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THE PATHOGENIC ACTION OF RHIZ
POTATO

H. T. GÜSSOW

WITH ONE FIGURE IN THE TEXT

It appears that satisfactory evidence has been la
strating the pathogenic action of Rhizoctonia (*Corti*
on various host plants, particularly the potato.

The attention which the well known disease has
world and more recently in the old, left, in my opi
quite a number of other investigators, several imp
plained. Every pathologist fully recognized the sy
ease which have come to be regarded as typical Rh
The unmistakable folding of the leaves of the grow
with the brown stem lesions so frequently described,
or in addition perhaps, the formation of aerial tubers
ness of subterranean tubers, are now well known as
resulting from an attack of Rhizoctonia.

Indeed, the folding of the leaves associated with
greatly from the curling of the leaves of plants affe
particularly noticeable is this difference when examinin
of an affected plant. Where, however, doubt existe
stem lesions was looked upon as final proof of a Rhi

I must confess, however, that the often surprising s
ent superficiality of these lesions, nay, often enough th
in what was otherwise unmistakably a plant infected
and not with leaf roll, frequently caused me surprise a
to readily convince the farmers on the occasion of fie
of the correctness of the diagnosis—which at times I sha
my own belief.

For some time I have endeavored to discover the true
of this fungus on the host plant. My colleague, Drayto
to my satisfaction the profuse permeation of the tissues
ing the lesions, but even that failed to convince me enti
of the characteristic symptoms, whicl statement is not

[1] Drayton, F. L. The Rhizoctonia lesions on potato stems.
1915

1917

F RHIZOCTONIA ON

W

HE TEXT

s been lacking clearly demon-
ia (*Corticium vagum* B. & C.)
tato.

ease has received in the new
n my opinion and in that of
eral important points unex-
l the symptoms of this dis-
ypical Rhizoctonia infection.
the growing plant, together
described, in other instances,
al tubers and peculiar small-
known as general symptoms

ed with this disease differs
ants affected with leaf roll,
examining the lower leaves
bt existed, the presence of
of a Rhizoctonia infection.
prising scarcity and appar-
nough their entire absence
infected with Rhizoctonia
urprise and certainly failed
on of field demonstrations
nes I shared much against

the true pathogenic action
, Drayton,[1] demonstrated
e tissues of and surround-
me entirely of the cause
t is not intended to infer

o stems. Phytopath. **5**: 59.

that I doubted Rhizoctonia to be associated with the same. But from what evidence was available, the actual injuries caused by the stem lesions were so infinitesimal that it was felt the true injury is done elsewhere, and what we did observe was the result of such mislocated but far more serious injury.

A careful study of diseased plants in the field revealed at first little or no additional clues. Lesions were sometimes present where the leaves were folded, the tubers were covered with more or less numerous lumpy fungous masses, indeed the roots often showed the well known pseudosclerotia. Pot experiments showed the presence of sclerotia on rootlets more abundantly than was the case in the field, and yet while abundant superficial and lesser amounts of intracellular hyphae of Rhizoctonia were found on microscopical examination, the evidence of an all round general soundness of the underground parts examined still left the seat of the injury undetermined.

On careful examination of the root system of a plant clearly affected with Rhizoctonia and no other disease, that had been pulled up from loose sandy soil, or had been lifted with care by means of a fork or spade, one factor at last attracted my attention, which later led to interesting observations. This was the almost entire absence of the fine fibrous rootlets, so common in sound plants. Surely such rootlets must have been present originally? When examining thereafter plants in various stages of infection one could observe a corresponding absence or presence of finer rootlets according to the amount of disease present. Of course, in this determination care is necessary, but after some experience one cannot but recognize the existing relation of rootlets to degree of disease.

What if the fungus acted upon the roots of the growing plant similar to the way in which it does on the roots of Rhizoctonia-infected tubers sprouted in a closed stender dish? The disastrous effects of the fungus at the early stages of growth are sometimes so pronounced as to kill off growth altogether; this is a well known fact.

Let us bear in mind that in a potato field we find many stages of severity of Rhizoctonia infection, from total "misses" to one, two or more shoots clearly affected up to the case where the plant bears plenty of aerial tubers and numerous little potatoes underground from which the popular name "little potato disease" has sprung. Aerial tubers have been commonly associated with Rhizoctonia, they are perhaps exclusively manufactured from material produced by the leaves, a comparatively slow process, but always indicating impaired root function in plants where they may be considered abnormal. Aerial tubers naturally may occur from any cause cutting off or interrupting root function, but only when such interruptions are gradual. We have, therefore, no aerial tubers in the black

FIG. 1. RHIZOCTONIA LESIONS ON ROOTS OF POTATO

A, Root of potato fourteen inches in length showing at *s*, sclerotial deposits and at *x*, many places where the fine absorbing roots were killed by the action of the fungus. Slightly reduced.

B, Portion of root shown in *A*. × about 1.5.

leg disease, where the cutting off of supplies is rather sudden. All these symptoms are the logical results of the absence of the abundant feeding roots. Roots are present in all growing plants, otherwise the plants would have died; small and fine roots are less in evidence in affected plants while a generous supply exists in healthy strong plants.

Deductions—however logical they may be—still are hypotheses and hypotheses are not facts, but the accompanying plate will provide some foundation for the observations recorded and may stimulate wider researches on this point than have been made so far. I am satisfied from the observations made, that the destruction—often very gradual—but very persistent all the same, of all or many of the feeding roots of the potato plant accounts for every one of the symptoms associated with this disease. The lesions which have so often been recorded are evidently not of serious consequence, as indicated by their general superficiality and frequent entire absence. In some instances indeed these lesions are not due to Rhizoctonia at all, but to *Actinomyces scabies* Güssow, which I hope to show in another paper, when they afford easy resting places in the unprotected superficial cells for the mycelial masses of Rhizoctonia shown in Mr. Drayton's photo-micrographs, as well as for the permeation of the hyphae into the interior, which, as must have been noticed, is not accompanied by any prominent injurious action upon the cells invaded. A study of Mr. Drayton's slides clearly confirms this observation as well as the photographs made from them which are accessible to our readers.

The pathogenic action is as follows: We are aware of the very profuse growth of mycelium of Rhizoctonia, particularly in the dark, as also of the production of enormous quantities of pseudo-sclerotia on roots and tubers. Whether the sclerotia are left over in the soil from preceding potato crops or other host plants, or whether they have been introduced by untreated infected seed potatoes—(and what "farmers' run" potatoes are not infected?)—does not matter much. The tips of the fresh rootlets soon fall a victim to the invading mycelium, the root cap being undoubtedly the most vulnerable point and soon the short roots have been destroyed, the mycelium meanwhile reaches older rootlets, which it much more rarely destroys, though that has occurred, but where the mycelium frequently produces resting mycelial masses from which invading hyphae issue almost simultaneously with new rootlets which are produced by the plant in its effort to reestablish its resources. This process goes on gradually and slowly or more rapidly depending naturally upon the vigor of the plant. Finally the persistent efforts of the fungus result in decreasing yields, in frustrating the growth of the tubers, because of lack of food supplies from the roots, and eventually in the production of aerial tubers. Meanwhile harvest-time has arrived, what tubers are there, are harvested.

but the roots infected with sclerotia remain in the ground, since they are not pulled up by the digger or are at any rate returned to the ground. With the diminishing food supply in these roots, sclerotia develop ready for subsequent attacks. This observation also accounts largely for the soil contamination and the persistence of the organism in land once infected. It also indirectly suggests a new means of control, viz., the prevention of infection by cultural methods or the application of fertilizers producing vigorous plants in the first instance and aiding in the production of a generous supply of new feeding roots.

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