Alterations in Lipid Composition of Seed Oil from *Brassica juncea* upon Infection by *Cuscuta reflexa*

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Among four varieties of Indian mustard, Sarson T42 was found more susceptible than Lahi T9 to infection by *Cuscuta reflexa*, whereas Varuna-59 and Rai Pusa Bold showed no apparent response. The infection by *Cuscuta* resulted in decrease in oil content of Sarson T42 and Lahi T9 by 84 and 36% per plant, respectively. A significant increase in free fatty acids and free sterols and decrease in total neutral glycerides of oil were also observed. The alteration in lipase activity induced by *Cuscuta* infestation was dependent on the developmental stage of seed. Although total fatty acid content decreased insignificantly, the fatty acid composition of oil showed appreciable differences upon infection. The percentage unsaturation of fatty acids in Sarson T42 and Lahi T9 was decreased by 23 and 12%, respectively, on infection by *Cuscuta*. Conclusively, in addition to a decrease in oil content, the quality (composition) of the oil of susceptible mustard varieties becomes poorer upon infection by *Cuscuta*.

INTRODUCTION

Chemical characteristics as well as fatty acid composition govern oil quality. Photoperiod, temperature, cultivar, location, edaphic, and seasonal variation as well as herbicide application (Wilkinson, 1970; Wilkinson and Hardcastle, 1971, 1972a-c, 1973, 1974; Hardcastle et al., 1974) influence triglyceride and fatty acid composition and quantity of the oil. Fatty acid composition is thus almost totally dependent upon the physiological condition of the plant. There are several papers available on alterations in seed yield, oil content, and oil quality of oil seed bearing plants as a consequence of various infectious diseases (Zimmer and Zimmerman, 1972; Zazzerini et al., 1985, 1987; Dart et al., 1987). Recently, changes in physicochemical components and fatty acid composition of sunflower oil due to *Sclerotium bataticola* infection have been reported by Conte et al. (1989). Studies conducted in this laboratory revealed alterations in lipid composition of host plants upon infection by *Cuscuta*, an angiosperm parasite (Mattoo and Mattoo, 1977; Sharma et al., 1985, 1986). It is a long-range problem and biologically very interesting. The present investigation was undertaken to determine the effect of parasitism by *Cuscuta reflexa* on yield, chemical constituents, and fatty acid composition of the oil of Indian mustard (*Brassica juncea*), an abundantly grown winter crop in India.

MATERIALS AND METHODS

Brassica juncea (Indian mustard) L. Cvs. Sarson T42, Lahi T9, Varuna 59, and Rai Pusa Bold belonging to the Cruciferae family were used as hosts for *C. reflexa* Roxb. Plants were grown in pots in garden soil from October to March. Twelve pots of each variety were selected. Half of the plants were infected with *Cuscuta*, and half served as controls (healthy ones). The infection was done before flowering [i.e., 31 days after sowing (DAS)] by twining about 20 cm of the parasite vine anticlockwise around the stem of the host plants. Growth of the parasite vine was well established in 2-5 days as evident by the formation and penetration of haustoria. The healthy plants were grown in close vicinity of the infected plants under similar environmental conditions. In view of no apparent response to infestation, Varuna 59 and Rai Pusa Bold varieties were not sampled for analysis.

Five stages of development were chosen to perform the present investigation. Those were 68, 75, 82, and 89 DAS and fully mature seed stage. Seed pods were removed from the plants, packed in polythene bags, kept in ice, and brought to laboratory. Seeds were immediately separated from the pods and subjected to freeze-drying. The freeze-dried seed samples were stored under N_2 in dry screw-capped tubes, which were kept in a desiccator at -20 °C until analysis.

For each set, 10 g of mature seeds was ground to break the seed coat and subjected to Soxhlet extraction with 150 mL of *n*-hexane for 6 h (Paquot, 1979). The extract was concentrated to almost dryness under reduced pressure, dissolved in a minimum amount of $CHCl_3$, and stored at -20 °C under N_2. The oil content was determined by drying an aliquot of the chloroform extract in a vacuum oven overnight and weighing the lipid residue.

Total neutral glyceride (tri-, di-, and mono-) content of the oil from mature seeds was determined according to a modified method of Van Handel and Zülversmit (1957). An oil sample (20-30 mg dissolved in $CHCl_3$) was quantitatively taken into a side arm flask. After $CHCl_3$ was evaporated under a stream of N_2, 5 mL of 10% ethanolic KOH was added and the content refluxed for 5 min. The hydrolysate was acidified to pH 2.0 with 1 M H_2SO_4. Excess ethanol was removed by heating the contents for 2 min in a boiling water bath. The sample was washed and eluted three or four times with benzene. The organic and aqueous phases were employed for the estimation of total fatty acids and neutral glycerides, respectively. To suitable aliquots of the aqueous phase was added water to make the volume up to 1 mL, followed by 0.5 mL of 0.01 M sodium metaperiodate. After 10 min, 0.5 mL of 20% sodium sulfite and 8 mL of chromotropic acid reagent were added and the contents mixed and heated in a boiling water bath for 30 min. The blank (without neutral glyceride) and the standard (tripalmitin) were treated in a similar manner, and absorbance was measured at 570 nm.

The method of Lowry and Tinsley (1976) was employed to determine total fatty acids as well as free fatty acids (FFA) in the oil sample. To suitable aliquots of organic phase was added benzene to 5 mL, followed by the addition of 1 mL of cupric acetate-pyridine reagent. The contents were mixed in a cyclomixer for 2 min and then centrifuged. The upper layer was separated and absorbance read at 650 nm in a spectrophotometer. Palmitic acid was used as a reference standard.

Total free sterol content in the oil was determined according to the protocol of Stadtman (1957). To 5-mL samples in $CHCl_3$ (40-50 mg), taken in dry test tubes, was added 2 mL of ice-cooled acetic anhydride-sulfuric acid (4:1 v/v) slowly with constant stirring. Tubes were placed in the dark at 20 °C for 20 min, and absorbance was measured at 625 nm. Cholesterol was used as a standard.

For determination of fatty acid composition of oil, the organic phase containing total fatty acids was subjected to methylation as described by Jham et al. (1982). Fatty acid methyl esters

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Table I. Morphological and Chemical Constituents of Healthy Mustard Plants and Plants Infected with *C. reflexa*

parameter[a]	Sarson T42			Lahi T9		
	healthy plants	infected plants	% inc (+) or dec (−) over healthy	healthy plants	infected plants	% inc (+) or dec (−) over healthy
pods/seed	292 ± 8	152 ± 5[b,c]	−48	185 ± 10	162 ± 9[d]	−13
seeds/pod	31 ± 2	16 ± 2[c]	−49	24 ± 2	20 ± 3[d]	−16
wt of 100 pods, g	36.8 ± 1.88	29.4 ± 1.05[c]	−20	30.0 ± 1.10	25.6 ± 1.50[d]	−15
oil content, g/plant	16 ± 2.5	2.0 ± 0.05[c]	−84	5.0 ± 0.08	3.2 ± 0.07[c]	−36
oil content, mg/seed	1.8 ± 0.07	1.0 ± 0.08[c]	−45	1.13 ± 0.03	0.98 ± 0.03[d]	−13
oil content, mg/g of dry wt of seed	412 ± 20	320 ± 21[c]	−22	421 ± 19	378 ± 20[e]	−10
total neutral glyceride, % of oil	97.4 ± 3.40	76.9 ± 1.80[c]	−21	95.1 ± 3.20	75.7 ± 2.10[c]	−20
total fatty acids, % of oil	88.5 ± 3.30	77.0 ± 1.20[d]	−13	87.1 ± 3.10	77.6 ± 1.10[d]	−11
free fatty acids, % of oil	1.2 ± 0.20	8.1 ± 0.60[c]	+575	1.5 ± 0.30	9.5 ± 0.90[c]	+533
free sterols, % of oil	1.4 ± 0.20	13.9 ± 0.90[c]	+893	3.3 ± 0.20	14.8 ± 0.80[c]	+350

[a] Studies in fully mature pods. [b] Mean ± standard deviation of three sets with four replicates in each set. [c] Significant at $P \leq 0.001$. [d] Significant at $P \leq 0.05$. [e] Difference insignificant.

Table II. Lipase Activity[a] in Developing Seeds from Healthy and *C. reflexa* Infected Mustard Plants

DAS[b]	Sarson T42			Lahi T9		
	healthy plants	infected plants	% inc over healthy	healthy plants	infected plants	% inc over healthy
68	70.4 ± 8.5	75.9 ± 9.2[c,d]	7.8	65.6 ± 8.2	71.6 ± 7.5[d]	9.1
75	73.5 ± 8.6	564.3 ± 15.4[e]	643	63.5 ± 8.5	511.0 ± 16.0[e]	705
82	72.2 ± 8.2	625.0 ± 22.5[e]	766	62.8 ± 9.0	572.2 ± 13.5[e]	811
89	70.6 ± 7.8	475.8 ± 13.7[e]	574	61.1 ± 7.3	450.0 ± 12.2[e]	636

[a] Expressed as nanomoles of fatty acids released per hour per seed. [b] Days after sowing. [c] Mean ± standard deviation of three sets with triplicates in each set. [d] Difference insignificant. [e] Significant at $P \leq 0.001$.

(FAME) were separated and detected by AIMIL-Nucon gas liquid chromatograph using 20% diethylene glycol–succinate (DEGS) on Chromosorb W (100–120 mesh) coated in a stainless steel column (1.8 × 2 mm i.d.). The operating conditions were as follows: oven temperature, 190 °C; flame ionization detector (FID) and injector temperatures, 220 °C; flow rate of H_2 and N_2, 30 mL/min. The identification of FAME was carried out by comparing their relative retention times (RRT) with those of standard FAME obtained from Sigma Chemical Co. as well as by using standard fatty acyl esters as internal standards. The peak area was calculated by measuring the height multiplied by the width of the peak at half the peak height. The values for each fatty acid are given as percent by weight of total fatty acids.

Lipase activity was measured spectrophotometrically by following the protocol of Schmidt et al. (1974). Olive oil suspension [5% (w/v) in 0.89% NaCl solution inclusive of 5% (w/v) gum acacia], sodium tauroglycocholate (10 mM), and triethanolamine hydrochloride buffer (1 M, pH 8.5) were thoroughly mixed in the ratio 50:5:45 (v/v) and equilibrated to 34 °C. To 0.1 mL of this suspension were added 0.5 mL of homogenate (prepared by grinding 100 seeds in 10 mL of homogenizing medium consisting of 0.6 M sucrose, 1 mM EDTA, 10 mM KCl, 1 mM $MgCl_2$, 2 mM DTT, and 0.15 M phosphate buffer, pH 7.5) and water, and incubation was carried out for 2 h at 34 °C in a water bath equipped with shaker. The control tubes contained heat-inactivated seed homogenate. The reaction was stopped by keeping the tubes in a boiling water bath for 2 min. After cooling, 5 mL of chloroform and 2.5 mL of copper reagent were added, and the contents were cyclomixed for 2 min and centrifuged. Suitable aliquots were taken from the lower organic phase, and the volume was made up to 3 mL with $CHCl_3$ followed by the addition of 0.25 mL of sodium diethyldithiocarbamate (11 mM in *sec*-butanol) and thorough shaking. After 2 h, absorbance was read at 440 nm in a spectrophotometer. Stearic acid was used as the standard. The lipase activity was expressed as nanomoles of fatty acids released per hour per seed.

The statistical significance of the difference between the healthy and infected sets was calculated with the help of Student's *t*-test.

RESULTS

The infection of the host plants by *Cuscuta* was dependent on the variety of the mustard plant. Sarson T42 and Lahi T9 were susceptible; Varuna 59 and Rai Pusa Bold varieties appeared to show no apparent response to infection, and the parasite did not produce attached haustoria. Of the susceptible varieties, Sarson T42 was more severely affected. Number of pods per plant and number of seeds per pod registered a highly significant decrease of about 50% of Sarson T42; a 15% decrease was seen for Lahi T9. Pods and seeds harvested from infected plants were smaller than pods and seeds from healthy plants. Pods (100) from infected Sarson T42 weighed 20% less and those from infected Lahi T9 15% less than the pods of the respective healthy plants. The decrease in oil content of Sarson T42 consequent upon infection by *Cuscuta* was 84, 45, and 22%, on a per plant, per seed, and per gram of dry weight basis, respectively. Lahi T9 also responded to infection with decreases in oil content of 36, 13, and 10%, respectively, on a per plant, per seed, and per gram of dry weight basis. The total fatty acid contents of Sarson T42 and Lahi T9 seed oil declined insignificantly as a result of parasitism by *Cuscuta*. FFA and sterol contents increased significantly, while total neutral glyceride content registered a significant decrease upon parasitization by *Cuscuta* (Table I).

As a consequence of infection, the alteration in lipase activity was negligible in seeds harvested at 68 DAS. For seeds harvested at 75, 82, and 89 DAS, the lipase activity registered a significant enhancement, with maximum for 82 DAS seeds (Table II).

Table III shows that the parasitism by *Cuscuta* resulted in highly significant increases in the percentage of palmitic, stearic, arachidic, behanic, oleic, and linoleic acids and decreases in palmitoleic, linolenic, eicosenoic, and erucic acids in both varieties of mustard, although the alteration in eicosenoic and erucic acid was insignificant. The percentage unsaturation of fatty acid decreased by 23 and 12% in Sarson T42 and Lahi T9, respectively, upon infection by *Cuscuta*.

DISCUSSION

C. reflexa is an angiospermic shoot parasite that causes morphological and metabolic perturbation in susceptible hosts. The number of flowers per plant of two varieties of Indian mustard, namely Sarson T42 and Lahi T9, was

Table III. Fatty Acid Composition (Percent of Total Fatty Acid) in Mustard Oil Extracted from Healthy and *C. reflexa* Infected Plants

	Sarson T42			Lahi T9		
fatty acid	healthy plants	infected plants	% inc (+) or dec (−) over healthy	healthy plants	infected plants	% inc (+) or dec (−) over healthy
myristic	0.9 ± 0.10	1.20 ± 0.21[a,b]	+25	0.3 ± 0.08	0.4 ± 0.09[b]	+37
palmitic	0.4 ± 0.09	4.2 ± 0.40[b]	+950	0.5 ± 0.08	1.4 ± 0.21[b]	+192
palmitoleic	5.1 ± 0.41	0.3 ± 0.09[b]	−93	2.7 ± 0.20	0.5 ± 0.09[b]	−83
stearic	2.7 ± 0.24	4.3 ± 0.20[b]	+59	2.3 ± 0.40	4.2 ± 0.41[b]	+87
oleic	2.7 ± 0.20	4.4 ± 0.20[b]	+63	1.4 ± 0.21	3.5 ± 0.31[b]	+150
linoleic	3.3 ± 0.14	4.1 ± 0.40[b]	+25	1.4 ± 0.30	3.8 ± 0.50[b]	+173
linolenic	39.6 ± 2.60	27.5 ± 2.62[b]	−31	52.0 ± 2.80	41.9 ± 1.40[b]	−20
arachidic	2.4 ± 0.24	6.0 ± 0.40[b]	+150	2.2 ± 0.45	4.5 ± 0.30[b]	+100
eicosenoic	10.1 ± 1.10	9.1 ± 1.30[c]	−10	10.4 ± 1.05	9.2 ± 1.20[c]	−12
behanic	18.8 ± 2.00	26.9 ± 2.20[b]	+42	10.7 ± 0.45	15.2 ± 1.02[b]	+42
erucic	13.8 ± 0.34	12.1 ± 0.42[c]	−5	16.2 ± 0.21	15.5 ± 0.45[c]	−4
% unsaturation	74.6	57.5	−23	84.0	74.4	−12

[a] Mean ± standard deviation of three sets with triplicates in each set. [b] Significant at $P \leq 0.001$. [c] Difference insignificant.

reduced as a consequence of infection by *C. reflexa*. This postinfestation happening led to poor pollination and fertilization and resulted in a decrease in the number of pods and seeds. It was observed that the other two varieties of mustard, namely Varuna 59 and Rai Pusa Bold, did not respond to infection by *Cuscuta*, with almost complete absence of visible symptoms. Of the two susceptible varieties, Sarson T42 was more severely affected than Lahi T9. It is not clear what makes one variety of mustard more susceptible to infection by *Cuscuta* than another. After infestation, competition for water, inorganic ions, and metabolites with the parasite is the simplest explanation for losses in host production. Awasthi (1987) inferred that the waxy surface of the crop is probably helpful in resisting the mechanical penetration of pathogen and may be helpful in combating disease stress.

Reduction in oil content per plant was significant in Sarson T42 and Lahi T9. This reduction is probably due to reduction in the number of pods and seeds. In the case of Sarson T42, the oil content per seed also decreased significantly. This reflects metabolic perturbation induced by the parasite. The spherosomes, which are present in the cell before the onset of oleosome formation, are suggested to originate from the endoplasmic reticulum (Appelqvist, 1980). The sudden appearance of "nascent lipid droplets", which then, most likely, turn into oleosomes, seems to start the rapid accumulation of seed lipids in developing oil seeds. The reduction in oil content may be due to either retardation in development of spherosomes or inhibition of metabolic steps leading to oil synthesis.

The infection by *Cuscuta* led to a decrease in the total neutral glyceride (a storage energy enriched lipid) level which may be due to the partial hydrolysis of triacylglycerol by lipase during seed development as well as the decreased synthesis of triglycerides. The infection also caused increase in free fatty acids and free sterols.

In both of the susceptible varieties of mustard, the total fatty acid content did not alter significantly, although the variation in its composition was highly significant as a result of infection. The percentage unsaturation of fatty acid decreased in both varieties, although less in Lahi T9. It appears to us that *Cuscuta* is drastically altering the ability of the *Brassica* to dehydrogenate the linoleic acid moiety. The basic locus of influence might be microsomes [cf. Stumpf (1980)]. The percentage reduction in palmitoleic and linolenic fatty acids in the present case upon infection could be due to the reduced desaturation of the palmitoyl and linoleoyl moieties, respectively. The reduction in the activity of oleoyl-ACP elongase might result in decreased synthesis of eicosenoic and erucic acids.

The quality of oil and its use are determined by its physicochemical characteristics as well as its fatty acid composition (Downey and Harvey, 1963). In the present study, a decrease in the level of total neutral glycerides and percent unsaturation of total fatty acids and an enhancement in FFA and free sterols are indicators of lower quality of oil. The high concentration of free sterols in the diet may cause several abnormalities in the human body, viz. carcinogenic effects of steroids (Bischoff, 1969), atherosclerosis (Engelberg, 1983), and inhibition of sperm fertility (Go and Wolf, 1983). FFA appear to be oxidized more easily as compared to when the fatty acids are conjugated into triacylglycerol or any other acyl lipids (Gurr and James, 1971). The derived decomposition products of FFA concomitant with increased free sterol level appear to be injurious to health and such an oil needs an extra refining step. In addition, linolenic acid, one of the unsaturated essential fatty acids (Achaya, 1987), was reduced upon parasitization of mustard plant, suggesting that the quality of oil is rendered poorer upon infection by *Cuscuta*.

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Registry No. Lipase, 9001-62-1; myristic acid, 544-63-8; palmitic acid, 57-10-3; palmitoleic acid, 373-49-9; stearic acid, 57-11-4; oleic acid, 112-80-1; linoleic acid, 60-33-3; linolenic acid, 463-40-1; arachidic acid, 506-30-9; eicosenoic acid, 28933-89-3; behenic acid, 112-85-6; erucic acid, 112-86-7.