STUDIES ON SCLEROTINIA SCLEROTIORUM, CAUSING WHITE MOULD ROT OF SUNFLOWERS IN HUNGARY

Dr. J.RATKOS
Research Centre of Seed Production and Marketing Company,
Postal address: 4400 Nyiregyháza Westsik Vilmos street 4-6.
Hungary

SUMMARY

Sclerotinia sclerotiorum is one of the most dangerous plant pathogens in Hungary, causing significant yield losses in a wide range of cultivated broad leaf plants. Nevertheless,the most serious quantitative and qualitative demages are caused by white mould on sunflower. It is a cosmopolitan fungus and widespread all over the world.
In order to work out sufficient control method against the disease it became absolutely essential to know more about the biology of the fungus and to reveal the most important epidemic factors.
An artificial inoculation method, similer to the natural infection have been worked out. For this purpose sclerotia are freezed in perlite or in composted soil at minus 15-20 C^{o} for 3-4 weeks, under diffuse light and 80-90 % relative humidity.After the appearance of the apothecia sporulation occur in 1-3 days. Discharged ascospores can be collected by suitable sporetraps, and can be used for artificial inoculation, in order to evaluate susceptibility of sunflowers in various phenophases, differences in the susceptibility of different hybrids and breeding materials, wild Helianthus spp., etc.
Artificial inoculation of various plants revealed, that Sclerotinia sclerotiorum can infect young bracnhes of locust-tree, and swingle, too.

Epidemic appearance of white mould can be forcasted by placeing overwintered, or freeze-treated sclerotia to a marked place of the sunflower field. After apothecium formation and ascospore discharge continuous humidity for at least 30-40 hours is essential for the actual infection.

When infection is forecasted, benomyl, carbendazine, procimidon or vinclozoline fungicides can prevent the development of white mould rot of sunflowers.

INTRODUCTION

The Sclerotinia sclerotiorum /white mould/ belongs to those "multi host" pathogenes, which causes significant quantitative and qualitative production losses in many field and culture plant.
/Molnárné at.al 1963, According tothe recent observations the fungus is capable to parasite the young branches of ligneous plants /Ratkos 1986./.

The pathogene can infect the plants in two ways. On the one hand from the "sclerocia" in the soil-which is the overwintered scheme of the fungus-micelia develops and infects the vegetative and reproductive surface in contact with the soil.

On the other hand by the ascospores getting free from the productive bodies, - in other words by the generative reproductive schemes which germinating on the surface of plants, through their enzym effect infecting the host plants by inbeding into their tissue.
/Adams at.a., 1976. Swartz at.al 1978. Lamarque 1981, Ratkos 1983./
For the development of "apotecia" the following ecological factors are highlightened by some authors: light and dampness /Honda at.al, 1975/; the depth of sclerocia in the soil /Williams at.al, 1965/; the characteristic of cultured plant /William at.al, 1965./; chemical cultivation of soil /Partyka at.al 1958. 1962./; agrotechnical cultivation of soil /Stevens at.a. 1911./

The askospores getting free through the explozive movement of "apotécia". /Ratkos, 1982./

The importance of genetical protection is highlighted by Vranceanu /1977/.
About the importance of agrotechnical protection writes Petróci /1985/.

About the role and importance of antagonist and hyperparasite organs write, Litkei /1986/.
In the chemical protection against the pathogene the diklozolin inprodion, procymidon, vinklozilin /Enisz 1985/, and the karboxin, /Achimovich 1977/, having pro-minent role.
In the interest of improving the efficiency of the protection Enisz /1985/, Vörös et.al /1986/, Milinkó et.al /1986/,write about the need to know more about the biology of the pathogene and to work out the protection.

The aim of the research was to find out at what enviromental condition gets the pathogenes reproduction scheme free and the generative reproduction schemes /askospores/ at what conditions infects the plants.
It is essential to get the answer for the above to be able to use inoculation tests with similar result as the spontenious infection and to work out a protection technology based on forecasting.

MATERIALS AND METHODS

The tests were carried out in the laboratory and provocation-garden of the Research Centre of Nyíregyháza and on its field.
The work can be summerized as follows:
- showing up the functional apotecia, sporogenesis tests
- inoculation experiments to study the process of pathogenesis
- determination of infection period on the field.

For our inoculation tests and field observations the sunflower was used.
The sclerocium needed for the tests was wintered in the field isolator. At the end of the winter, after the infection it was put into perlit with 5-10 cm thickness and then we have placed the material in a foil-hut.

During the test we observed the temperature, light and relative humidity conditious. We have followed the development of apotecia, their change of colour, and their askospore productivity.
To collect the askospores we have used a suitable spore-trap /Ratkos, 1983/.
We observed the daily rhytmic of sporulation and measured one apotecium's askospore production.
The spores-collected in spore traps-were blended in 2 ml destilled water, then we determined the concentration with the help of Bürker-chamber.
We checked the sporulation time in the function of concentration.
We checked the flora of parasited sclerocia and apotecia and identified the flora's element. The study of the pathogenesis of Sclerotinia sclerotiorum was done with the inoculation of GK-70 sunflower under foil-hut.
We did the inoculation with $4x10^4$ off/ml concentrated spore-suspension. Similarly to the sporogenesis tests we have measured the enviromental conditions at the pathogenesis tests as well. /With Thermo-hygrograf, Lux-meter, maximum-minimum thermometers/.
We have studied in a provocation garden the phenological susceptibility responsiveness of the sunflower.
This was done under isolating hut and we have ensured the climatic conditions by water spraying. Until the developing of symptoms we have measured the surface dampness of the plants by DEFI-DOFA instrument, DEW-DINAMIC SYSTEM micro-processor controlled measuring instruments and LUFT instrument.
The inoculation was carried out in the following phenophases: star bud, 4-5 cm flower bud and half-blooming stage. We have sprayed 3 ml suspension onto the leaves, stem and the blossoming head of each sunflower.
For the simultanious inoculation of different genotypes at the opening of 3^{ED} -4^{TH} flower ring we have sprayed 3 ml suspension containing 4x10 spores/ml, onto the blossoming head.

RESULTS

The results of the sporogeneses test were the following: The sclerocia can develop apotecia between 10-30 ^{o}C but the most intensive is between 15-25 ^{o}C, 85-100 % RH. and at 1500-6000 LUX /average/ light density. In case of less light density the apotecia are smaller, the stem of apotecia /stigma/ is longer, expanded. The 3-5 cm soil coverage is the ideal, but they can develop from 7-8 cm depth as well.
Between the shape size and number of apotecia there is no positive correlation except the extreme values. Their life span /the product of one sclerocia/ is 3-4 week in balanced condition. During this time their colour and parasitation change also.
The most common saprofita flora elements are the following: Trihoteceum roseum, Alternaria ssp, Aspergillus ssp, Trichoderma ssp.
The askospore production from the fully developed apotecia begins 1-3 days after the first appearance.

Above 90 % relative humidity there is no sporulation.
In 12 hours they can force the apotecium to produce 1-3 askospores, but with less intensity one /1/ apotecium produce $2x10^6$ spores as an average. During 4-5 hours the ascospores germinate. The "PH" optimum of germination is between 6-7.
During the pathogeneses test the obtained results were the following: The development of micelium is the most intensive between 16-24 ^{o}C.
High concentration /10^6-10^7 off/ml/ of ascospores on the surface can result in selfantagonism regarding the germination.
The amino acids are improving the germination. In case the hyphal dries out it looses it's vitality. For the infection 35-40 hours and 85-100 % RH are needed.
The symptoms will develop in 4-5 days and the development of fungus is intensive. Testing the phenelogical susceptibility of sunflower the following result were obtained: at the star-bud, green-bud and half-blossom stages infection can also be done. The infection is the highest at the inoculation carried out during half-blossoming. During the inoculation in respect of susceptibility essential differences showed up between the verious genetypes.

DISCUSSION

At the testing of the sporogeneses of the Sclerotinia sclerotiorum it was found that the apotecia develop well in perlit and soil at ideal conditions. The advantage of perlit is that in it the apotecium less prone to parasitation but require more watering then in the soil.
The temperature is of less importance. The dececive condition is the light and dampness. In case the soil is damp but the light is intensive the development of apotecia ara slow. If however the light intensity drops then the development of apotecia will begin more rapidly.
The phenological susceptibility of sunflower is changing. It is more susceptible in seed-leaf stage and then in star-bud, budding and half-blossoming stage. Placing the inocolum onto the dried up head of the sunflower there can be substantial differences between the various genotypes regarding the frequency and spreading of the infection. This is genospecific feature and is typical characteristic of the bred and caltivated genotypes.

CONCLUSION

Our tests produced new informations, utilizable in practice.
The more exact knowledge of the sporogeneses and pathogeneses of Sclerotinia sclerotiorum can make the genetical, agrotechnical, biologycal and chemical protection more integrated.
The infection method using ascospores is comparable to that of spontaneous infection load so the research of gene-sources and fungoids can be more exact.
With more exact knowledge of the phenological susceptibility of the sunflower or the generative breeding cycle of pathogenes the prevention can be achieved on the field in large scale farming.

The apotecia can appear at the closing of foliage and the sunflower-because of " stress " caused by the begining of the generative phase-is physiologycally less resistant. At favourable eclogycal condition epidemic can break out. The justification of foliage treatment is depend on the interaction of meteorologycal factors, in which the dampness condition the environment is decisive.
With the help of the above information a successful, economical and an environment considerate production technology can be realized.

ACKNOWLEDGEMENTS

The author gratefully acknowledge the facilities and encouragement received from Dr József Vörös professor of biology, Plant Protection Institute, Hungarian Academy of Sciences and the cooperation of Steven Guth in preparing this paper.

REFERENCES

1. Acimovic M. 1977, Laboratory examinations of some fungicides for controll of Sclerotinia libertiane Fuckel.
Zastita Bilja, 28. pp. 143-146.
2. Adams P.B., Tate C.I. 1976, Mycelial germination of Sclerotinia sclerotiorum on soil.
Plant Disease, Reporter, 6o. 6. pp. 516-518.
3. Békési P. /et.al/ 198o, Sunflower cultivation.
Agricultural Publishing House, Budapest.
4. Honda Y., Yunoky T. 1975, On spectral dependence for maturation of apothecia in Sclerotinia trifoliorum.
Erik Ann. Phytopathol Soc. Japan 41. 4. pp. 383-389.
5. Jones D. 1974, Fungicidal effects of the fumigant dasomet on sclerotia of Sclerotinia sclerotiorum in soil.
Trans.Br.Mycol.Soc. 63. pp. 249-254.
6. Lamarque C. 1981, Conditions nécessaires á la contamination du tournesol par les ascospores de Sclerotinia sclerotiorum /Lib./ de Bary application á la prevision des epidemies locales.
Informations Techniques CETIOM, 75. pp. 4-6.
7. Litkei I. 1986, Hyperparasite ofGlyocladium varietas on Sclerotinia sclerotiorum.
Scientific Journal of the University of Agricultural Sciences of Debrecen, 224 p.
8. Milinkó I., Piscul G., Kadlicskó S. 1986, About pathogens of sunflower. MÉM NÖVOV Seminar. Budapest, pp.256-262.
9. Molnárné, Vörös I. 1963, Stem rotting caused by the Sclerotinia sclerotiorum on the soya-bean.
Plantproduction Tom. 12.No.1. pp. 51-55.
1o. Petrőczi I. 1982, Plant protection on the field.
Mezőgazdasági Kiadó, Budapest. pp. 362-37o.
11. Ratkos J. 1982, The possibility of obtaining Sclerotinia sclerotiorum /Lib./ de Bary ascospore on the field.
Plant protection XVIII.Vol.4. part pp. 170-177.
12. Ratkos J., Galambosné L.J., Kucy J. 1983, Possibilities to work out protection against the disease coured by the Sclerotinia sclerotiorum.
Plant protection XIX. Vol. 2 th part. pp. 356.

13. Ratkos J., Nagy I., Sallay F. 1985, Increasing the safety of sunflower cultivation by reducing plant hygiene problems.
Agroinform Szed. Budapest XII. 3.p. pp. 44-57.

14. Ratkos J. 1986, Futher results of studies on the Sclerotinia sclerotiorum /Lib./ de Bary / white mould / fungus.
Plant protecting Scientific Days 1986.
Budapest, F. pp. 99.

15. Ratkos J. 1986, Conditions of Sclerotinia sclerotiorum reproduction on the open field. More profitable sunflower cultivation.
MÉM, NÖMÖV Seminar, Budapest, pp. 288-293.

16. Schwartz H.T., Steadman J.R. 1978, Factors affecting sclerotium populations of and apothecium productium by Sclerotinia sclerotiorum.
Phytopathology 68. 3. pp. 383-388.

17. Stevens F.L., Hall I.G. 1911, A serious lettuce disease /Sclerotiniose/ and a method of controll.
p. 88-145 in N.C. Agric.Exp.Stu.Tech.Bull.8. 145 p.

18. Williams G.M., Western I.H. 1965, The biology of Sclerotinia trifoliorum.
Erikss. and order species of sclerotium forming fungi.
The survival of sclerotinia in soil.
Ann.Appl.Biol.56. pp. 261-268.

19. Vranceanu A. 1977, The sunflower.
Agricultural Publishing House,
Budapest, pp. 248-251.

2o. Vörös J. 1983, Plantprotection of Sunflower in Hungary, International agricultural Review, 27. pp. 49-52.