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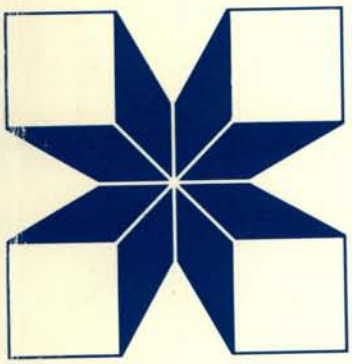

CANADA

OIL CROPS:
PROCEEDINGS OF THE THREE MEETINGS HELD AT PANTNAGAR AND HYDERABAD, INDIA, 4 – 17 JANUARY 1989

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La présente série est réservée aux documents issus de colloques, aux rapports internes et aux documents techniques susceptibles d'être publiés plus tard dans une série de publications plus soignées. D'un tirage restreint, le rapport manuscrit est destiné à un public très spécialisé.

Esta serie incluye ponencias de reuniones, informes internos y documentos técnicos que pueden posteriormente conformar la base de una publicación formal. El informe recibe distribución limitada entre una audiencia altamente especializada.

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OIL CROPS:
PROCEEDINGS OF THE THREE MEETINGS HELD AT PANTNAGAR AND HYDERABAD, INDIA, 4-17 JANUARY 1989

1. The Brassica Subnetwork-II
2. The Other Oil Crops Subnetwork-I
3. The Oil Crops Network Steering Committee-I

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THE PRESENT STATUS OF NIGER AND LINSEED PATHOLOGY RESEARCH WORK IN INDIA

G.S. Saharan

NIGER

Niger (*Guizotia abyssinica* Cass) is cultivated in India in an area of about 600,000 ha annually chiefly in Madhya Pradesh, Orissa, Maharashtra, Bihar, Karnataka and Andhra Pradesh, on hill tops and slops. Although a number of diseases are known to occur, Table 1, on this crop in India but no systematic approach has been attempted to investigate any disease in detail. The information is available only on the occurrence and symptomatology of the diseases. As regards management, seed treatment with Thiram @0.3% has been found to protect seed rots along with higher seed germination. Spraying of Zineb @ 0.2% is useful for the control of leaf spot diseases (48, 49).

Table 1. Diseases of niger in India.

Disease	Cause
Powdery mildew	*Sphaerotheca* sp.
Leaf spot	*Cercospora guizoticola*
Wilt	*Ozonium texanum* var. *Parasiticum*
Root rot, stem rot and blight	*Rhizoctonia Solani*, *Macrophomina Phaseolina*, *Sclerotium rolfsii*, *Rhizoctonia bataticola*, *Alternaria alternata*
Seed rot	*Asperqillus* sp., *Cladosporium*, *Alternaria* sp. *Emericella* sp., *Fusarium* sp. *Phyllosticta* sp.
Bacterial leaf spot	*Xanthomonas campestris* pv. *guizotiae*
Phanerogamic parasite	*Cuscuta hyalina*

LINSEED

At present, we in India cultivate linseed in India is exclusively cultivated for seed in about 2 mill. ha of land which comes to about 25% of the total world acreage under this crop. Among rabi oilseed crops, linseed is next to rapeseed and mustard. It is an important oilseed crop of Uttar Pradesh, Madhya Pradesh, Maharashtra and Bihar, contributing more than 85% to its national acreage and production. The area under linseed is increasing in other states year by year. The diseases to which linseed is exposed and prone are many, Table 2, but Alternaria blight, rust, powdery mildew and wilt diseases are most damaging to this crop and have been investigated in depth.

Table 2. Diseases of linseed in India

Disease	Cause
Rust	*Melampsora lini*
Blight	*Alternaria lini*, *A. alternata*
Wilt	*Fusarium oxysporum f. lini* *Rhizoctonia bataticola*
Powdery mildew	*Oidium lini*
Root rot, stem rot and blight	*Macrophomina phaseolina* *Rhizoctonia solani* *Sclerotium rolfsii* *Sclerotinia sclerotiorum*
Leaf spot/Pasmo	*Colletotrichum lini* *Septoria linicola* (*Mycosphaerella linorum*)
Seed rot	*Aspergillus* sp. *Alternaria* sp. *Ascochyta linicola* *Botrytis* sp. *Cladosporium herbarum* *Colletotrichum lini* *Fusarium* sp. *Macrophomina* sp. *Polypora lini*
Phaneroganic parasite	*Cuscuta hylina*

Rust

Linseed rust caused by *Melampsora lini* (Ehrenb) Lev. causes severe epidemics year after year with losses estimated to range between 40 to 100% depending upon the amount of initial inoculum, time to first appearance of the disease and subsequent build up and dissemination of the pathogen. A reduction of 13.1% has been reported in oil content of heavily rusted plants (62).

Disease development

This has been studied mainly in relation to the prevalence of different races as a function of resgenes present in host cultivars and the number of unnecessary virulence genes present in the pathogen. Some studies have also been carried out to study the effect of light, temperature and humidity on uredospore germination, infection and disease development under field conditions.

Race prevalence in relation to virulence

It has been demonstrated (14) that races of flax rust attacking widely grown cultivars increased whereas races unable to do so, decreased. The predominant races were those carrying the smallest number of genes for virulence that permitted survival. In a recent study (56) relationship of virulence to race prevalence as hypothesized by Flor (14) could not be clearly established since races which were virulent on only one resgene as well as those virulent on 5 or 6 resgenes were equally rare.

Environment and disease development

Uredial development takes place at temperatures ranging from 7^0 to 30^{0}C with optimum at 16-22^{0}C (20). Infection was slight at 7-14^{0}C and at 26-30^{0}C. Subsequently, it was found (42) that temperature between 13^{0}C and 21^{0}C are most conductive to host infection and rust development, with symptoms appearing in 7 days. For infection of linseed by uredospores of *M.lini*, under controlled conditions of constant temperature and continuous leaf wetness, a temperature range of 15^0 to 25^{0}C was found optimum (47). Considerably cultivar specificity with regard to the effect of light intensity on incubation and latent periods was recorded (52).

Although under controlled conditions, the latent period of *M.lini* was influenced considerably by light and temperature, under field conditions it is subject to the influence of a large number of environmental factors which may have suppressive or additive effects on each other and hence on latent period. Analysis of simple correlation coefficients indicated that environmental factors like temperature (TEMP), mean relative humidity (MRH), RH, and cloudiness (CD) have a highly significant positive correlation with latent period whereas MTEMP had a highly significant negative correlation. The variation explained by any of the combinations of variables was of a high order. Even a single environmental factor like number of days with relative humidity $\geq$90% could account for more than 92% of the observed variation in latent period. Thus, under field conditions, the latent period of *M.lini* is greatly influenced by duration of relative humidity of 90% or more (53).

Studies on the periodic increase in the number of pustules per tiller under field conditions showed that MTEMP had a significant negative correlation with rust development. When partial regression coefficients were tested in different combinations for the progress of rust, the partial

regression coefficient for TEMP was significant in TEMP, MRH and CD and TEMP and CD combinations. The multiple regression equation build up from different combinations of variables for the progress of the disease with significant R^2 values explained the variation ranging from 39.9 to 61.5% (55).

Physiologic specialization

Physiologic specialization in *M.lini* on flax was first demonstrated by Flor (11), who developed a set of rust differentiating lines each supposedly carrying a single gene for resistance in the background of Bison susceptible to all the races occurring in North America by back-crossing (13,15). In the absence of availability of differential varieties developed by Flor the occurrence of 5 physiologic races based on reactions of 7 collections on 50 linseed cultivars was demonstrated in India (42). The information on the race-flora of linseed rust prevalent in India has been compiled (30, 36, 38, 42, 51, 58, 65). So far, 18 physiologic races have been found to occur in India. These have been designated, I-1 to I-17 and 43 in the order of their discovery as they do not correspond to any of the races mentioned in Flor's key except the later one. Out of these, 17 have been reported from Himachal Pradesh (38, 51, 58). Collectively, these races are virulent on resgenes L1 (Burke), L2(Stewart), L5(Wilden), L7(Barnes), L9(Bison), L10(Bolley Golden sel), M1 (Williston brown), M2(Ward), N(Bombay), P(Koto), and P1(Akmolinsk). On the basis of reaction to Bison, the Indian races can be separated into group A(I-1, I-3, I-4 and I-7) to which Bison is resistant and Group B(I-2, I-5 and I-6) to which Bison is susceptible.

Sources of resistance

Comprehensive screening of a large number of genotypes under artificial inoculation conditions against all the prevalent races of *M.lini* in India indicated LC 216, LC 255, LC 256, Ottawa 770B(L), Dakota(M), Vicotry A(M4), Tower (L8), Marshall (N2) and Pale blue crimped (L3) as excellent sources of resistance (51,53). Most of them carry single or few dominant genes for resistance to all the races in India and can be utilized for developing resistant cultivars. A large number of genotypes free from rust were reported (59,60). Amongst the recommended cvs. for growing in different regions of India (22), LC-54, Himalini, Jawahar-7, Jawahar-17, Jawahar-552 and JLS(J)-1 are resistant to rust. In AICORPO trials conducted for three years at different locations of India under UDN (uniform disease nursery), genotypes like LCK-38, LCK-59 and RLC-2 were found resistant (2).

Genetics of resistance

Studies on inheritance of resistance to *M.lini* in flax were initiated (12,13,21,40). In India, large number of cultivars have been screened against different races or with mixtures of races 8,9,28,34, 43,51,53). It was found that in cultivars, NP (RR) 262, NP (RR) 45 and NP (RR) 204, field resistance was controlled by one dominant gene, two complementary genes and two dominant genes, respectively (19,24,39,59). However, they did not use known races for these studies. In a subsequent study (25), resistance in NP (RR) 45 to race I-1 was found to be governed by two complementary genes. Genes for resistance to 5 Indian races of *M.lini* in the flax rust differentials and two allied species, *L.affricans* and *L.marginale* were determined (34,35). The differential cultivars Dakota, Ward, Cass, Victory-A, Polk and leona carry two resgenes operative against Indian

races, while Bowman has three. Other differentials were found to be monogenic for resistance to races under study.

Resistance in LC 255 to races I-8 and I-9c was governed by 2 dominant genes, whereas torace I-9c, it was due to a single dominant gene (51,53). In LC 256 resistance to races I-8 and I-9c was also governed by two dominant genes. In LC 215, resistance to races I-8 and I-9b was due to single dominant genes but to race I-9c it was due to two dominant genes. Reaction of F_2 population of a cross between LC 216 and LC 255 to sub-race I-9b indicated that the gene for resistance in LC 216 is different from the genes for resistance in LC 255. LC 216 and LC 255 have one parent in common, viz., EI5643. Thus, LC 216 and LC 255 which are resistant to all the races against which they were tested, possess different genes for resistance to race I-9b and can be effectively used in incorporating broad-spectrum rust resistance in important cultivars. In cultivars K_2 and LC45 which have been released for cultivation in Himachal Pradesh, resistance to the widely prevalent race I-9c was governed by one dominant gene each. However, inoculation of these cultivars with different races showed that both these cultivars are susceptible to several races and hence their utility as donors for rust resistance is limited. Cultivar K_2 which is susceptible to many races, is resistant to the widely virulent race I-17 and may thus be useful as a source of resistance in other areas. A perusal of data on the virulence spectrum of different races shows that there is a large reservoir of resgenes still available for exploitation in breeding linseed cultivars for rust resistance.

Rust resistance in EC-77959 and A-7-1-1 is governed by the same gene (39) BS-44 carries two resgenes, one of which was the same or allelic to that of EC-77959 and A-7-1-1, Hira carries two resgenes different from others. EC-77959 is a useful donor owing to its resistance to both rust and powdery mildew. In a recent study using mixture of races (I-1 to I-7), inheritance of rust resistance in cvs. R-17, R0552, JLS(J), R-556 and ILS-73-25 was governed by a single dominant gene (19). Dominant duplicate gene was observed in Himalini, C-59 and Dhar local-2. Crosses between Dhar local-2x Himalini and Dhar local-2xC.59 exhibited 255R: Is indicating operation of 4 diverse dominant genes.

Annual recurrence

In the plains of India unlike other countries teliospores do not remain viable through the summer which is the critical period for their survival, as they are killed by exposure to intense heat in May to September (44). On the other hand linseed rust pathogen can survive in uredial form on self sown plants and among seeds in the form of stem bits bearing telia as contaminent in Simla and Kangra hills of Himachal Pradesh. Evidence has been obtained on the active role of such teliospores in initiating fresh outbreaks (33). From such places in the hills, uredospores are blown down to the foot hills and in the presence of high humidity the rust spreads to other places. In Kangra valley, rust out break has been reported in late October (47) and places like Gurdaspur, situated in the plains close to the valley get very early rust outbreaks.

Management

In Madhya Pradesh region, sow the crop early within third week of October and grow resistant cultivars like Jawahar-7, Jawahar-

17, Jawahar-552 and JLS(J)-1. In Punjab and Himachal Pradesh grow resistant cultivars like CL-54, LC-185, K-2 and Himalini. If required then spray the crop with Dithane M-45 or Dithane Z-78 (0.25%) @2 kg/ha or dust with Sulphur @17.5 kg/ha at 10 days interval. In hills diseased plant debris should be destroyed to reduce source of primary inoculum (2,48,49). The cultivars like K_2 and LC 45 which are resistant to some of the races should be grown in plains. However, in the hills the cultivars like LC 216, LC 255, LC 256 should be grown since these are resistant to all the prevalent races. It will curb the pathogen at source of perpetuation and will also check outbreaks and spread of rust to the plains (51,53).

Powdery Mildew

Powdery mildew of linseed caused by *Oidium lini* Skoric is only next in importance to rust. Most of the rust resistant cvs of linseed are highly susceptible to powdery mildew. When powdery mildew appears in severe form at early stage of the crop growth yield reduction is very high. Infected plants produce poor quality of seed and fibre.

Disease development

The pathogen is believed to perennate in soil on diseased plant parts through the formation of parethecia. In the next season when favourable weather conditions prevail, asci and ascospores are released which initiate the primary infection. Usually in Northern and Central parts of India disease appears in the last week of February and reaches its maximum level by the middle of March when the temperature is between 20-25^{0}C and humidity is less than 65%. Rainfall is unfavourable for disease development.

Physiologic specialization

There are enough evidences that powdery mildew pathogen exists in different forms of pathotypes. To identify the pathotypes of *Oidium lini* occurring in India ten host differentials viz., LMH-7, LMH-27, LCK-24 , LCK 242, KL-37, A-4-3-2, SPS-48-5, SPS-19-13, R-552 and Chambal or T-397 have been identified (2).

Sources of resistance

ED-22587 and EC-22684 genotypes were reported to be free from powdery mildew under M.P. conditions by Agarwal (1975). After exhaustive screening of available germplasm under field and greenhouse conditions, four lines, viz., LC-216, LC 255, LC-256 and LC-269 were found highly resistant both in seedling and adult plant stages by Singh and Saharan (1979). Under uniform disease nursery trials conducted at different locations all over India for three years (2) have shown genotypes, viz., R-552, SPS-48-5, RLC-5, and RLC-19 as resistant to powdery mildew. Amongst the cvs recommended for growing in different regions, LC-185, LC-54, Himalini, and Jawahar-552 are resistant to powdery mildew under field conditions. High HCN (Hydro cyanic acid) content were reported in resistant genotypes like EC-77959 and EC-1456 whereas susceptible genotypes were low in HCN (41).

Inheritance of resistance

In an exotic collection, EC-9832 resistance to *O.lini* was reported to be governed by a single dominant gene (29) whereas one partially dominant gene was reported governing resistance in EI-5665 (3). In an exhaustive study (61) resistance to *O.lini* in each of the four linseed cvs, viz., EC-216, LC-255, LC-256 and LC-269 is

conditioned by one dominant gene. Crosses between the resistant parents indicated that the same gene was present in all the cultivars and is designated as *Ol*. A single pair of dominant genes governing resistance to the two diseases, powdery mildew and rust individually was found (37). Resgenes for *M.lini* in EC-77959 appeared to be the same or allelic to that of A-7-1-1. The resgenes for *M. lini* and *O. lini* appeared to be tightly linked in EC-77957. This offers an opportunity to use it as a donor for evolving varieties resistant to the two diseases simultaneously.

Management

In Madhya Pradesh region sow the crop before the third week of October and grow resistant cultivar R-552. Under Punjab and Himachal Pradesh region grow K-2, LC-54 and LC-185 cultivars which are resistant to this disease. Spray Bavistin (0.2%) or Sulfex (0.3%) or Karathane (0.2%) or Wettable sulphur (0.25%) at weekly interval depending on the intensity of the disease. Proper coverage of the crop with fungicidal spray is essential to curb the disease (2,48,49).

Blight

It is caused by *Alternaria alternata* (Fr.) Kiessler. Leaf infection causes damage from 27 to 60%. However, bud infection can cause loss up to 90%. The disease is most harmful when buds and capsules are affected (6). There is significant negative correlation (r=0.7567) between the disease intensity and yield. Yield loss can be estimated through regression equation, Y=733.35-8.24X (17).

Disease development

The pathogen perpetuates through the seed and diseased plant debris. The temperature between 26-33°C and humid conditions are found to be most favourable for the growth of the fungus and for infection of the plant. If RH falls below 75% than disease development becomes restricted (17).

Source of resistance

All the recommended cvs. of linseed being grown in different parts of the country are susceptible to this disease. Under artificial inoculation tests, four genotypes, viz., FRW-12, EC-4162, My-1 and *Linum strictum* were found resistant to this disease (26,63). In AICORPO trials the genotypes of linseed namely KL-37 and LCK-240 were found resistant to this disease under uniform disease nursery trials conducted for three years at different locations (2) of India.

Management

Sow seeds after treatment with Bavistin (2g/kg seed) or Agrasan or Thiram @3g/kg seed to avoid seed borne inoculum. Grow resistant cultivars like, R-7, R-17 and R-552 in M.P. region. Spray with Dithane M-45 (0.25%) @2 kg/hac at 15 days interval depending on the intensity of infection (2). A mixture of calixin (0.05%) and dithane M-45 (0.25%) spray is very useful for controlling blight and powdery mildew both (2).

Wilt

Linseed wilt is caused by *Fusarium oxysporum* Scht. f.sp. *lini* (Bolley) Snyder and Hansen. Continuous cropping in the infected fields creates soil sickness. Up to 80% losses to the crop due to wilt pathogen was reported (57).

Disease development

The pathogen is soil borne and can survive in soil for many years. It

has been isolated from the seed as well. It can be carried over by the infected root pieces in the soil from year to year during continuous cropping in the same field. The temperature between 25-28°C is most suitable to get maximum infection with an optimum of 24°C. At unfavourable soil temperature of 12°C and 38°C even susceptible cvs escape much damage (22,27). In India, It has been reported that the disease is favoured by low moisture and light sandy soils (18). Farmyard manure decreases the incidence of the disease. Potassium content of the plants has been reported to be correlated with their resistance to wilt (7).

Physiologic specialization

It has been demonstrated that wilt pathogen consists of several cultural and pathogenic races (4) which differ from each other in their cultural character, pathogenicity and temperature requirements. Antagonism exists between some races. Continuous cropping in the same field increases virulence of the pathogen.

Sources of resistance

The cvs like K-2, LC-185, LC-54, Himalini, and Jawahar-552 are tolerant to wilt disease. Genotypes namely, RIC-1, RLC-5, RLC-6, RLC-18, RLC-19 and R-552 have been found to be resistant to wilt pathogen tested at different locations under UDN for three years in AICORPO (2). BR-9, BR-29, Indore-1, Malvi-1, N.P.-12, Bison-69, Bison-70 and Canadian Western No.1 were found to be resistant to wilt (5).

Inheritance of resistance

When wilt resistant genotypes viz., BR-1, EC-544 and NP-RR 65 and rust resistant cv. Norman were crosed than in F_2 resistance to wilt and rust in respective parents was found to be governed by separate single pair of dominant genes. The resgenes for the two diseases in the parents showed independent assortment (28).

Management

Continuous cropping of linseed on the same field should be avoided to check the development of wilt inoculum in the soil. To eradicate seed borne inoculum seed treatment with Bavistin @2g/kg seed or captan or Thiram @3g/kg seed is essential. Grow tolerant/resistant cvs like R-552 in M.P. and K-2, LC-54, LC-185 and Himalini in H.P., Punjab and Haryana (2). It is believed that flooding of the field and crop rotation reduces wilt inoculum in the soil (48,49).

Gaps in Linseed Pathology Research, Future Priorities And Thrust

Most of us explain from time to time about the gaps in our knowledge on various aspects of our research work. It is not uncommon to hear statements that gives a few more staff or a little more in the way of funding and equipment, it would be a relatively simple matter to fill in these gaps. The task is more complicated than one would had envisaged since it is imperative to define and describe the resistance of gaps, where they are? what they are? and why they exist? I shall outline the gaps in linseed pathology research as under.

1. Identification of physiologic races: In India linseed rust race picture is based on their reaction on differentials developed by flor which are supposed to be apparently monogenic. However, later studies proved the presence of additional (L^9) gene in some of the differentials known to be monogenic for alleles in K, M, L and P loci. It implies, therefore that differentiation of races could have

been bliterated in the past, in relation to differentials carrying additional Bison-gene (L^9). So, it is very important to evolve monogenic differentials in the background of a universally susceptible local cultivar to know the precise race-picture in India. There is an urgent need to standardize host differentials for identification of pathotypes of wilt, blight and powdery mildew pathogens of linseed.

2. Genetics of resistance: Lines having single, two and three resgenes conditioning resistance to all the races have been indicated. To make resgenes more effective and to avoid repeated breakdown of resistance attempts should be made to incorporate more than one resgene in a single cultivar. Within limits of all elism a more efficient test for all elism is a study of test cross population. The study by using large test cross population is needed to incorporate more than 5 resgenes in a particular cultivar.

We quite often talk about multigene resistance and multiple resistance, the efforts in this direction can be made to locate genes effective against other diseases (powdery mildew, wilt, blight) in addition to rust.

Although manifestation of horizontal resistance/durable resistance in linseed to diseases has not been demonstrated but more studies should be conducted actively on the components of horizontal resistance.

The principle of gene deployment and gene pyramiding should be put into practice for the management of linseed diseases in India.

3. Epidemiology: To predict the development of linseed rust in epidemic form, more information is required on the analysis of environmental factors responsible for rust development in different agroclimatic zones of the country. Some basic studies on the prevalence of races in relation to virulence are needed to know their behaviour with regard to competitive virulence potential and ability to survive. Detailed study on the epidemiology of other diseases should be taken up on priority.

4. Annual recurrence: The role of southern hills in the survival and spread of the diseases needs attention. More information should be collected on the role of wild species of *Linum* for perpetuation of the pathogens and for sources of resistance.

5. Sources of resistance: The history is full of examples that in most of the host-parasite interactions repeated breakdown of resistance has resulted in depletion of genes for effective resistance. Attempts should, therefore, be made to search for new resgenes.

6. Control: The emphasis should be laid on the management of disease in linseed through management of genes, since it is easy to incorporate, deploy and do resgene pyramiding in this crop.

7. Information gap: Some problem arise in linseed research from lack of information on the work already done in India and in other countries. This can lead to expensive duplication of efforts. There is a great need for more newsletters, review articles and information leaflets which can present new data and results of recent research. There is also the problem of informal exchange of research data and material. I hope the present review will be helpful in filling the information gap to some extent on practical aspects of linseed diseases.

8. Co-operation gap: We have several times stressed for an interdisciplinary approach to research but in practice we have failed to implement it. Apart from cooperation or integration with other disciplines a joint research program of breeder and pathologist is must to develop disease resistant cultivars. Every effort should be made to encourage a team approach to tackle disease problems.

In conclusion one has to admit that pitfalls or gaps in linseed pathology research may be many more but filling of big gaps should be given priority so that linseed pathology research vehicle can move smoothly for keeping up present production and increasing future production of linseed in India.

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