrease in severity at the podding stage was also observed in Indianhead, although the response was smaller and was not statistically significant. This smaller response resulted in significant interactions. In the growth chamber experiment, disease severity at the vegetative and flowering stages was highest in Eston, intermediate in Laird, and lowest in Indianhead. In the field, disease severity at flowering was similar in Eston and Laird and lowest in Indianhead. At the podding stage, severity was low in all three cultivars, particularly in the field.

Growth stage also significantly affected the incidence of diseased leaflets and the number of lesions on leaflets and stems. However, the interactions of growth stage and cultivar were not significant.

TABLE 2. Split-plot analyses of variance for length and width of, and number of pycnidia in, lesions of Ascochyta blight in leaflets and stems of three lentil cultivars incubated at three temperatures after inoculation

Source	df	Mean square		
		Lesion length	Lesion width	Pycnidia per lesion
Leaflet				
Temperature (T)	2	27.9	7.9	1,092.1
Error A [Rep (T)]	6	24.0	5.5	767.2
Cultivar (C)	2	442.8**[z]	123.1**	3,941.5**
T × C	4	26.5**	3.7	197.0**
Error B	14	4.2	1.9	30.8
Stem				
Temperature (T)	2	39.7	0.96	1.6
Error A [Rep (T)]	6	61.7	3.96	37.8
Cultivar (C)	1	190.8**	5.67**	123.2**
T × C	2	0.3	0.19	2.1
Error B	6	7.8	0.2	4.3

[z] ** = Significant at $P = 0.01$.

When averaged over cultivar, incidence of diseased leaflets and number of leaflet and stem lesions were highest on plants at either the vegetative or flowering stage and lowest on those at the podding stage (Table 3). In the growth chamber, incidence of diseased leaflets and number of leaflet lesions were higher on Eston than on Laird and Indianhead and stem lesions were highest on Eston and Laird and lowest on Indianhead. Similar trends were observed in the field.

DISCUSSION

Wetness after inoculation was critical for leaflet and stem infection, but the duration had little effect on subsequent development of Ascochyta blight, i.e., incubation period, latent period, lesion size, or pycnidial production. The longest wetness period

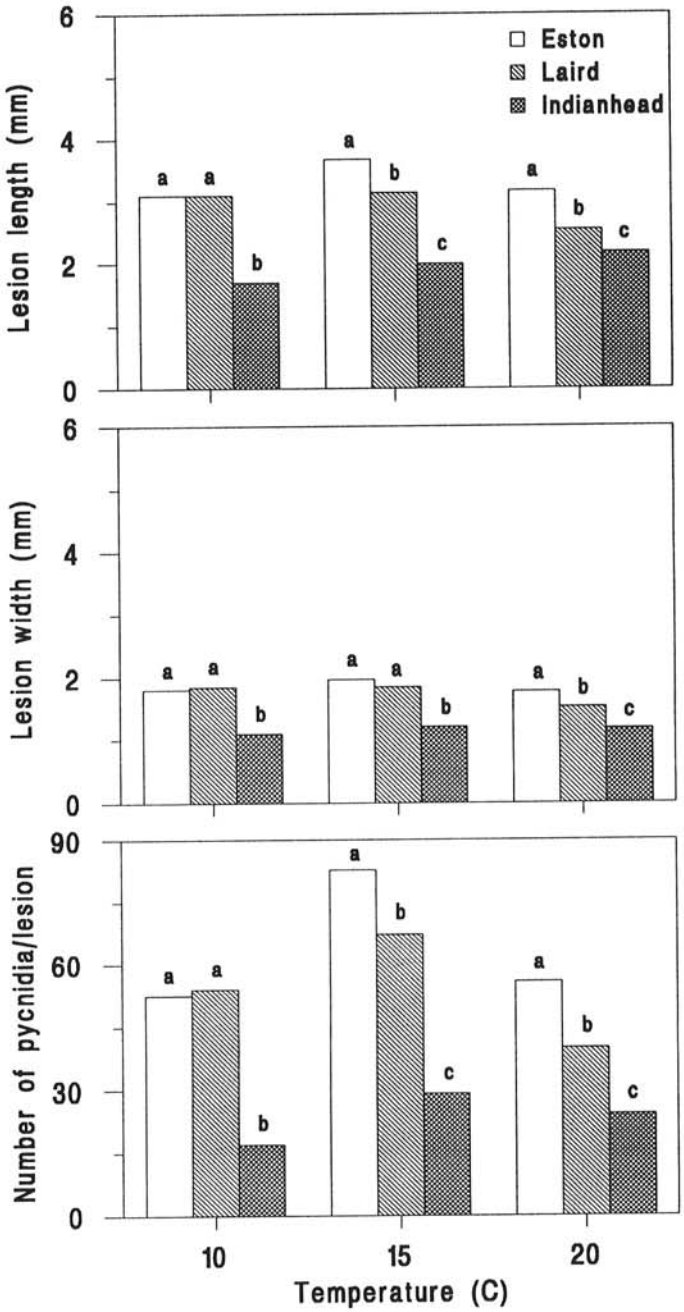

Fig. 5. Effect of temperature after inoculation on mean length and width of Ascochyta blight lesions and mean number of pycnidia per lesion in leaflets of lentil cvs. Eston, Laird, and Indianhead. Each bar indicates the mean of 60 plants. Within each temperature, bars with the same letter are not significantly different at $P = 0.05$ according to Duncan's new multiple range test.

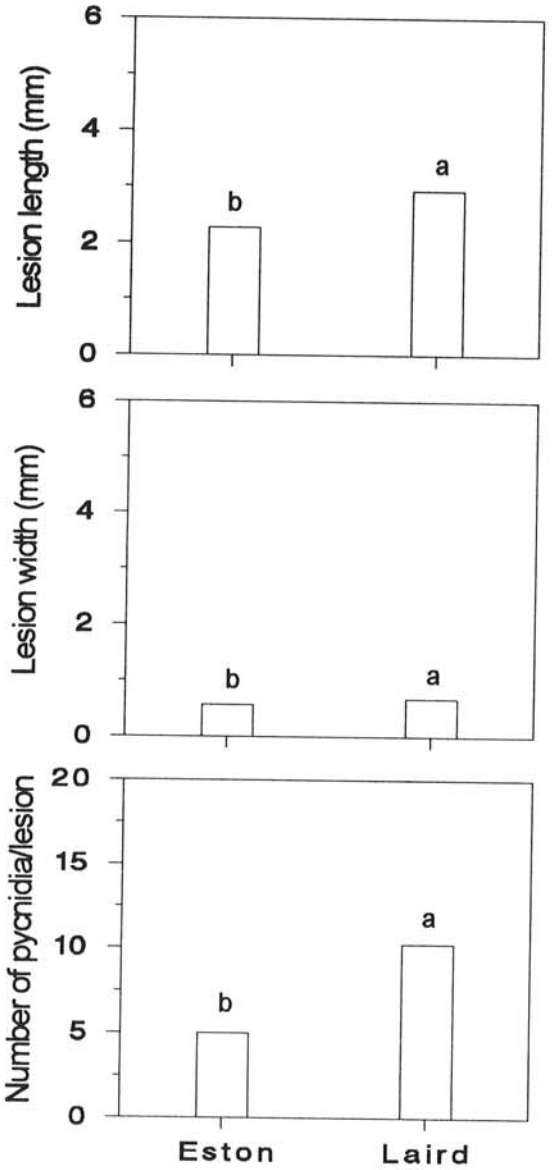

Fig. 6. Mean length and width of Ascochyta blight lesions and mean number of pycnidia per lesion in stems of lentil cvs. Eston and Laird. Each bar indicates the mean of 180 plants, and those with the same letter are not significantly different at $P = 0.01$ according to analysis of variance.

was 72 h, which was long enough to affect prepenetration development and penetration but probably too short to have much effect on postpenetration development. At least 6 h of leaf wetness was required for infection, and the optimal period for all three cultivars was 24–48 h. The interaction between temperature and leaf wetness was not examined, but the optimal wetness period may change with temperature. In chickpea, longer periods of leaf wetness are required for significant infection by *A. rabiei* (Pass.) Labrousse at temperatures lower or higher than 20 C (16). The reduction in infection that occurred with a 72-h wetness period may have resulted from longer exposure of the germinated spores to higher daytime temperatures or from plant stress. Seedlings exposed to 72 h of wetness often appeared wilted, probably because radiant heating raised the temperature inside the plastic chambers 4–5 C above that in the growth chamber. Compensating for the temperature differential by decreasing the temperature in the growth chamber was not possible because seedlings in different treatments were in the plastic chambers for different periods.

Temperature had large effects on leaflet and stem infection and on subsequent development of Ascochyta blight, particularly in the susceptible Eston and the moderately resistant Laird. Lesion counts indicated that the optimal temperature for infection ranged from 10 to 15 C and that very little infection occurred at 25 C. Regression equations predicted the shortest incubation period as 5.9 days at 20 C for Eston and Laird and at 21 C for Indianhead. The shortest predicted latent periods for Eston, Laird, and Indianhead were 6.3, 6.0, and 6.9 days at 20, 19, and 21 C, respectively. At 10 C, the predicted incubation and latent periods were much longer, respectively, 11.1 and 12.7 days for Eston, 11.9 and 12.9 days for Laird, and 12.3 and 13.6 days for Indianhead. Latent periods observed in the growth chamber at 15 C were similar to those observed in the field experiment under cool, moist conditions.

Although lesions appeared faster and pathogen reproduction occurred faster at 20 C, temperatures from 10 to 20 C had little effect on lesion size or on the number of pycnidia per lesion. A temperature of 25 C appeared to be less favorable for disease development than the lower temperatures. Leaflet lesions at 25 C ranged from 1.3 to 1.5 mm long and 0.8 to 1.0 mm wide with only three to six pycnidia per lesion. However, these means were based on few lesions and so were not analyzed with the data collected at 10, 15, and 20 C.

The resistance to Ascochyta blight in Indianhead reduced infection frequency, lesion size, and pycnidial production but had little effect on the incubation and latent periods. The level of resistance in Laird was lower than that in Indianhead and was not always evident, particularly at 10 C. When resistance in Laird was evident, it was also through reduced infection frequency, lesion size, and pycnidial production. The resistance in Indianhead was expressed in leaflets and especially in stems, whereas the

TABLE 3. Effect of growth stage on disease severity, incidence of diseased leaflets, and number of lesions on the top four nodes of the main stems of three lentil cultivars in the growth chamber and in the field at Saskatoon, 1993

	Severity (%)				Incidence (%)				Number of lesions							
									Leaflets				Stems			
Cultivar	Veg[y]	Flw	Pod	Mean	Veg	Flw	Pod	Mean	Veg	Flw	Pod	Mean	Veg	Flw	Pod	Mean
Growth chamber																
Eston	38 a[z]	36 a	9 b	28 A	61 a	63 a	25 b	50 A	62 a	61 a	18 b	47 A	14 a	15 a	7 b	12 A
Laird	14 a	11 a	3 b	9 B	42 a	32 ab	14 b	29 B	43 a	32 a	9 b	28 B	18 a	14 a	6 b	13 A
Indianhead	6 a	3 a	2 a	4 C	31 a	23 ab	10 b	21 B	28 a	20 a	6 a	18 B	1 a	1 a	1 a	1 B
Mean	19 a	17 a	5 b		45 a	39 a	16 b		44 a	38 a	11 b		11 a	10 a	5 b	
Field																
Eston		12 a	2 b	7 AB		31 a	2 b	17 A						3 AB		
Laird		14 a	1 b	8 A		28 a	1 b	15 A						6 A		
Indianhead		3 a	2 a	2 B		7 a	1 a	4 A						1 B		
Mean		10 a	2 b			22 a	1 b							3		

[y] Veg = vegetative, Flw = flowering, and Pod = podding.
[z] Values followed by the same lowercase letter in rows or uppercase letter in columns are not significantly different at $P = 0.05$ according to Duncan's new multiple range test.

resistance in Laird was expressed only in leaflets. Thus, resistance in stems and leaves may be governed by different genes. Stems of Laird were actually more susceptible to infection than those of Eston. The relative epidemiological importance of stem lesions in this disease is not known. However, unlike infected leaflets, which are quickly shed from the plant (12), infected stems remain in the canopy and may act as a source of inoculum for a much longer period.

Gossen and Morrall (4) and Tay (15) suggested that the moderate resistance of Laird breaks down at late podding. An increase in susceptibility with increasing plant age has also been reported for Ascochyta blight in chickpea (16). Conversely, in this study, not only did the resistance of Laird and Indianhead increase at the podding stage, but the susceptible Eston also became more resistant. This resistance greatly reduced infection frequency in leaflets and stems but had little effect on incubation and latent periods. It is not known if lesion size and pycnidial production were affected, as they were not measured. However, plants inoculated at the podding stage in both the growth chamber and the field showed a lower incidence of diseased leaflets and much lower disease severity than plants inoculated at the flowering stage. Resistance appeared to be related to tissue age, since inoculation of plants at the vegetative or flowering stages often resulted in infection of the youngest tissues (the top four or five nodes of the main stem and secondary branches) but rarely of the older plant tissues. A similar phenomenon has been reported for blast disease on rice (13). In lentil, this resistance probably becomes more apparent at the podding stage because vegetative growth slows during pod filling and the proportion of young tissue in the plant is reduced. The apparent breakdown of resistance in Laird at the podding stage is probably related to cultivar maturity. The earlier maturing Eston reaches the podding stage and becomes more resistant to Ascochyta blight 10–14 days earlier than Laird. Thus, when conditions are favorable, the disease may continue to increase on Laird and may result in a higher level of disease than on Eston.

LITERATURE CITED

1. Beauchamp, C. J., Morrall, R. A. A., and Slinkard, A. E. 1986. The potential for control of ascochyta blight of lentil with foliar-applied fungicides. Can. J. Plant Pathol. 8:254-259.
2. Beauchamp, C. J., Morrall, R. A. A., and Slinkard, A. E. 1986. Effects of scheduling applications of benomyl, captafol and chlorothalonil on ascochyta blight of lentil. Can. J. Plant Pathol. 8:260-268.
3. Bedi, S. 1990. Sources of inoculum and management of *Ascochyta fabae* f. sp. *lentis* on lentil. M.Sc. thesis. University of Saskatchewan, Saskatoon.
4. Gossen, B. D., and Morrall, R. A. A. 1983. Effects of ascochyta blight on seed yield and quality of lentils. Can. J. Plant Pathol. 5:168-173.
5. Gossen, B. D., and Morrall, R. A. A. 1984. Seed quality loss at harvest due to ascochyta blight of lentil. Can. J. Plant Pathol. 6:233-237.
6. Gossen, B. D., and Morrall, R. A. A. 1986. Transmission of *Ascochyta lentis* from infected lentil seed and plant residue. Can. J. Plant Pathol. 8:28-32.
7. Horsfall, J. G., and Barratt, R. W. 1945. An improved grading system for measuring plant diseases. (Abstr.) Phytopathology 35:655.
8. Kaiser, W. J., and Hannan, R. M. 1986. Incidence of seedborne *Ascochyta lentis* in lentil germ plasm. Phytopathology 76:355-360.
9. Martens, J. W., Seaman, W. L., and Atkinson, T. G. 1988. Diseases of Field Crops in Canada. Rev. ed. Canadian Phytopathological Society, Harrow, ON.
10. Morrall, R. A. A. 1987. Evaluation of fungicides to reduce seed-to-seedling transmission of ascochyta. Page 217 in: Pesticide Research Report, Agriculture Canada, Ottawa.
11. Morrall, R. A. A. 1992. Significance of seed-borne inoculum of lentil pathogens in western Canada. Pages 313-314 in: Proc. Eur. Conf. Grain Legumes 1st.
12. Pedersen, E. A., Morrall, R. A. A., McCartney, H. A., and Fitt, B. D. L. 1994. Dispersal of conidia of *Ascochyta fabae* f. sp. *lentis* from infected lentil plants by simulated wind and rain. Plant Pathol. 43:50-55.
13. Roumen, E. C., Bonman, J. M., and Parlevliet, J. E. 1992. Leaf age-related partial resistance to *Pyricularia oryzae* in tropical lowland rice cultivars as measured by the number of sporulating lesions. Phytopathology 82:1414-1417.
14. Steel, R. G. D., and Torrie, J. H. 1980. Principles and Procedures of Statistics: A Biometrical Approach. McGraw-Hill, New York.
15. Tay, J. 1989. Inheritance of resistance to ascochyta blight in lentil. M.Sc. thesis. University of Saskatchewan, Saskatoon.
16. Trapero-Casas, A., and Kaiser, W. J. 1992. Influence of temperature, wetness period, plant age, and inoculum concentration on infection and development of Ascochyta blight of chickpea. Phytopathology 82:589-596.