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Scientific Research

Susceptibility of Wheat Varieties to Soil-Borne *Rhizoctonia* Infection

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ABSTRACT

Response of 19 wheat varieties cultivated in Hungary varied within large limits to soil borne *Rhizoctonia* infection. The most frequent symptom, usually leading to damping off was the root neck necrosis. Four significant factors influencing the susceptibility of wheat comprised 71% of total variation but none of them was dominant. The inhibition of development of survivors in *Rhizoctonia* infested soil correlated with overall susceptibility of variety concerned. The varieties Emese, Kikelet and Palotás are proved to be less susceptible, but none of the varieties could be certified as tolerant. No relationships were revealed between pathogenicity of 26 *Rhizoctonia* strains studied and their taxonomic position or origin. The anamorph strains of *Athelia*, *Ceratobasidium*, *Ceratorhiza* and *Waitea* similar to *Thanatephorus* anamorphs selectively infected the wheat varieties, but the syndromatic pictures were undistinguishable with unarmed eye. *R. solani* was proved to be more aggressive against germinating wheat than *R. cerealis*. Nine significant factors influencing the virulence of *Rhizoctonia* strains comprised 82% of total variation, and six of them influenced exclusively *Thanatephorus* anamorphs.

Keywords: Wheat; *Rhizoctonia*; Tolerance; Brown Patch; Soil-Borne; Virulence

1. Introduction

In August 2002, brown patches were observed on turf grasses in parks at four locations in Budapest. The symptoms observed were necrotic lesions on the roots and stems, as well as brown lesions on the leaves. Two types of sclerotia, nearly globose, pinkish to orange and irregularly shaped, dark brown were found on roots. On potato dextrose agar fast growing, colourless colonies with small reddish/coloured scelrotia uprose of the first type. This fungus was identified as *Rhizoctonia zeae* Voorhees (teleomorph *Waitea circinata* Warcup and P.H.B. Talbot) [1]. Buff-coloured, fast growing colonies of *Rhizoctonia solani* Kühn (teleomorph *Thanatephorus cucumeris* (A.B. Frank) Donk) arose of the second type. The study of more than 150 plant species cultivated in Hungary [2] revealed that *R. zeae* attacked monocotyledonous species more aggressively than dicotyledonous ones, contrarily to *R. solani*. This latter species was formerly reported as pathogen of winter wheat [3] and oat [4] in Hungary.

Traditionally, farmers paid little attention to field damage caused by soilborne *Rhizoctonia* infection in wheat, because either seedborne or airborne fungi (rust, mildew, smut etc.) infecting stem, leaves and spikelets had been the main constrains of yield. Due to success in breading and arousal of new synthetic fungicides, these fungi presently do not cause catastrophic yield losses. However, in the last two decades increasing number of papers was published on yield losses (30% to 50%) caused by *Rhizoctonia* species [2,5] in main wheat cultivating areas [6-8]. In Europe and North America winter wheat suffered mainly of *R. solani* AG-8 strains [7] with the *R. cerealis* [6-8], while in Australia AG-1 and AG-8 and in Turkey five different anastomosis groups of *R. solani* [9,10] were revealed. In South Eastern Hungary damage by the *R. cerealis* and *R. solani* has been observed in spring wheat [3].

Rhizoctonia species are well known soil borne pathogens frequently causing damping off prevalently in moist and cool conditions that are the main stress factors re-

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quested to induce disposition to increased susceptibility of potential host plants [11]. These species have no vegetative bodies for spreading, but infected seeds and propagating material can distribute infecting propagula. The disease is more severe in sandy soils, as the fungus can grow more rapidly [12], and the hyphae tend to colonize rhizoplane reducing the vitality of plant even without penetrating into tissues. Infection cushions are formed on the surface before individual hyphae penetrate with minor morphological modifications [13]. The ability to produce discrete appressoria is highly variable and regulated seemingly by numerous factors. Hyphae may penetrate without an appressorium through stomata or wounds [14-16]. All *Rhizoctonia* species are obligate aerobic fungi, which are habiting mainly in rhizosphere, however, they may survive as saprobionts in the upper layer of the soil forming a mycelial web, thus the undisturbed soil enhance the risk of the infection of young roots [17]. *Rhizoctonia* root rot is more frequent and severe under reduced or conservation tillage [18,19] as the conventional tillage disrupts the mycelial web [20]. This underlines the importance of the elaboration of new root health management practices, because formerly minor pest problems, such as *Rhizoctonia* root rot of wheat became major problems. In last two decades new research has been started and several hundred scientific papers reported data on etiology of root rot and sharp eyespot caused by *Rhizoctonia* species, on genetic background of susceptibility of wheat as well as on *Rhizoctonia* virulence. Actually, we can conclude that environment dependent mechanisms regulate the progress of this disease with complex etiology that is not under gene for gene control [21], and further research is requested to understand the multicomponent syndromatic picture.

The disease caused by *R. zeae* was described for the first time in Florida, by Voorhees in 1934 [22], as sclerotial rot of corn. Since that this fungus distributed in temperate regions, in Europe firstly reported in 2004 [1]. In a host-range study, the isolate proved to be highly pathogenic to germlings of several ornamental and cultivated plants, including *Beta vulgaris* ssp. *vulgaris*, *Callistephus sinensis*, *Dahlia variabilis*, *Daucus carota*, *Lupinus polyphyllus*, *Papaver somniferum*, *Pennisetum glaucum*, *Phaseolus vulgaris*, *Sesamum indicum*, *Solanum melongena*, *S. tuberosum*, *Sorghum bicolor* and *Triticum aestivum* [1,2,23]. The estimation of risk on wheat production caused by this pathogen was the aim of our work.

Actually, both seed and soil borne infections might be well controlled in early stage of germination with seed dressing [24], however, we have no effective methods with reliable cost/benefit ratio for protection of wheat against *Rhizoctonia* during the vegetation. Although mineral nutrients can manipulate the reaction of plants [25], such treatments can not combat serious yield loss. Several attempts were made to explore antifungal potential of eubiotic preparations and to desing suppressive soil [26-32], however, in order to prevent the harm, these developments have had not satisfactory results. In the case of soil-borne infections correlative influences among members of microbial consortia associated to potential host plant may influence both the invading pathogen and disposition of potential host to adverse factors of environment thus change both the virulence of pathogen and the susceptibility of host to pathogen at any time depending on the genetic potential of these partners. Due to complexity of interactions there is difficult to predict the success of biocontrol measures. Currently the effective protective method is the appropriate crop rotation, and the breeding of wheat cultivars for improved tolerance to factors inducing disposition to increased susceptibility to the presence of *Rhizoctonia* in the soil can be considered.

Our objectives of this study were the comparative evaluation of responses of germinating wheat seeds to *Rhizoctonia* strains of various origin and taxonomic position as well as to reveal patterns in factors influencing the wheat/*Rhizoctonia* interaction.

2. Materials and Methods

Greenhouse experiment was undertaken to compare the infective potential of *R. zeae* strain with 25 *Rhizoctonia* strains of various taxonomic position. Susceptibility of two sortiments of *Triticum aestivum* L., moreover, *T. monococcum* L., *T. turgidum* L. and four small seed grains were involved into the tests. No seed dressing or any other manners to depress the microbiota of spermosphere were applied. The potting medium was made by mixing forest soil with peat before autoclaving (1.15 atm per 20 min), at the ratio of 3:1.

2.1. Test Plants

Seeds of wheat varieties (**Table 1**) were gifted by Elitmag Kft (Martonvásár, Hungary). Except Alkor (*Triticum monococcum* L.) and Hegyes (*T. turgidum* L.) all are *T. aestivum* L. cultivars. Small seed grains *Eleusine coroacana* Gaertn., *Panicum milliaceum* L., *Phalaris canadiensis* L. and *Setaria italica* (L.) P. Beauvois were purchased of the market (HERMES Ltd., Budapest, Hungary).

2.2. Test Fungi

Rhizoctonia strains were originated of different locations and various hosts:

Rhizoctonia solani strains of CBS collection: B-415 (AG-1, *Pinus sylvestris* L., Canada, CBS 522.96), B-432 (AG-2, *Daucus carota* L., Netherlands, CBS 326.84), B-446 (AG-3, S*olanum tuberosum* L., Spain, CBS 117248), B-417 (AG-4, *Citrus* sp., Argentina, CBS 341.35), B-430

Table 1. Evaluation of the response of wheat varieties to soil borne *Rhizoctonia* infection.

Rhizoctonia strains (2)		Wheat varieties (1)																					Small grains			
		Pannon Standard 2010							Pannon Prémium 2010						Other varieties											
No.	Code (3)	Toborzó	Bodri	Emese	Petrence	Lucilla	Magvas	Palotás	Menüett	Toldi	Suba	Ködmön	Kolo	Mazurka	R23	Kikelet	Karizma	Lona	Hegyes	Alkor	Symptomless (4)	Killed (5)	*Eleusine*	*Panicum*	*Phalaris*	*Setaria*
1	B-415	2	0	2	1	1	0	0	1	1	0	0	1	2	0	1	3	0	1	1	0	7	1	3	2	0
2	B-432	5	5	5	5	5	4	5	5	5	3	2	5	3	5	4	4	3	5	5	12	0	4	5	4	4
3	B-446	5	2	4	4	2	3	4	4	4	5	4	3	5	5	5	4	1	4	4	4	0	5	5	5	0
4	B-417	1	2	4	5	5	2	5	2	2	3	3	3	4	5	5	4	1	5	4	6	0	5	5	5	5
5	B-430	2	3	4	3	5	3	5	4	4	3	4	4	5	3	5	5	4	5	5	7	0	5	5	5	5
6	B-418	3	1	1	4	3	0	4	1	2	3	2	4	3	3	1	3	1	4	3	0	1	5	2	3	5
7	B-419	1	2	5	4	4	2	5	4	5	2	2	5	5	5	5	3	2	5	5	9	0	5	4	4	5
8	B-420	2	2	4	5	3	5	5	1	3	5	3	3	5	5	0	3	2	2	3	6	0	5	5	5	5
9	**B-421**	4	5	2	5	2	1	5	2	4	4	4	3	3	5	4	3	1	3	5	5	0	4	5	4	4
10	B-422	2	1	4	4	2	1	4	2	1	1	1	0	4	5	5	3	1	4	1	2	1	5	4	1	5
11	**B-423**	5	2	4	5	3	5	5	4	3	3	3	5	5	2	5	5	3	5	5	10	0	5	5	5	5
12	B-424	3	3	5	5	3	3	5	4	4	1	1	4	4	4	4	2	0	5	4	4	0	5	5	4	5
13	B-434	1	3	5	4	2	4	5	5	3	5	4	5	4	5	4	3	2	1	3	6	0	5	5	4	5
14	B-411	1	2	4	3	0	2	3	0	0	2	0	1	2	1	5	4	0	5	5	2	5	4	3	3	4
15	B-410	2	1	4	4	2	0	1	0	0	0	0	0	3	3	5	3	1	4	1	1	6	3	1	2	1
16	B-413	0	3	5	5	3	2	2	1	1	4	3	3	4	1	2	4	0	3	3	2	2	3	2	2	0
17	B-409	3	5	5	4	4	2	3	0	5	3	3	1	5	3	3	5	1	3	3	5	0	3	3	3	2
18	B-245	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	18	0	0	0	0
19	B-521	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	1	18	0	0	0	1
20	B-433	4	3	2	4	4	0	5	1	0	3	2	2	4	3	5	4	1	5	3	3	1	5	3	2	1
21	B-441	4	3	5	4	3	4	3	3	5	5	4	5	3	4	5	5	1	5	5	8	0	5	2	5	2
22	B-405	4	2	5	4	4	3	5	3	0	5	3	1	2	4	3	3	0	5	3	4	2	5	2	5	1
23	B-438	3	2	5	3	2	1	4	1	0	0	1	4	3	1	3	4	1	3	3	1	2	5	3	5	2
24	B-427	1	1	5	3	0	3	4	2	0	3	3	3	3	3	3	3	1	5	4	2	0	5	2	3	2
25	**B-447**	5	5	4	4	4	4	4	1	0	2	5	5	1	4	5	5	2	5	4	6	1	3	2	3	2
26	B-442	0	1	2	5	3	3	4	1	2	2	4	4	4	5	1	5	2	5	3	4	11	4	2	2	2
Symptomless		4	4	10	8	3	2	11	2	4	5	1	6	6	9	11	6	0	13	7			15	9	8	9
Killed (5)		4	3	2	2	4	6	3	5	9	5	5	4	2	1	3	2	7	2	2			2	2	2	4

Border limits of the scale of evalutaion: 0 = all germlings killed, 5 = all seedlings survived. (1) Wheat varieties; *T. monococcum* cv Alkor, *T. turgidum* cv Hegyes and all others are *T. aestivum*. (2) Strains 1-13 refer to anastomosis groups AG-1-AG-11 and AG-E of *R. solani* of CBS collection, respectively, strains 14-20 are Hungarian isolates of *R. solani* and *R. stahlii* (21); all are *Thanatephorus* anamorphs. *Rhizoctonia* strains 22-26 are anamorphs of *Waitea* (22), *Cerathorrhiza* (23, 24), *Ceratobasidium* (25) and *Athelia* (26), respectively. (3) Code; accession numbers of the Mycological Collection of Plant Protection Institute HAS (WDCM824). The bold labelled strains had been isolated of wheat. (4) Symptomless = no visual symptoms observed at 8th days. (5) Killed = no one survived over 8th days.

(AG-4, *Phaseolus* sp., England, CBS 340.51), B-418 (AG-5, *Zeae mays* L., Netherlands, CBS 339.84), B-419 (AG-6, *Conyza canadensis* (L.) Cronquist, CBS 137.82, USA), B-420 (AG-7, soil, Japan, CBS 214.84), B-421 (AG-8, *Triticum aestivum* L., Australia, CBS 101782), B-422 (AG-9, *S. tuberosum*, USA, CBS 970.96), B-423 (AG-10, *T. aestivum*, USA, CBS 971.96), B-424 (AG-11, *Lupinus angustifolus* L., Australia, CBS 974.96), B-434 (AG-E, *Malus* sp., Netherlands, CBS 340.84).

R. solani strains isolated in Hungary: B-411 (*S. tuberosum*, cv Desirée) and B-410 (*S. tuberosum cv* Kisvárdai rózsa), B-413 (*Malus domestica* L.); B-409 (*Hibiscus rosa-chinensis* L., imported of Lybia, Tripoli); B-245 (*Allium cepa* L., imported of China, Henan); B-521 (*Impatiens balsamina* L.); B-433 (*Festuca arundinacea* Schreb.).

R. stahlii Burgeff (teleomorph: *Thanatephorus* sp.): B-441 (*Platanthera chlorantha* (Custer) Rchb.), Germany, CBS 119.92).

R. fragariae S. Husain & W.E. McKeen (teleomorph: *Ceratorhiza fragariae* (S.S. Husain & W.E. McKeen) R.T. Moore): B-438 (*Fragaria* × *ananassa* Duchense, Canada).

R. ramicola W.A. Weber & D.A. Roberts (teleomorph: *Ceratorhiza ramicola* (W.A. Weber & D.A. Roberts) R.T. Moore): B-427 (*Pittosporum tobira* (Thunb.) W. T. Aiton, Florida, USA, CBS 400.51).

R. cerealis E.P. Hoeven (teleomorph: *Ceratobasidium cereale* D.I. Murray & Burpee): B-447 (*T. aestivum* L., Germany, CBS 559.77).

R. zeae: B-405 (mixed grass of *Festuca* and *Lolium*, Hungary).

Athelia rolfsii (Curzi) C.C. Tu & Kimbr. (Syn: *Scelotium rolfsii* Sacc.): B-442 (*S. tuberosum*, Italy, CBS 464.48).

The strains were maintained on potato dextrose agar (Merck, Darmstadt, Germany) amended with 2 g soya peptone L44 (Oxoid, Basingstoke, UK).

2.3. Test for Pathogenicity

The potting medium was made by mixing forest soil with peat before autoclaving (1.15 atm per 20 min), at the ratio of 3:1.

The soil was inoculated with *Rhizoctonia* by the following manner: the sterile soil prepared as above was admixed with chickpea seeds previously infected with the pathogen (10 seeds per 250 g pot), than incubated 96 hours at 26˚C - 28˚C for evolving the mycelial net. The seeds were put on the surface of infested soil (1 × 1 cm), than covered with 5 mm layer of sterile soil. Sterile distilled water was used to moist the surface (15 mL per pot), and covered with plastic wrap layer to avoid dessiccation. Subsequently, the pots were evaluated each day counting the emerged germlings, and observing the occurrence of disease symptoms (damping off and leaf spots). The height of seedlings was regularly measured to nearest millimetre to follow the dinamics of growth. The control plants were grown up in *Rhizoctonia* free soil. The growth inhibition was calculated as a ratio between control and treated plants.

When the coleptyles of control plants had been fully developed (8 days after emergence of first germling) the pathological status of all seedlings was evaluated, their height and mass of measured to nearest millimetre and milligramm, respectively. Inhibition rates were calculated as related to control. The percentages were transformed into probit values, and this transformed data were analyzed according to Sváb [33]. The state of roots was assessed as well, and tissue sections were examined under microscope in cases where no visual symptoms were observed. The method was discussed in detail previously [23]. The following six fold scale was used to assess the tolerance of test plants at the 8th days: 0 = all seedlings were destroyed; 1 = the majority of seedlings was dead, but at least one survivor was presented either symptomless or bearing severe symptoms (the coleoptyle and the roots damaged, the root neck scoring), 2 = less than half of seedlings survived, the survivor were either symptomless or bearing severe symptoms (the coleoptyle and the roots damaged, the root neck scoring), 3 = more than half of seedlings survived, the symtoms of disease syndrome largely varied, 4 = most of seedlings were similar to control, but as minimum as one diseased, 5 = none of seedlings had any symptoms visible to the naked eye. The results of observations were compiled into data matrix ((19 wheat varieties + 4 small grains) × 26 *Rhizoctonia* strains).

Surviving specimens were grown up to 21 days and their development and evolution of disease syndrome were observed.

2.4. Data Analysis

Box plot analysis was applied to demonstrate alterations both in tolerance of test plants and and virulence of *Rhizoctonia* strains as well as variations in growth parameters of wheat varieties as influenced by the presence of *Rhizoctonia* in the soil. The relationships between host (wheat varieties and small grains) and *Rhizoctonia* strains (potential soil borne pathogens) have been analyzed by multivariate methods: Non-linear Mapping (NLM) [34], Cluster Analysis (CA), Principal Component Analysis (PCA), Regression Analysis combined with Canonical Correlation Analysis (CCA) and Potency Mapping (PM) technique [35] following a previously described scheme [36]. PCA was carried out on the correlation matrix [37] and only the components having an eigenvalue greater

than one were included in the evaluation of data to demonstrate potential number of factors influencing on host parasite system, and the results were not delineated in details. The protocol of experiments is shown in **Figure 1**.

Statistical functions of Microsoft Office Excel 2003 (Microsoft, Redmondton, USA) and Statistica5 program (StatSoft 5.0., Tusla, USA) were used for analysis of data. The graphical presentation of result of data analysis was edited uniformly in MS Office Power Point 2003.

3. Results

3.1. Dynamics of Germination

The dynamics of germination of wheat was altered by *Rhizoctonia* strains in cultivar dependent manner (**Figure 2**). In majority of cases first germlings emerged within 2 days after sowing in *Rhizoctonia* free soil, moreover, the process was finished rapidly. The seeds of Pannon Premium sortiment germinated more uniformly, than the other varieties. In the presence of *Rhizoctonia* strains emergence had been delayed with exception of most tolerant varieties (Petrence, Emese, Alkor, Hegyes). The last day means the limit after that no further germlings outcropped. Several seeds having been destroyed, seemingly, the susceptible individual were killed either before emergence or suffered damping off within 2 - 3 days after outcropping. This ratio strongly varied, and it was not possible to carry out the statistical analysis within frames of the experimental model applied.

3.2. Syndromatic Picture of Disease

The growth of infected or diseased seedlings of wheat was conspiciously retarded than the control, however, not all symptoms of disease syndrome turned up. Even seedlings without any visible symptoms suffered damping off before full development of coleoptyle, the roots of such individuals were symptomless in many cases, although brown spots could be frequently observed on roots and root neck, even in the cases of robust survivors too. In some cases small black spots (<1 mm) were found on root necks, but these were not spread later. Leaf spots with dark brown edge were randomly observed after 6 days. The development of seedlings which survived the infec-

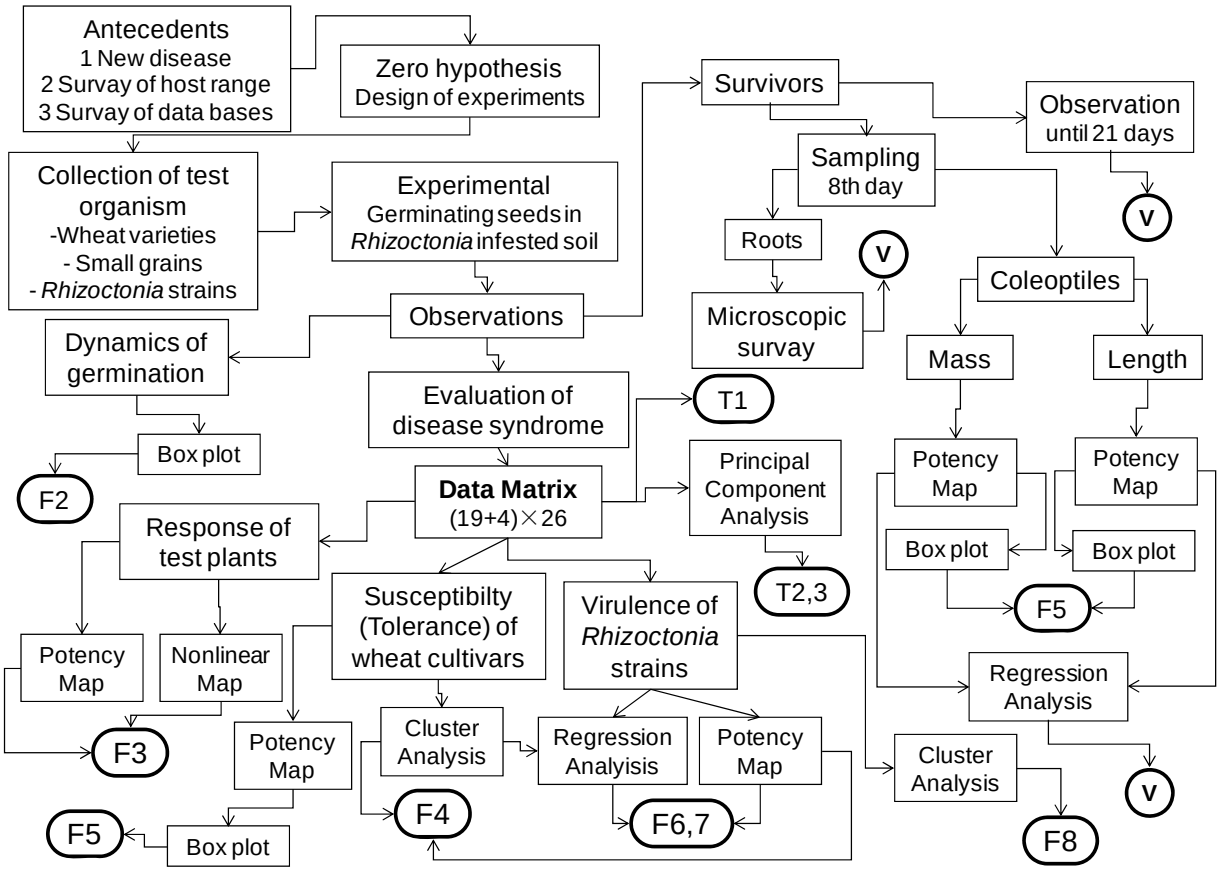

Figure 1. Flow diagram of the experimental protocol. The labels F and T in ellipses mark figures and tables in the body text where the results of computations were used for demonstration, while V means verbal interpretation of result. The zero hypothesis was: on the base of screening large number of varieties there is possible to select candidates for breeding wheat cultivar tolerant to soil borne *Rhizoctonia* infection.

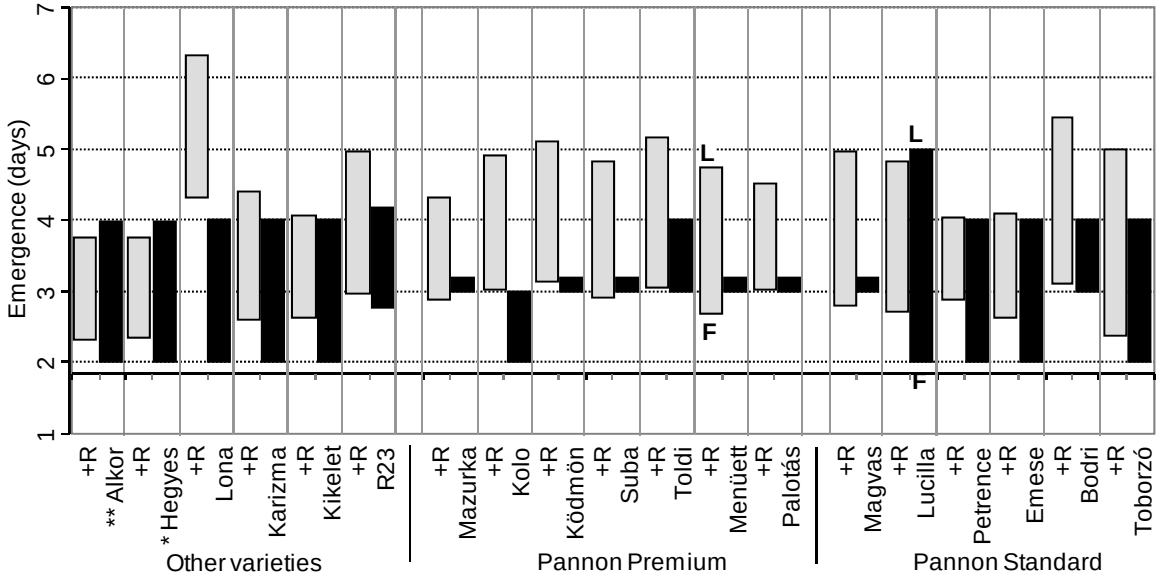

Figure 2. Susceptibility of wheat varieties to soil borne *Rhizoctonia* infection. The gray and black strips mark varieties grown up in *Rhizoctonia* infected (+R) and non-inculated (control) pots, respectively. Wheat varieties are listed on the x-axis, grouped as: other wheat varieties, Pannon Premium group of varieties, Pannon Standard group of varieties. F and L are upper and lover values of the limits of emergence of first and last seeds, respectively.

tion without root neck symptoms was retarded at various degree even two weeks later. The incidence of robust survivors varied greatly within series, the root system of such individuals was not retarded with exception of most susceptible varieties (Bodri, Lona, Lucilla, Menüett). The reaction of germlings to the presence of *Rhizoctionia* in the soil was extremely heterogenic, the coefficient of variation was over 30 percent within pot that made impossible the reliable statistical analysis. This was the reason of use of the six fold scale for assessment of plant response to *Rhizoctonia* infection. In several cases half of seedlings survived while the other half was killed independently of host/pathogen pair, most probably due to environmental effects.

The behaviour of small grains was similar to that of wheat including symptoms and development of disease syndrome. The leaf spots on *Eleusine* appeared rarely, but this was not in direct contact with virulence of *Rhizoctonia* strains. The survivors of small grains were not analyzed in details.

3.3. Susceptibility of Wheat Varieties

The test plants tolerated the *Rhizoctonia* in strain dependent manner (**Table 1**). Among *T. aestivum* cultivars Lona, Menüett and Bodri proved to be less tolerant, while Emese, Palotás and Petrence exhibited low susceptibility. The response of *T. monococcum* and *T. turgidum* was similar to more tolerant *T. aestivum* cultivars. The *Eleusine* was less susceptible than other small grains. Unfortunately, none of the test plants tolerated the majority of *Rhizoctonia* strains at high degree.

The response of wheat cultivars to *R. zeae*, the new pathogen in Europe, altered significantly of that of *R. solani* strains. This difference manifested clearly, when variety dependent virulence of *R. zeae* and associated *R. solani* strain (B-433) was compared ($R^2 = 0.317$, n = 19), although their average pathogenicity (AP) was similar (2.9 and 3.1, respectively). *R. cerealis* proved to be moderately aggressive against germinating cereals (AP = 3.6), and the similarity between activity spectrum of *R. solani* strains and *R. cerealis* was low ($r^2 < 0.23$).

The susceptibility of wheat varieties to *Ceratorhiza* (B-438) isolated of strawberry and *Athelia* (B-442) was low, however, the *C. ramicola* (B-427) isolated of orchid heavily injured five *T. aesticum* cultivars (Toborzó, Bodri, Lucilla, Toldi and Lona). The orchid symbiont *R. stahlii* (B-441) exhibited low pathogenicity. Test plants were related applying Nonlinear Mapping based on data comprised in **Table 1** (**Figure 3**). Plants similarly responding to soil borne *Rhizoctonia* infection formed a loose group, but no clear patterns were revealed.

The taxonomic position did not influence the grouping. This type of plotting did not gave information on the structure of relationships among wheat cultivars, so another nonlinear method, the Cluster Analysis was carried out (**Figure 4**).

Wheat cultivars clustered into two well separated groups on the dendrogram. *Triticum aestivum* cultivars were distributed between groups, however *T. monococum* and *T. turgidum* were in different clusters. The average tolerance level of groups was similar (A = 3.1 and B = 2.7), but alterations were revealed in the spectrum of suscepti-

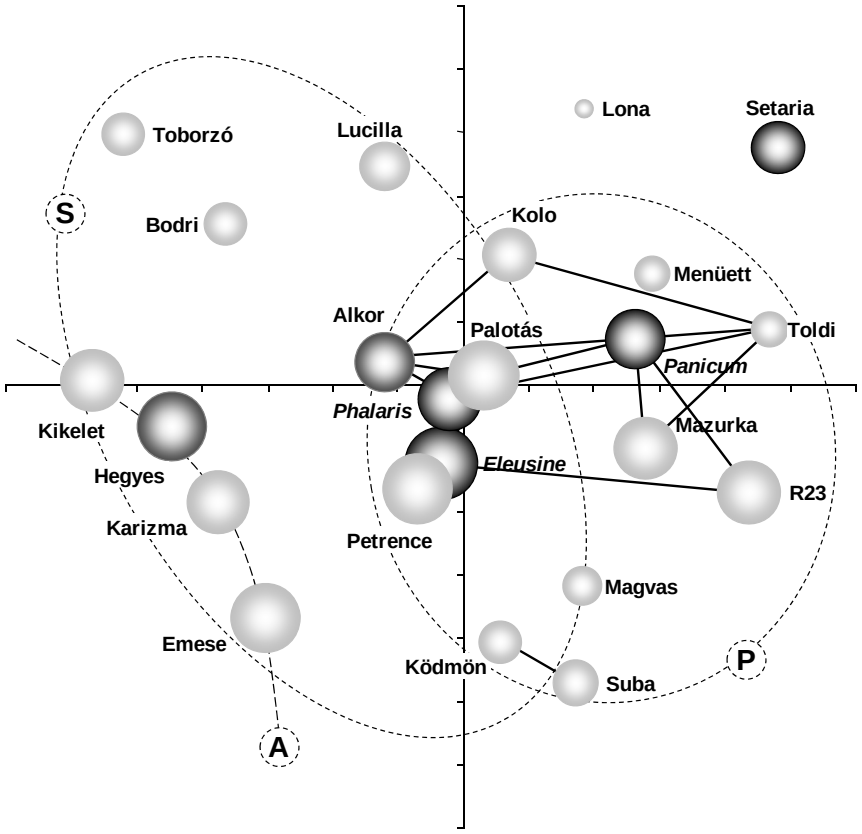

Figure 3. Nonlinear map of test plants. The size of balls is proportional to potential tolerance of test plant to *Rhizoctonias*. *Triticum aestivum* cultivars are marked with light grey, those correlated by their response to *Rhizoctonia* strains (r > 0.5) are linked with lines. The clusters P and S comprise varieties of Pannon Standard and Pannon Premium sortiments. The curve A marks possible pattern.

	Varieties	A(1)	C(2)	C(3)	AG1	AG8	AG10	T(4)	W(5)
	Toborzó	0	5	2	2	4	5	2.4	4
	Bodri	1	5	2	0	5	2	2.3	2
A	Lucilla	3	4	1	1	2	3	2.7	4
	Emese	2	4	5	2	2	4	3.5	5
	Kikelet	1	5	3	1	4	5	3.5	3
	*Hegyes	5	5	4	1	3	5	3.5	5
	Karizma	5	5	4	3	3	5	3.3	3
	Petrence	5	4	3	1	5	5	3.7	4
	Palotás	4	4	4	0	5	5	3.5	5
	R-23	5	4	2	0	5	2	3.3	4
	Mazurka	4	1	3	2	3	5	3.5	2
	Magvas	3	4	2	0	1	5	2.0	3
	Kolo	4	5	4	1	3	5	2.7	1
B	**Alkor	3	4	4	1	5	5	3.2	3
	Suba	2	2	2	0	4	3	2.6	5
	Ködmön	4	5	2	0	4	3	2.1	3
	Menüett	1	1	2	1	2	4	2.1	3
	Toldi	2	0	0	1	4	3	2.5	0
	Lona	2	2	1	0	1	3	1.2	0
Mean (6)	A	2.4	4.7	2.6	1.4	3.3	4.1	3.1	3.7
	B	3.3	3.0	2.7	0.6	3.5	4.0	2.7	2.8

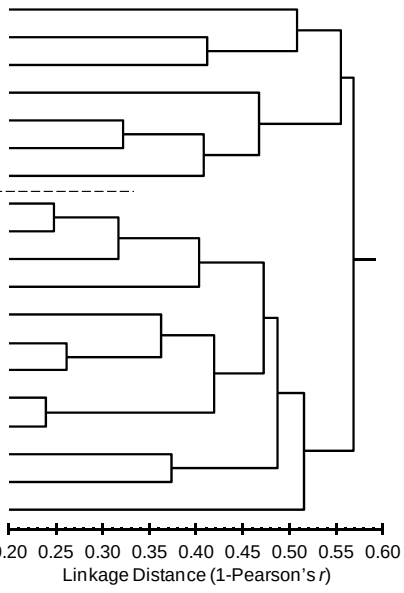

Figure 4. Grouping of wheat varieties based on their responses to soil borne *Rhizoctonia* infection. The data of Table 1 were clustered of Pearson's correlation matrix applying Unweighted Group Average method. Tolerance values to *A. rolfsii* (1), *C. cerelae* (2), *C. ramicola* (3), *Thanatephorus* anamorphs (4), *W. circinata* (5) and anastomosis groups (AG1, AG8 and AG10) of *R. solani* are of Table 1 (0 = susceptible, 5 = tolerant). (6) Average values of tolerance calculated for groups A and B.

bility. The difference was particularly spectacular in the case of AG-1 strain of *R. solani* isolated in Australia, which proved to be especially virulent against *T. turgidum* and cultivars of Pannon Standard sortiment.

3.4. Analysis of Survivors

The inhibition of growth and mass accumulation of survivors were proportional to the potential susceptibility and inhibition of emergence (**Figure 5**). The rates of inhibition of growth and mass accumulation well correlated to each other: ($p < 0.001$):

Length = 0.8149*Mass* + 0.8876 (FG = 18, $r^2 = 0.8066$). Consequently, the response of survivors can be characterized with inhibition of growth. This has importance when survivors have to be grown up for selection or further studies.

3.5. Factors Influencing the Plant Response

Four substantial factors could be revealed by PCA (**Table 2**) that explained 71% of total variation of data matrix edited of the **Table 1**. None of principal components (PCs) comprised dominant part of variation, and the cultivars clustered into four well defined groups (A = 6, B = 5, C = 3 and D = 4 varieties, respectively). The performance of majority of cultivars was determined by one dominant factor except three (Toborzó, Suba and Karizma) where two factors influenced the response (**Table 2**). All this indicates, that tolerance of wheat cultivars to soil borne *Rhizoctonia* infection was regulated by different genetic factors.

3.6. Virulence of *Rhizoctonia* Strains

Majority of *Rhizoctonia* strains holded back the germination (**Figure 6**) in the case as minimum as one cultivar. However, this effect was not strictly related to response of seedling in posterior stages of evolution of wheat/*Rhizoctonia* association. For example, the AG-1 strain of *R. solani* proved to be later more aggressive than *R. cerealis*, although these two strains altered the germination by similar manner. Seemingly, the genetic background regulating the formation of anastomosis between hyphae is not connected directly to expression of pathogenicity against wheat, the host spectrum of two AG-4 strains was different, and these strains inhibited the emergence at various degree. The AG-8 strain of *R. solani* that causes severe yield losses in China and Australia altered the dynamics of emergence insignificantly, contrary to isolates of imported propagating material of China and Netherlands (B-245, B-521).

Based on results shown in the **Figure 4** two matrices were edited of the data of **Table 1**. The Canonical Correlation Analysis resulted three significant canonic functions ($R^2 = 0.886$, 0.847, 0.684 and $\chi^2 = 103.2$, 70.5, 42.4,

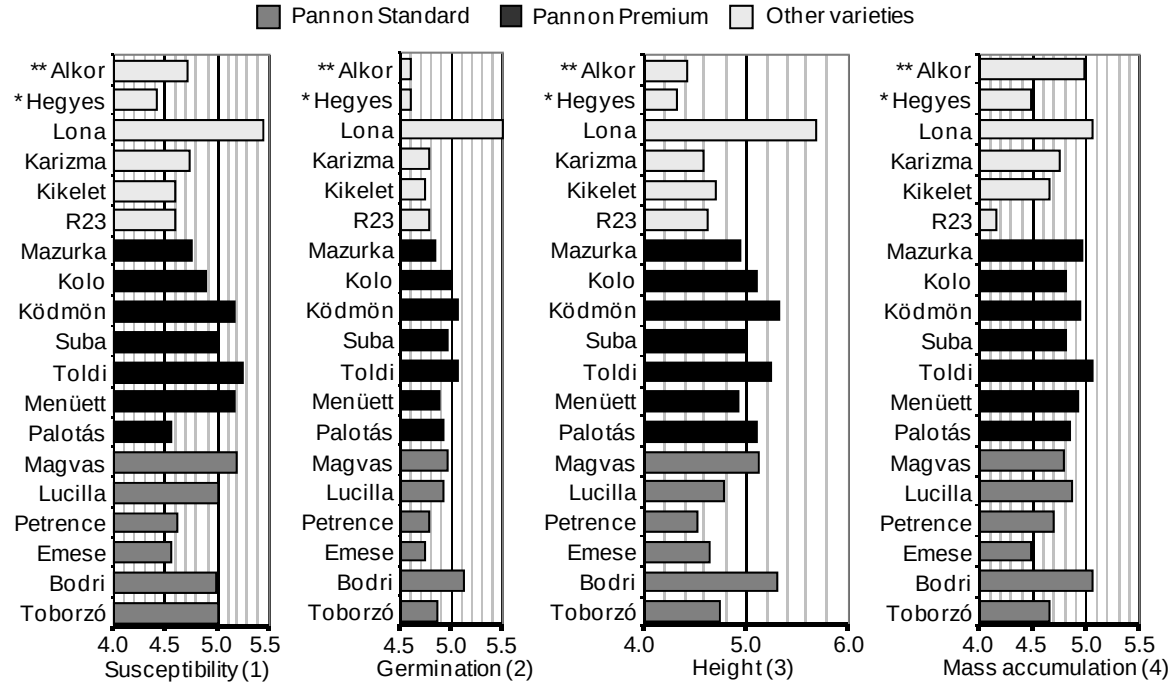

Figure 5. Responses of wheat varieties to *Rhizoctonia* strains. Inhibition rates (%) as related to control are given in probits. The varieties marked with one and two asterisks are *T. monococcum* and *T. turgidum*, while non-marked are *T. aestivum* cultivars, respectively. 1 = proportion of seedlings bearing symptoms of disease syndrome, 2 = proportion of killed germinating seeds and seedlings suffered damping off, 3 = growth inhibition, 4 = inhibition of mass accumulation. $r_{1,2} = 0.797$, $r_{1,3} = 0.772$, $r_{1,4} = 0.728$, $r_{2,3} = 0.941$, $r_{2,4} = 0.742$, $r_{3,4} = 0.802 < r_{0,01} = 0.515$ (FG = 18).

Table 2. Factors influencing the response of wheat varieties to soil-borne *Rhizoctonia* infection.

				Pricipal Components			
No.	Varieties	Groups	PS%	PC-1	PC-2	PC-3	PC-4
1	Toborzó	A	52	0.513	0.244	0.508	−0.182
2	Bodri	C	55	0.416	0.231	0.674	0.080
3	Emese	A	27	0.561	0.278	0.107	0.379
4	Petrence	D	25	0.459	0.154	0.380	0.683
5	Lucilla	-	47	0.435	0.262	0.376	0.407
6	Magvas	B	56	0.146	0.575	0.465	0.295
7	Palotás	D	27	0.426	0.436	0.312	0.536
8	Menüett	B	60	0.234	0.852	0.039	0.230
9	Toldi	B	58	0.076	0.683	0.214	0.338
10	Suba	C	48	−0.015	0.248	0.656	0.508
11	Ködmön	C	53	0.083	0.316	0.765	0.385
12	Kolo	B	43	0.241	0.701	0.404	0.146
13	Mazurka	D	34	0.306	0.332	0.028	0.744
14	R23	D	34	0.121	0.364	0.245	0.661
15	Kikelet	A	32	0.841	0.229	0.017	0.113
16	Karizma	A	31	0.626	0.095	0.500	0.251
17	Lona	B	76	0.232	0.662	0.224	0.177
18	Hegyes	A	25	0.820	0.100	0.146	0.300
19	Alkor	A	35	0.558	0.494	0.422	0.204
		Eigenvalue		3.73	3.64	3.11	3.01
	Proportion (%) of total variation			19.61	19.18	16.37	15.86

The PC loadings influencing the response of cultivars significantly were underlined. Varieties 1-6 and 7-13 are of Pannon Standard and Pannon Premium sortiments, respectively. G = Varieties influenced by the same factor were marked with the same letter. PS = potential susceptibility of variety to *Rhizoctonia* calculated in percents of the **Table 1**.

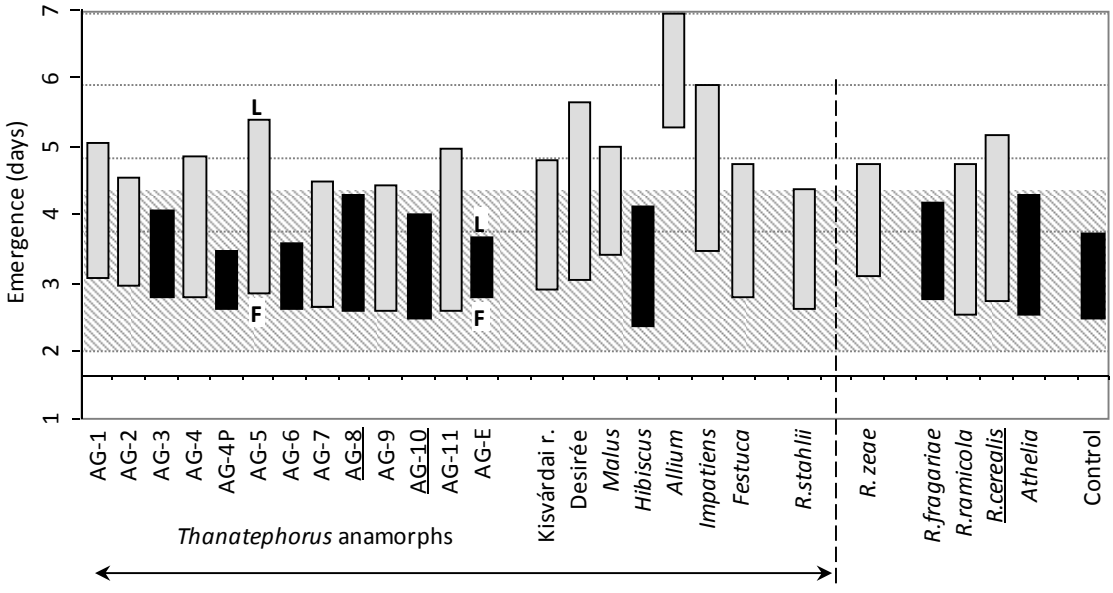

Figure 6. Influence of *Rhizoctonia* strains on germination of wheat seeds. The gray strips mark strains that significantly delayed the germination. F and L are the limits of emergence of first and last seeds calculated as average for 19 wheat cultivars. The skew lined area mark the interval (p = 0.05) of germination of seeds in *Rhizoctonia* free soil.

respectively). Plotting the strains as canonic scores by first two roots strict linear relationship was revealed, where only few strains deviated (AG-1, AG-5). However, the strains on plot of third canonic function (**Figure 7**) clustered into two well separated groups ($p < 0.001$). The members of each group differ by their virulence, and the strains of the same anastomosis group split within two clusters, indicating, that the properties responsible for separation into anastomosis groups have minor importance in formation of these clusters.

3.7. Relationships among *Rhizoctonia* Strains

The *Rhizoctonia* strains were clustered on the base of their pathogenicity against wheat cultivars (**Table 1**) based on correlation matrix applying Unweighted Pair Group Average method (**Figure 8**). No clear patterns can be revealed on dendrogram. Two highly virulent strains isolated of onion (B-245) and garden jewelweed (B-521) with poorly virulent AG-E strain separated of others, most probably due to low diversity of response data. The properties responsible for taxonomic position of strains seemingly have minor importance as the anamorphs of *Athelia*, *Ceratobasidium*, *Ceratorrhiza* and *Waitea* species formed mixed clusters with *Thanatephorus* anamorphs, indicating that these properties have no decisive role in expression of pathogenicity. The physiological characters, which take place in anastomose contact have also minor importance, for example two *R. solani* strains of AG-4 anastomosis group (B-417 and B-430) are in notably different position. Similarly, the source of strain had no influence on clustering. For example, the strains isolated of potato tubers were located in different clusters, while anamorphs (B-441 and B-427) of *Ceratorrhiza* and *Thanatephorus* of orchid and potato, respectively, were closely related. The taxonomic position of source (host plant) also had no importance, the strains of mono- and dicot plants were placed into the same cluster (see B-417 of *Citrus* and B-433 of *Festuca*). Unfortunately, we have few data on exact geographic origin, but surprisingly, all isolates of Hungarian origin were separated into the same cluster. The strains B-413 and B-521 were of imported propagating material. This might be related to role of environmental (biogeographic and bioclimatic) factors. Being typical soil habitants and forming mycelial web in soil [38,39], the soil biota plays a crucial role in the microevolution of *Rhizoctonia* species, while the assemblage of soil biota significantly depends on both structure and composition of mineral matrix and climatic conditions [40].

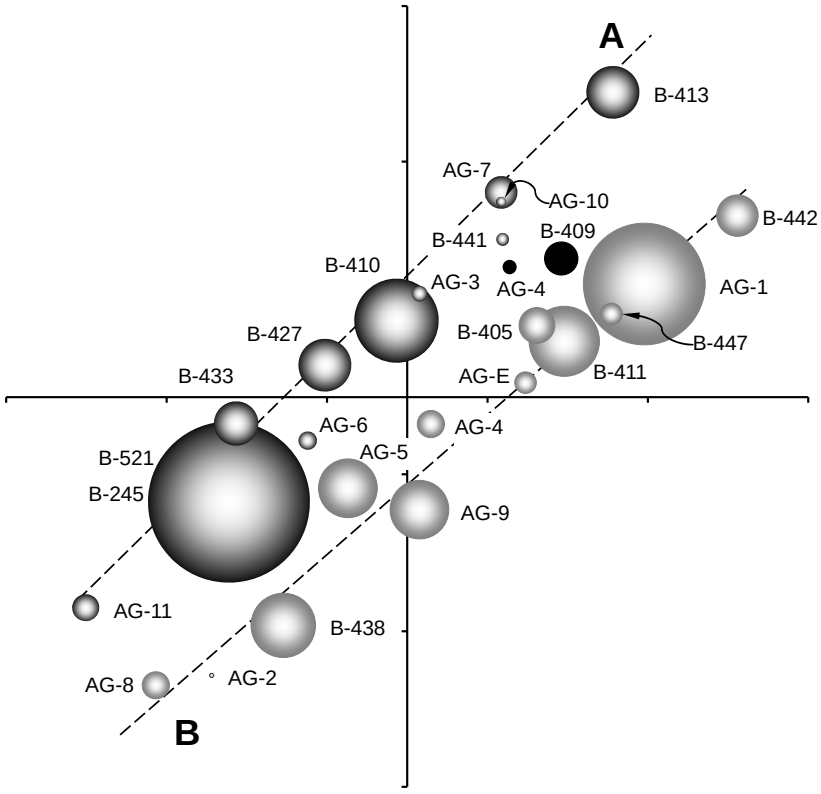

Figure 7. Separation of *Rhizoctonia* strains with Canonical Correlation Analysis. According to groups shown in Figure 3 two submatrices were edited of the data comprised in Table 1 and were related by means of CCA. Strains B-409 and B-430 (AG-4) were omitted of calculations. The size of black (A) and gray (B) balls is proportional to potential aggressivity of strains to wheat, respectively. The fitness of regression was over $r = 0.97$ ($p < 0.001$) for both function.

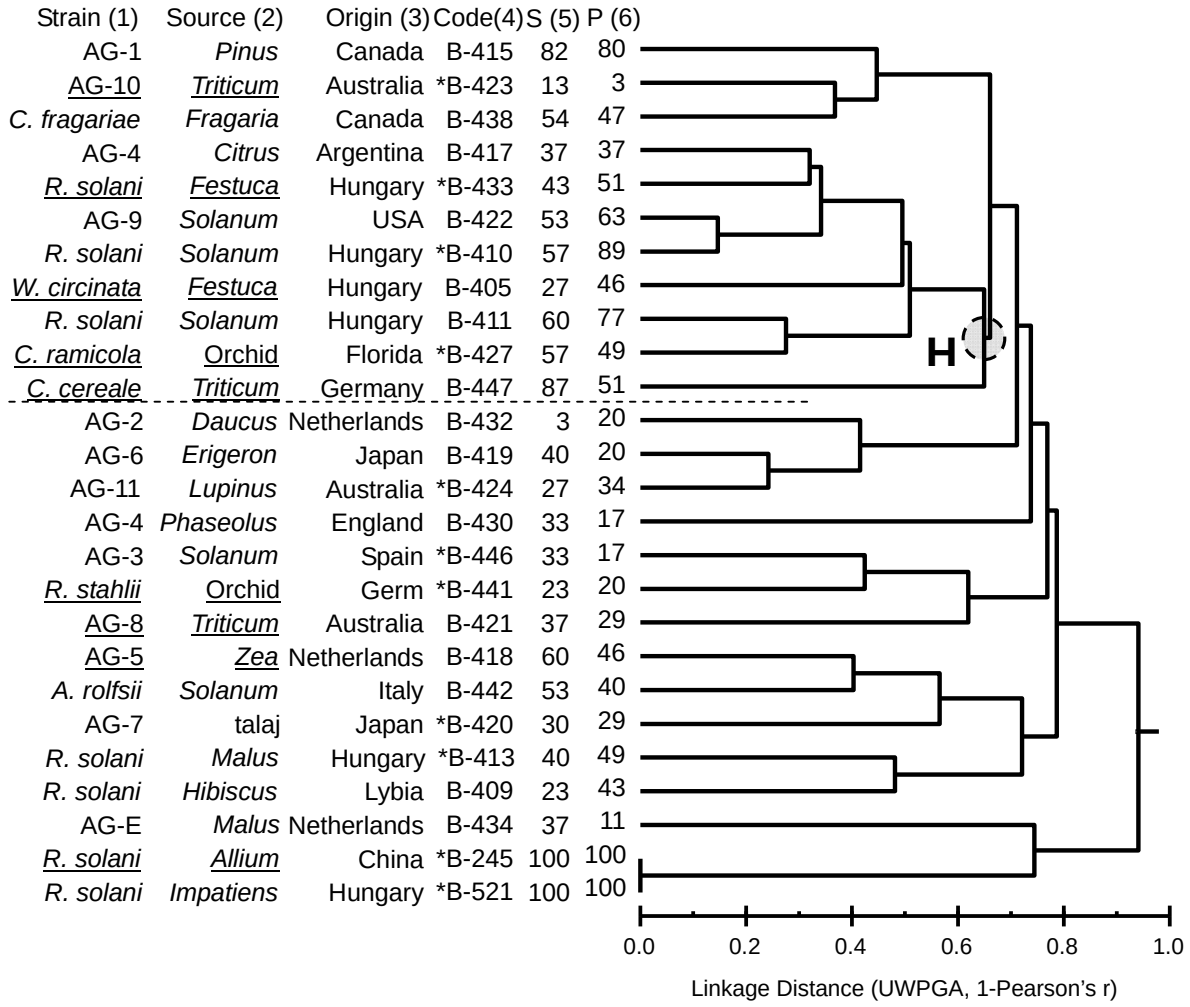

Figure 8. Grouping of *Rhizoctonia* strains based on their inhibitory effect on germinating seeds of various wheat varieties. The data of Table 1 were clustered of Pearson's correlation matrix applying Unweighted Group Average method. (1) Strains underlined were isolated of monocots, (2) Source, the monocots are underlined, (3) Location, (4) Code. The codes of members of the group I on Figure 4 are marked with asterisks. The columns S(5) and P(6) comprise potential tolerance values of varieties of Pannon Standard and Pannon Premium sortiments, respectively (0 = tolerant, 100 = susceptible). H = isolated of sources collected in Hungary.

3.8. Factors Influencing Virulence of *Rhizoctonia* Strains

The Principal Component Analysis resulted in nine notable components that explain 82% of total variance (**Table 3**). Similarly to factors influencing the response of wheat cultivars none of them was superior, the major PC has about twice more weight than the minimally significant one. The groups were sharply separated and formed by strains of various taxonomic positions. Only in two cases was the performance of strains (B-419 and B-410) affected by two factors. The quantitative aspect of virulence was not connected *per se* to groupping, for example the most virulent AG-1 strain of *R. solani* (B-415) was linked to significantly less virulent AG-10 strain (B-423). The findings support the concept of multilocal character of interaction between wheat and attacking *Rhizoctonia*.

4. Discussion

We focused on soil borne *Rhizoctonia* infection that has the greatest effect on the growth and yield of wheat among soil borne pathogens [41], and the brown patch disease became devastating in last two decades. This might be related to the changes in both pest management practices and cultivation techniques that resulted the increased frequency of the specialized pathogen genotype in some geographic areas [42,43]. The metalloorganics of broad antimicrobial spectrum of activity applied formerly for seed dressing have been banned, and the monosite inhibitors either do not inhibit *Rhizoctonia* like fungi or they loose activity rapidly due to acquired resistance. Moreover, the formerly dominant tillage based wheat

Table 3. Factors influencing virulence of *Rhizoctonia* strains against wheat cultivars.

Rhizoctonia strains				Principal Components								
No.	Code	Group	PV%	PC-1	PC-2	PC-3	PC-4	PC-5	PC-6	PC-7	PC-8	PC-9
1	B-415	A	82	0.03	−0.24	0.07	−0.07	0.15	0.16	*0.81	0.07	0.27
2	B-432	B	13	0.11	0.11	−0.05	−0.14	−0.02	*0.94	0.04	0.10	0.03
3	B-446	C	24	0.09	0.27	0.05	0.07	*0.89	−0.06	0.28	0.14	0.02
4	B-417	D	32	0.47	0.27	*0.64	0.26	0.14	0.15	0.03	0.28	0.22
5	B-430	D	20	0.04	−0.26	*0.90	−0.04	−0.10	−0.04	0.11	0.11	0.02
6	B-418	E	52	0.14	0.03	0.14	0.20	0.14	0.16	0.09	*0.90	0.01
7	B-419	DF	25	−0.07	0.16	*0.76	0.17	0.20	*0.53	0.08	0.07	0.01
8	B-420	F	36	−0.05	0.22	−0.14	*0.85	0.05	−0.04	0.01	0.23	0.16
9	B-421	C	32	0.17	0.20	−0.01	−0.10	*0.51	0.18	−0.40	0.48	0.31
10	B-422	G	52	0.41	*0.56	0.45	0.01	0.21	0.08	0.41	−0.01	0.17
11	B-423	A	19	0.31	−0.44	0.19	0.09	0.25	0.17	*0.57	0.19	−0.35
12	B-424	B	33	0.17	0.11	0.33	0.13	0.27	*0.78	0.20	0.05	0.03
13	B-434	F	28	−0.08	0.17	0.17	*0.61	0.37	0.07	−0.25	−0.31	−0.16
14	B-411	H	58	*0.67	−0.19	0.38	0.02	0.34	0.16	0.17	−0.04	0.14
15	B-410	HG	64	*0.54	0.31	0.36	−0.14	0.07	0.09	*0.51	−0.03	0.34
16	B-413	F	48	0.37	−0.33	0.22	*0.59	0.13	−0.01	0.06	0.02	0.49
17	B-409	I	36	0.07	−0.01	0.06	0.10	0.11	0.05	0.17	0.08	*0.93
18	B-245	G	99	0.00	*0.93	−0.03	0.13	0.05	0.10	−0.14	0.07	−0.04
19	B-521	G	99	0.00	*0.93	−0.03	0.13	0.05	0.10	−0.14	0.07	−0.04
20	B-433	H	42	*0.61	0.11	0.31	−0.21	0.16	−0.04	0.19	*0.51	0.22
21	B-441	H	47	0.24	−0.19	0.02	0.15	*0.68	0.26	0.02	0.03	0.15
22	B-405	C	20	*0.67	0.20	−0.05	0.22	0.30	0.02	0.09	0.10	0.04
23	B-438	H	38	*0.56	−0.25	0.24	0.11	−0.02	0.39	0.44	0.12	0.07
24	B-427	H	27	*0.60	−0.03	0.34	0.48	0.35	0.04	0.10	−0.06	−0.22
25	B-447	H	54	*0.86	−0.05	−0.15	−0.09	−0.11	0.11	−0.10	0.17	0.001
26	B-442	F	41	0.19	0.19	0.36	*0.60	−0.14	−0.04	0.05	*0.50	0.01
Eigenvalues				3.81	3.10	2.96	2.45	2.38	2.22	2.15	1.95	1.80
Proportion of total variance				14.6	11.9	11.4	9.4	9.2	8.5	8.3	7.5	6.9

Strains 1-13 refer to anastomosis groups AG-1-AG-11 and AG-E of *R. solani*, respectively, of CBS collection, strains 14-20 are Hungarian isolates of *R. solani* and *R. stahlii* (21), all are *Thanatephorus* anamorphs. *Rhizoctonia* strains 22 - 26 are anamorphs of *Waitea* (22), *Cerathorrhiza* (23, 24), *Ceratobasidium* (25) and *Athelia* (26). PV% = potential virulence of *Rhizoctonia* strains against wheat cultivars calculated of the **Table 1** applying Potency Mapping. The PC loadings influencing the virulence of strains significantly, were marked with asterisks. The small grains were omitted of calculations.

cropping system in dryland farming has been changed to minimum or not-till cropping systems that favours to survival of mycelial web forming *Rhizoctonia* [39,44-47]. The efforts to control soil borne infections applying various eubiotic preparations were not successful yet in large scale, mainly due to the low reproducibility of the effect in field conditions which makes the calculation of cost/benefit ratio unreliable. Actually, seed dressing results only sufficient control of pathogens threatening the cultivated plant in early stage of development. Thus the importance of selection of resistant wheat cultivars increased.

4.1. Performance of Wheat/*Rhizoctonia* Pathosystem

Rhizoctonia species are abundant in soils as mutualistic members of microbial consortium associated with plants. Their relationship with host plants may change from symbiosis (as in various orchids) to destructive parasitism, and these fungi usually do not cause visible disease symptoms. The infection may remain latent for a long period and can rapidly generalize, when environmental stress factors overhelm the homeostatic regulation of host plant, *i.e.*, the disposition of host favours to pathogen either in phyllosphere of in roots. The amount of thallus varies within large limits (0.09 - 6 ng per g tissue) in symptomless plants [48]. The symptomatic picture is usually variable to a high extent as stunting growth, decrease in mass accumulation, leaf spots of various size, deformations of various organs and rooting tissues can be observed on infected plant alone or in combinations together, the disease syndrome may evolve rapidly to a fatal consequence

in formerly symptomless host (damping off and wilting). In our experiments all the above mentioned variations were observed, and the overall susceptibility of one respective variety influenced only the frequency of various symptoms of disease syndrome caused by soil borne *Rhizoctonia* infection independently on taxonomic position or origin of the strains. This indicates that some traits used for taxonomic classification can not be tightly associated with the properties determining the character of wheat/*Rhizoctonia* interaction. The small black spots that were occasionally observed on scutellum and mesocotyl of surviving individuals may be result of hypersensitive reaction (HR) suggesting the defense mechanism against *Rhizoctonia* attack rapidly activates. Further studies requested to connect this property with wheat response to soil borne *Rhizoctonia* infection as the involvement or manipulation of HR into breeding programs may open door towards to the control of brown patch disease [49]. Furthermore, the role of *Rhizoctonia* toxins in pathogenesis has to be elucidated as well as factors regulating development of survivors in presence of *Rhizoctonia* should be identified.

The genetic background of tolerance to *Rhizoctonias* is not elucidated yet. Discoveries show that both anatomic and physiological features are involved into manifestation. Thus, the structure and composition of cell wall might have importance; the resistance to fungal xylanase is a tolerance factor [50]. The multivariate analysis of our experimental results supports the multigenic character of wheat response, although no dominant factor was revealed. Unfortunately, the screening of tolerance to *Rhizoctonia* in microscale provocative experiments can give data for only preliminary selection of wheat lines, and the survivors should be evaluated in field conditions as well [51]. Nevertheless, the data obtained are encouraging and on our opinion the screening of gene banks applying the method demonstrated here can result germlings useful for further manipulations (for example cultivars Emese, Petrence and Toldi). The high variation observed in response of wheat to soil borne *Rhizoctonia* might be caused by lack of preliminary selection of tested plants.

Members of the genus *Rhizoctonia* are considered as a complex mixture of filamentous fungi, having in common the possession of a non-spored imperfect state, usually referred to as the anamorphs of five genera: *Athelia*, *Ceratobasidium*, *Ceratocystis*, *Thanathephorus* and *Waitea*. Here we included data on virulence of strains of the above five teleomorph genera, all of them attacked germinating seeds of cereals tested. Our results approve presumtion of Tomaso-Peterson and Trevathan [52] that the new for Europe pathogen, *R. zeae*, is considered as a hazard for wheat cultivation, with special regard to warm climate areas.

The multivariate analysis of experimental data revealed nine significant factors influencing the aggressivity of these strains; moreover, these factors are clearly not related to traits used for taxonomic purposes. The hyphal anastomosis interactions has been widely used for clustering of *Rhizoctonia* anamorphs within the complex, since other types of diagnostic features are usually scarce in these fungi [53]. It was considered, that the anastomosis groups are specialized to defined host plants [54]. Our experiments do not support this presumption in the case of wheat. Seemingly, the host range of single strains might be different, and reversely, strains clustered into different taxons, may have similar host range. By this reason, on our opinion, —based on presented results here, —for primary screening of wheat cultivars as minimum as six different strains should be used, including *A. rolfsii*, *C. cerealis* and *W. circinata*.

4.2. Future Prospects

The plant rhizosphere is a dynamic environment in which many parameters may influence the population structure, diversity and activity of the microbial community. The soil C:N ratio has critical role in disease incidence caused by *Rhizoctonia* as it was demonstrated by Kuhn *et al*. [55]. The roles of mycorrhiza in facilitating the acquisition and transfer of carbon (C) and nitrogen (N) is well known. A considerable amount of bidirectional transfer of C between host plant and its fungal symbiont, and a fungus-dependent pathway for organic N can be realized rapidly, thus influencing positively the stress tolerance of plant [56]. The micro- and mesofauna also can alter the disease incidence either wounding the roots [57] or decreasing the size of inoculum [58]. Induction of suppressive soil by using mixed cropping or applying eubiotic preparations is contradictory, as the iron deficiency may harm the wheat although this can be overcame by leaf nutrition. Nevertheless, we can expect new, usable knowledge of the environmental research on microbial communities and plant microbe interaction studies, which can help to design and sustain suppressive soil that would be the most convenient and economic method for comatting yield losses caused by soil-borne diseases [59].

High number of papers describe antifungal effect of various plant extracts, but only few of them report convincing comparative data on efficacy, and in minority of cases these effects have been comparable with marketed fungicides [60,61]. Most of the active substances identified are terpenoids, and on our view there is a few probability to develop potentially effective and environmentally safer alternative fungicide to xenobiotics. However, these plants might be sources for transgenic modification to upgrade the sheath blight tolerance of wheat (**Table 4**). The breading is the most promising measure to improve the tolerance to soil-borne pathogen complex, because the use of any xenobiotic has adverse effects on soil biota

Table 4. Potential candidates for transgenic manipulation of wheat for improvement of tolerance to *Rhizoctonia*.

Plant	Factor	Results (example)	Ref.
Hordeum vulgare	rip30	increased tolerance (potato)	[71]
Hordeum vulgare	chitinase	increased tolerance (tobacco)	[72]
Pennisetum glaucum	lipid transfer protein	antifungal	[73]
Triticum sp.	puroindoline	increased tolerance (rice)	[74]
Celastrus hypoleucus	pristimerin	inhibiting the formation of infective body	[75]
Celastrus hypoleucus	celastrol	inhibiting the formation of infective body	[75]
Prokaryote	5-enolpyruvoyl-shikimate-3-phosphate synthetase	Increased tolerance (wheat/*Puccinia*)	[76]
Oryza sativa	thaumatin like protein	increased tolerance (rice)	[70]
Oryza sativa	OsPR-4b gene encoding pathogenesis related protein	enhanced resistance	[77]
Solanum tuberosum	Potide G	proteinase inhibitor	[78]
Bacillus subtilis	Iturin A	Antifungal	[79]
Bacillus subtilis	flagellin	Antifungal	[80]
Raphanus sativus	defensin	increased tolerance (wheat)	[81]
Solanum tuberosum	Snakin 1	enhanced resistance	[82]
Dasypyrum villosum	unknown	tolerance to AG 8	[83]
Oryza sativa	Rice chitinase	increased tolerance (*Musa*/*Mycosphaerella*)	[84]
Oryza sativa	Rice chitinase	increased tolerance (*Eleusine*/*Magnaporthe*)	[85]
Tichoderma harzianum	glucanase	inhibiting the formation of infective body	[86]
Tephrosia villosa	defensin	increased tolerance (tobacco)	[87]
Arabidopsis thaliana	NADPH oxydase	induced resistance	[88]
Oryza sativa	ACCA synthase	induced resistance	[89]

as well as can predispose host plant to pathogen [62,63].

In our experiments individual resistant to some *Rhizoctonia*, which attack the majority of its fellows, occurred in each variety, likely to observations of other authors [64]. Such survivors can be objects of further breeding or genetic engineering. Unfortunately, the mechanistic approach on concern surrounding the genetically modified maize with *Bacillus thuringiensis* toxin or the glyphosate tolerant crops borne overall social resistance to gene technology. We should clarify, most of these concerns have no scientific base. First of all, the usefulness of genetically modified (GM) crops can be sustained with careful management [65].

Although, *Rhizoctonia* species, which cause bare-patch disease and sharp eyespot in wheat are not among the top ten pathogens [66], economic improtance of their control is increasing from severe to catasrophic yield losses reported from main wheat cultivating areas [67-69]. There is an urgent need in sustainable management strategy to combat damages induced by soil habiting *Rhizoctonia* complex, first of all, new tolerant wheat cultivars. No major resistance genes to this pathogen have been identified so far inspite of increasing efforts in studies of physiology and genetic background of wheat/ *Rhizoctonia* interaction. Nevertheless, candidates for transgenic manipulation can be selected (**Table 4**). The possible improvement of tolerance demonstrated on rice [70] might serve as example for wheat.

Some terpenoid phytoanticipins of *Pelargonium graveolens* [90], *Artemisia arborescens* [91], *Helianthus tuberosus* [92] exhibited good antifungal effect, but on our opinion the use of antifungal polypeptides seems to be more promising for transgenic manipulation. For example, a basic oligopeptide of *Bacillus subtilis* exhibited excellent and broad spectrum antimicrobial activity in our experiments [93] and would fit for control of all four important soil borne pathogens of wheat. The incorporation of *Rhizoctonia* specific mycovirus into genome of cereals also is a promising possibility [94], with special regard to root border cells. These, detached living cells form the "front line" in the soil, the special part of rhizosphere, described as a system first by Hawes [95], where the plant controls the microenvironment with these specially progammed cells [96]. The border cells have indisputably key function in plant defense controlling the dynamics of adjacent microbial populations in the soil to foster beneficial associations and inhibit pathogenic invasion [97], thus these cells are plausible objects of genetic engineering to desing wheat plant with optimum characteristics for root health management.

PCR based molecular methods enable the comparative analysis of genes involved in plant defense [98]. The task is complex because the design of PCR based molecular markers linked to the *Rhizoctonia* resistance genes of

wheat cultivars seems to be complicated due to high number of factors influencing on the type of plant response. Synergic joint action of several minor factor may result more stabile repel than a single major one as one gene mutation can not eliminate this type of defense [90]. However, we understand little about how genes interact because very few possible genetic interactions have been explored experimentally [99], thus depending on the insertion event, a particular transgene can have large effects on the entire phenotype of a plant and that these effects can sometimes be reversed when plants are moved from the glasshouse to the field [100]. On our opinion the more intense research of complex repertoire of small RNAs (microRNAs [miRNAs] and siRNAs) used as guides for post-transcriptional and epigenetic regulation would help to design new, either intraspecific or intrageneric wheat varieties. To promote research in this field it is necessary to make public the data on cereal crop genomes and discuss the contribution: what proteins, and their genome sequence organisation, play in plant defence. Although these data are extremely valuable tool for detailed analysis, and the emergence of informatic market makes difficulties as patent applications back out of scientific disputation [101], nevertheless, such free disputation in large scale of scientific community would significantly accelerate the progress in breeding of wheat tolerant to soil borne diseases.

5. Conclusions

No relationship was found between taxonomic position and origin of *Rhizoctonia* strains, indicating that traits used for their classification are not closely related to expression of their pathogenicity against wheat cultivars. Nine factors were revealed that significantly affect their virulence in wheat/*Rhizoctonia* system.

We have got empirical evidence from plant/pathogen system on the possibility of selection; the wheat phenoltype resistant to soil-borne *Rhizoctonia*, that verifies our approach of using simplified scale for disease assessment.

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REFERENCES

[1] L. Vajna and G. Oros, "First Report of *Rhizoctonia zeae* in Hungary," *Plant Pathology*, Vol. 54, No. 2, 2005, p. 250. http://dx.doi.org/10.1111/j.1365-3059.2005.01172.x

[2] G. Oros, "*Rhizoctonia* Species. Gaps to Be Filled in the Hungarian Soil Science," *Tiszántúli Növényvédelmi Fórum Előadásai*, Debrecen, 18-20 October 2004, pp. 321-329.

[3] G. J. Kövics and N. Lőrincz, "Causal Agents of Stem-Base Diseases of Winter Wheat in Eastern Hungary," In: P*roceedings of Conference*, *Resources of the Environment and Sustained Development*, Oradea, 24-26 May 2001, *Analele Universitátii din Oradea. Tom. VII. Scientific Communication Session*: *Partea I-a. Fascicula Agricultură-Horticultura*, pp. 37-44.

[4] E. I. Simay, "Outbreak of *Rhizoctonia solani* Kuhn on oat (*Avena sativa*) in Hungary," *Cereal Research Communications*, Vol. 17, No. 3-4, 1998, pp. 233-235.

[5] F. Unal and F. S. Dolar, "First Report of *Rhizoctonia solani* AG 8 on Wheat in Turkey," *Journal of Phytopathology*, Vol. 160, No. 1, 2012, pp. 52-54. http://dx.doi.org/10.1111/j.1439-0434.2011.01856.x

[6] L. Chen, Y. Y. Zhang, H. X. Liang, H. X. Liu, L. P. Du, H. J. Xu and Z. Y. Xin, "Overexpression of TiERF1 Enhances Resistance to Sharp Eyespot in Transgenic Wheat," *Journal of Experimental Botany*, Vol. 59, No. 15, 2008, pp. 4195-4204. http://dx.doi.org/10.1093/jxb/ern259

[7] M. S. Hamada, Y. N. Yin, H. G. Chen and Z. H. Ma, "The Escalating Threat of *Rhizoctonia cerealis*, the Causal Agent of Sharp Eyespot in Wheat," *Pest Management Science*, Vol. 67, No. 11, 2011, pp. 1411-1419. http://dx.doi.org/10.1002/ps.2236

[8] Y. P. Guo, W. Li, H. Y. Sun, N. Wang, H. S. Yu and H. G. Chen, "Detection and Quantification of *Rhizoctonia cerealis* in Soil Using Real-Time PCR," *Journal of General Plant Pathology*, Vol. 78, No. 4, 2012, pp. 247-254. http://dx.doi.org/10.1007/s10327-012-0390-x

[9] E. Demirci, "*Rhizoctonia* Species and Anastomosis Groups Isolated from Barley and Wheat in Erzurum, Turkey," *Plant Pathology*, Vol. 47, No. 1, 1998, pp. 10-15. http://dx.doi.org/10.1046/j.1365-3059.1998.00214.x

[10] B. Tunali, J. M. Nicol, D. Hodson, Z. Uckun, O. Buyuk, D. Erdurmus, H. Hekimhan, H. Aktas, M. A. Akbudak and S. A. Bagci, "Root and Crown Rot Fungi Associated with Spring, Facultative, and Winter Wheat in Turkey," *Plant Disease*, Vol. 92, No. 9, 2008, pp. 1299-1306. http://dx.doi.org/10.1094/PDIS-92-9-1299

[11] R. G. Grogan, "The Science and Art of Plant Disease Diagnosis," *Annual Review of Plant Pathology*, Vol. 19, 1981, pp. 333-351. http://dx.doi.org/10.1146/annurev.py.25.090187.000245

[12] J. S. Gill, K. Sivasithamparam and K. R. J. Smettem, "Soil Types with Different Texture Affects Development of *Rhizoctonia* Root Rot of Wheat Seedlings," *Plant and Soil*, Vol. 221, No. 2, 2000, pp. 113-120. http://dx.doi.org/10.1023/A:1004606016745

[13] J. Keijer, "The Initial Steps of the Infection Process In *Rhizoctonia solani*," In: B. Sneh, S. Jabaji-Hare, S. Neate, and G. Dijst, Eds., *Rhizoctonia Species: Taxonomy, Ecology*, *Pathology and Disease Control*, Kluwer, Dordrecht, 1996, pp. 149-162. http://dx.doi.org/10.1007/978-94-017-2901-7_13

[14] V. N. Armentrout anf A. J. Downer, "Infection Cushion Development by *Rhizoctonia solani* on Cotton," *Phytopathogy*, Vol. 77, 1987, pp. 623-630. http://dx.doi.org/10.1094/Phyto-77-623

[15] D. I. L. Murray, "Penetration of Barley Root and Coleoptile

Surfaces by *Rhizoctonia solani*," *Transactios of British Mycological Society*, Vol. 79, No. 2, 1982, pp. 354-360. http://dx.doi.org/10.1016/S0007-1536(82)80130-7

[16] D. K. Roget, S. M. Neate and A. D. Rovira, "Effect of Sowing Point Design and Tillage Practice on the Incidence of *Rhizoctonia* Root Rot, Take-All and Cereal Cyst Nematode in Wheat and Barley," *Australian Journal of Experimental Agriculture*, Vol. 36, No. 6, 1996, pp. 683-693. http://dx.doi.org/10.1071/EA9960683

[17] R. J. Cook and R. J. Veseth, "Wheat Health Management," APS Press, St. Paul, 1991, p. 152.

[18] W. W. Bockus and J. P. Shroyer, "The Impact of Reduced Tillage on Soilborne Plant Pathogens," *Annual Review of Phytopathology*, Vol. 36, 1998, pp. 485-500. http://dx.doi.org/10.1146/annurev.phyto.36.1.485

[19] T. C. Paulitz, R. W. Smiley and R. J. Cook, "Insights into the Prevalence and Management of Soilborne Cereal Pathogens under Direct Seeding in the Pacific Northwest, USA," *Canadian Journal of Plant Pathology-Revue Canadienne de Phytopathologie*, Vol. 24, No. 4, 2002, pp. 416-428. http://dx.doi.org/10.1080/07060660209507029

[20] D. K. Roget, S. M. Neate and A. D. Rovira, "Effect of Sowing Point Design and Tillage Practice on the Incidence of *Rhizoctonia* Root Rot, Take-All and Cereal Cyst Nematode in Wheat and Barley," *Australian Journal of Experimental Agriculture*, Vol. 36, No. 6, 1996, pp. 683-693. http://dx.doi.org/10.1071/EA9960683

[21] S. A. Frank, "Recognition and Polymorphism in Host-Parasite Genetics," *Philosophical Transaactions of the Royal Society*, *London Series B*, Vol. 346, No. 1317, 1994, pp. 283-293. http://dx.doi.org/10.1098/rstb.1994.0145

[22] R. K. Voorhees, "Sclerotial Rot of Corn Caused by *Rhizoctonia zeae*, n. sp.," *Phytopathology*, Vol. 24, No. 11, 1934, pp. 1290-303.

[23] L. Vajna and G. Oros, "Turfgrass Blight in Hungary. The Role of *Rhizoctonia solani* and *R. zeae* in the Disease Development," *Plant Protection*, Vol. 59, 2005, pp. 149-157. [in Hungarian]

[24] M. H. F. Hashmi and A. Ghaffar, "Seed-Borne Mycoflora of Wheat, Sorghum and Barley," *Pakistan Journal of Botany*, Vol. 38, No. 1, 2006, pp. 185-192.

[25] T. C. Streeter, Z. Rengel, S. M. Neate and R. D. Graham, "Zinc Fertilisation Increases Tolerance to *Rhizoctonia solani* (AG 8) in *Medicago truncatula*," *Plant and Soil*, Vol. 228, No. 2, 2001, pp. 233-242. http://dx.doi.org/10.1023/A:1004874027331

[26] J. A. Lewis, R. D. Lumsden and J. C. Locke, "Biocontrol of Damping-Off Diseases Caused by *Rhizoctonia solani* and *Pythium ultimum* with Alginate Prills of *Gliocladium virens*, *Trichoderma hamatum* and Various Food Bases," *Biocontrol Science and Technology*, Vol. 6, No. 2, 1996, pp. 163-173. http://dx.doi.org/10.1080/09583159650039368

[27] R. J. Cook, D. M. Weller, A. Y. El-Banna, D. Vakoch and H. Zhang, "Yield Responses of Direct-Seeded Wheat to Rhizobacteria and Fungicide Seed Treatments," *Plant Disease*, Vol. 86, No. 7, 2002, pp. 780-784. http://dx.doi.org/10.1094/PDIS.2002.86.7.780

[28] S. J. Barnett, D. K. Roget and M. H. Ryder, "Suppression of *Rhizoctonia solani* AG-8 Induced Disease on Wheat by the Interaction between *Pantoea*, *Exiguobacterium*, and *Microbacteria*," *Australian Journal of Soil Research*, Vol. 44, No. 4, 2006, pp. 331-342. http://dx.doi.org/10.1071/SR05113

[29] S. A. Wakelin, S. T. Anstis, R. A. Warren and M. H. Ryder, "The Role of Pathogen Suppression on the Growth Promotion of Wheat by *Penicillium radicum*," *Australasian Plant Pathology*, Vol. 35, No. 2, 2006, pp. 253-258. http://dx.doi.org/10.1071/AP06008

[30] O. V. Mavrodi, N. Walter, S. Elateek, C. G. Taylor and P. A. Okubara, "Suppression of *Rhizoctonia* and *Pythium* Root Rot of Wheat by New Strains of *Pseudomonas*," Vol. *Biological Control*, 62, No. 2, 2012, pp. 93-102. http://dx.doi.org/10.1016/j.biocontrol.2012.03.013

[31] D. V. Mavrodi, O. V. Mavrodi, J. A. Parejko, R. F. Bonsall, Y. S. Kwak, T. C. Paulitz, L. S. Thomashow and D. M. Weller, "Accumulation of the Antibiotic Phenazine-1-Carboxylic Acid in the Rhizosphere of Dryland Cereals," *Applied and Environmental Microbiology*, Vol. 78, No. 3, 2012, pp. 804-812. http://dx.doi.org/10.1128/AEM.06784-11

[32] S. Kildea, V. Ransbotyn, M. R. Khan, B. Fagan, G. Leonard, E. Mullins and F. M. Doohan, "*Bacillus megaterium* Shows Potential for the Biocontrol of *Septoria tritici* Blotch of Wheat," *Biological Control*, Vol. 47, No. 1, 2008, pp. 37-45. http://dx.doi.org/10.1016/j.biocontrol.2008.07.001

[33] J. Sváb, "Biometrical Methods in Research Work," Mezőgazdasági Kiadó, Budapest, 1981 (in Hungarian).

[34] J. W. Sammon, "A Nonlinear Mapping for Data Structure Analysis," *IEEE Transactions on Computers*, Vol. 18, No. 5, 1969, pp. 401-407.

[35] P. J. Lewi, "Spectral Mapping, a Personal and Historical Account of an Adventure in Multivariate Data Analysis," *Chemometrics and Intelligent Laboratory Systems*, Vol. 77, No. 1-2, 2005, pp. 215-223.

[36] D. Magyar and G. Oros, "Application of the Principal Component Analysis to Disclose Factors Influencing on the Composition of Fungal Consortia Deteriorating Remained Fruit Stalks on Sour Cherry Trees," In: P. Sanguansat, Ed., *Principal Component Analysis*, InTech, Rijeka, 2012, pp. 89-110.

[37] K. Pearson, "On Lines and Planes of Closest Fit to Systems of Points in Space," *Philosophical Magazine*, Vol. 6, No. 2, 1901, pp. 559-572.

[38] T. C. Paulitz and K. L. Schroeder, "A New Method for the Quantification of *Rhizoctonia solani* and *R. oryzae* from Soil," *Plant Disease*, Vol. 89, No. 7, 2005, pp. 767-772.http://dx.doi.org/10.1094/PD-89-0767

[39] J. S. Gill, K. Sivasithamparam and K. R. J. Smettem, "Size of Bare-Patches in Wheat Caused by *Rhizoctonia solani* AG-8 Is Determined by the Established Mycelial Network at Sowing," *Soil Biology & Biochemistry*, Vol. 34, No. 6, 2002, pp. 889-893. http://dx.doi.org/10.1016/S0038-0717(02)00015-9

[40] I. M. Szabó, "*The Microbiology of the Biosphere*," Akadémiai Kiadó, Budapest, 1989.

[41] A. H. A. Al-Abdalall, "Assessment of Yield Loss Caused by Root Rots in Wheat and Barley," *Journal of Food Agriculture & Environment*, Vol. 8, No. 2, 2010, pp. 638-641.

[42] R. J. Cook, "Management of Wheat and Barley Root Diseases in Modern Farming Systems," *Australasian Plant Pathology*, Vol. 30, No. 2, 2001, pp. 119-126. http://dx.doi.org/10.1071/AP01010

[43] R. A. Davis, D. Huggins, R. J. Cook and T. C. Paulitz, "Can Placement of Seed Away from Relic Stubble Limit *Rhizoctonia* Root Rot in Direct-Seeded Wheat?" *Soil & Tillage Research*, Vol. 101, No. 1-2, 2008, pp. 37-43. http://dx.doi.org/10.1016/j.still.2008.05.014

[44] J. S. Gill, K. Sivasithamparam and K. R. J. Smettem, "Influence of Depth of Soil Disturbance on Root Growth Dynamics of Wheat Seedlings Associated with *Rhizoctonia solani* AG-8 Disease Severity in Sandy and Loamy Sand Soils of Western Australia," *Soil & Tillage Research*, Vol. 62, No. 1-2, 2001, pp. 73-83. http://dx.doi.org/10.1016/S0167-1987(01)00217-3

[45] T. C. Paulitz, R. W. Smiley and R. J. Cook, "Insights into the Prevalence and Management of Soilborne Cereal Pathogens under Direct Seeding in the Pacific Northwest, USA," *Canadian Journal of Plant Pathology-Revue Canadienne de Phytopathologie*, Vol. 24, No. 4, 2002, pp. 416-428. http://dx.doi.org/10.1080/07060660209507029

[46] C. E. Pankhurst, H. J. McDonald, B. G. Hawke and C. A. Kirkby, "Effect of Tillage and Stubble Management on Chemical and Microbiological Properties and the Development of Suppression towards Cereal Root Disease in Soils from Two Sites in NSW, Australia," *Soil Biology & Biochemistry*, Vol. 34, No. 6, 2002, pp. 833-840. http://dx.doi.org/10.1016/S0038-0717(02)00014-7

[47] W. T. Schillinger, A. C. Kennedy and D. L. Young, "Eight Years of Annual No-Till Cropping in Washington's Winter Wheat-Summer Fallow Region," *Agriculture Ecosystems & Environment*, Vol. 120, No. 2-4, 2007, pp. 345-358. http://dx.doi.org/10.1016/j.agee.2006.10.017

[48] M. Su'udi, J. M. Park, W. R. Kang, D. J. Hwang, S. Kim and I. P. Ahn, "Quantification of Rice Sheath Blight Progression Caused by *Rhizoctonia solani*," *Journal of Microbiology*, Vol. 51, No. 3, 2013, pp. 380-388. http://dx.doi.org/10.1007/s12275-013-3274-7

[49] E. T. Iakimova, L. Michalczuk and E. J. Woltering, "Review. Hypersensitive Cell Death in Plants—Its Mechnisms and Role in Plant Defence against Pathogens," *Journal of Fruit and Ornamental Plant Research*, Vol. 13, 2005, pp. 135-158.

[50] J. Wu, Y. Wang, S. T. Kim, S. G. Kim and K. Y. Kang, "Characterization of a Newly Identified Rice Chitinase-Like Protein (OsCLP) Homologous to Xylanase Inhibitor," *BMC Biotechnology*, Vol. 13, 2013, p. 4.

[51] J. D. Smith, K. K. Kidwell, M. A. Evans, R. J. Cook and R. W. Smiley, "Assessment of Spring Wheat Genotypes for Disease Reaction to *Rhizoctonia solani* AG-8 in Controlled Environment and Direct-Seeded Field Evaluations," *Crop Science*, Vol. 43, No. 2, 2003, pp. 694-700 http://dx.doi.org/10.2135/cropsci2003.0694

[52] M. Tomaso-Peterson and L. E. Trevathan, "Characterization of *Rhizoctonia* like Fungi Isolated from Agronomic Crops and Turfgrasses in Mississippi," *Plant Disease*, Vol. 91, No. 3, 2007, pp. 260-265. http://dx.doi.org/10.1094/PDIS-91-3-0260

[53] D. E. Carling, "Grouping in *Rhizoctonia solani* by Hyphal Anastomosis Interactions," In: B. Sneh, S. Jabaji-Hare, S. Neate and G. Dijst, Eds., *Rhizoctonia Species*: *Taxonomy, Ecology*, *Pathology and Disease Control*, III.1., Kluwer, Dordrecht, 1996, pp. 35-48. http://dx.doi.org/10.1007/978-94-017-2901-7_3

[54] V. G. Garcia, M. A. P. Onco and V. R. Susan, "Review. Biology and Systematics of the Form Genus *Rhizoctonia*," *Spanish Journal of Agricultural Research*, Vol. 4, No. 1, 2006, pp. 55-79.

[55] J. Kuhn, R. Rippel and U. Schmidhalter, "Abiotic Soil Properties and the Occurrence of *Rhizoctonia* Crown and Root Rot in Sugar Beet," *Journal of Plant Nutrition and Soil Science-Zeitschrift fur Pflanzenernahrung und Bodenkunde*, Vol. 172, No. 5, 2009, pp. 661-668. http://dx.doi.org/10.1002/jpln.200700186

[56] T. J. Lewandowski, K. E. Dunfield and P. M. Antunes, "Isolate Identity Determines Plant Tolerance to Pathogen Attack in Assembled Mycorrhizal Communities," *PLOS One*, Vol. 8, No. 4, 2013, Article ID: E61329.

[57] M. Mazzola, "Manipulation of Rhizosphere Bacterial Communities to Induce Suppressive Soils," *Journal of Nematology*, Vol. 39, No. 3, 2007, pp. 213-220.

[58] J. Varga, Z. Naár and C. Dobolyi, "Selective Feeding of Collembolan Species *Tomocerus longicornis* (Müll.) and *Orchesella cincta* (L.) on Moss Inhabiting Fungi," *Pedobiologia*, Vol. 46, No. 6, 2002, pp. 526-538. http://dx.doi.org/10.1078/0031-4056-00157

[59] M. Mazzola and Y. H. Gu, "Wheat Genotype-Specific Induction of Soil Microbial Communities Suppressive to Disease Incited by *Rhizoctonia solani* Anastomosis Group AG-5 and AG-8," *Phytopathology*, Vol. 92, No. 12, 2002, pp. 1300-1307. http://dx.doi.org/10.1094/PHYTO.2002.92.12.1300

[60] S. A. Deepak, G. Oros, S. G. Sathyanarayana, H. Shekar Shetty and S. Sashikanth, "Antisporulant Activity of Watery Extracts of Plants against *Sclerospora graminicola* Causing Downy Mildew Disease of Pearl Millet," *American Journal of Agricultural and Biological Sciences*, Vol. 2, No. 1, 2007, pp. 36-42. http://dx.doi.org/10.3844/ajabssp.2007.36.42

[61] S. A. Deepak, G. Oros, S. G. Sathyanarayana, N. P. Shetty, H. S. Shetty and S. Sashikanth, "Antisporulant Activity of Leaf Extracts of Indian Plants against *Sclerospora graminicola* Causing Downy Mildew Disease of Pearl Millet," *Archives of Phytopathology and Plant Protection*, Vol. 38, No. 1, 2005, pp. 31-39. http://dx.doi.org/10.1080/03235400400007558

[62] C. A. Bradley, G. L. Hartman, L. M. Wax and W. L. Pedersen, "Influence of Herbicides on *Rhizoctonia* Root and Hypocotyl Rot of Soybean," *Crop Protection*, Vol. 21, No. 8, 2002, pp. 679-687. http://dx.doi.org/10.1016/S0261-2194(02)00021-2

[63] H. Lee, S. E. Ullrich, I. C. Burke, J. Yenish and T. C. Paulitz, "Interactions between the Root Pathogen *Rhizo-*

ctonia solani AG-8 and Acetolactate-Synthase-Inhibiting Herbicides in Barley," *Pest Management Science*, Vol. 68, No. 6, 2012, pp. 845-852. http://dx.doi.org/10.1002/ps.2336

[64] J. B. S. Haldane, "Disease and Evolution," *La Ricera Scientifica Supplemento*, Vol. 19, 1949, pp. 1-11. *Reproduced in Current Science*, Vol. 63, No. 9-10, 1992, pp. 599-603.

[65] D. J. Lyon, A. J. Bussan, J. O. Evans, C. A. Mallory-Smith and T. F. Peeper, "Pest Management Implications of Glyphosate-Resistant Wheat (*Triticum aestivum*) in the Western United States," *Weed Technology*, Vol. 16, No. 3, 2002, pp. 680-690. http://dx.doi.org/10.1614/0890-037X(2002)016[0680:PMIOGR]2.0.CO;2

[66] R. Dean, J. A. L. Van Kan, Y. A. Pretorius, K. E. Hammond-Kosack, A. Di Pietro, P. D. Spanu, J. J. Rudd, M. Dickman, R. Kahmann, J. Ellis and G. D. Foster, "The Top 10 Fungal Pathogens in Molecular Plant Pathology," *Molecular Plant Pathology*, Vol. 13, No. 4, 2012, pp. 414-430. http://dx.doi.org/10.1111/j.1364-3703.2011.00783.x

[67] M. G. Cromey, R. C. Butler, H. J. Boddington and A. R. Moorhead, "Effects of Sharp Eyespot on Yield of Wheat (*Triticum aestivum*) in New Zealand," *New Zealand Journal of Crop and Horticultural Science*, Vol. 30, No. 1, 2002, pp. 9-17. http://dx.doi.org/10.1080/01140671.2002.9514194

[68] K. L. Schroeder and T. C. Paulitz, "Effect of Inoculum Density and Soil Tillage on the Development and Severity of *Rhizoctonia* Root Rot," *Phytopathology*, Vol. 98, No. 3, 2008, pp. 304-314. http://dx.doi.org/10.1094/PHYTO-98-3-0304

[69] M. Anees, V. Edel-Hermann and C. Steinberg, "Build up of Patches Caused by *Rhizoctonia solani*," *Soil Biology & Biochemistry*, Vol. 42, No. 10, 2010, pp. 1661-1672. http://dx.doi.org/10.1016/j.soilbio.2010.05.013

[70] K. Kalpana, S. Maruthasalam, T. Rajesh, K. Poovannan, K. K. Kumar, E. Kokiladevi, J. A. J. Raja, D. Sudhakar, R. Velazhahan, R. Samiyappan and P. Balasubramanian, "Engineering Sheath Blight Resistance in Elite Indica Rice Cultivars Using Genes Encoding Defense Proteins," *Plant Science*, Vol. 170, No. 2, 2006, pp. 203-215. http://dx.doi.org/10.1016/j.plantsci.2005.08.002

[71] M. M'hamdi, H. Chikh-Rouhou, N. Boughalleb and J. I. R. de Galarreta, "Ribosome Inactivating Protein of Barley Enhanced Resistance to *Rhizoctonia solani* in Transgenic Potato Cultivar 'Desiree' in greenhouse conditions," *Biotechnologie Agronomie Societe et Environnement*, Vol. 17, No. 1, 2013, pp. 20-26.

[72] P. A. O'Brien, G. C. MacNish and N. M. K. Milton, "Transgenic Tobacco Plants Show Different Resistance to *Rhizoctonia solani* AG 4 and AG 8," *Australasian Plant Pathology*, Vol. 30, No. 3, 2001, pp. 221-225. http://dx.doi.org/10.1071/AP01030

[73] R. Velazhahan, R. Radhajeyalakshmi, R. Thangavelu and S. Muthukrishnan, "An Antifungal Protein Purified from Pearl Millet Seeds Shows Sequence Homology to Lipid Transfer Proteins," *Biologia Plantarum*, Vol. 44, No. 3, 2001, pp. 417-421. http://dx.doi.org/10.1023/A:1012463315579

[74] K. Krishnamurthy, C. Balconi, J. E. Sherwood and M. J. Giroux, "Wheat Puroindolines Enhance Fungal Disease Resistance in Transgenic Rice," *Molecular Plant-Microbe Interactions*, Vol. 14, No. 10, 2001, pp. 1255-1260. http://dx.doi.org/10.1094/MPMI.2001.14.10.1255

[75] D. Q. Luo, H. Wang, X. Tian, H. J. Shao and J. K. Liu, "Antifungal Properties of Pristimerin and Celastrol Isolated from *Celastrus hypoleucus*," *Pest Management Science*, Vol. 61, No. 1, 2005, pp. 85-90. http://dx.doi.org/10.1002/ps.953

[76] J. A. Anderson and J. A. Kolmer, "Rust Control in Glyphosate Tolerant Wheat Following Application of the Herbicide Glyphosate," *Plant Disease*, Vol. 89, No. 11, 2005, pp. 1136-1142. http://dx.doi.org/10.1094/PD-89-1136

[77] T. Zhu, F. Song and Z. Zheng, "Molecular Characterization of the Rice Pathogenesis-Related Protein, OsPR-4b, and Its Antifungal Activity against *Rhizoctonia solani*," *Journal of Phytopathology*, Vol. 154, No. 6, 2006, pp. 378-384. http://dx.doi.org/10.1111/j.1439-0434.2006.01110.x

[78] M. H. Kim, S. C. Park, J. Y. Kim, S. Y. Lee, H. T. Lim, H. Cheong, K. S. Hahm and Y. Park, "Purification and Characterization of a Heat-Stable Serine Protease Inhibitor from the Tubers of New Potato Variety 'Golden Valley'," *Biochemical and Biophysical Research Communications*, Vol. 346, No. 3, 2006, pp. 681-686. http://dx.doi.org/10.1016/j.bbrc.2006.05.186

[79] S. M. Zhang, Y. X. Wang, L. Q. Meng, J. Li, X. Y. Zhao, X. Cao, X. L. Chen, A. X. Wang and J. F. Li, "Isolation and Characterization of Antifungal Lipopeptides Produced by Endophytic *Bacillus amyloliquefaciens* TF28," *African Journal of Microbiology Research*, Vol. 6, No. 8, 2012, pp. 1747-1755. http://dx.doi.org/10.5897/AJMR11.1025

[80] X. Y. Zhao, X. M. Zhao, Y. M. Wei, Q. X. Shang and Z. P. Liu, "Isolation and Identification of a Novel Antifungal Protein from a Rhizobacterium *Bacillus subtilis* Strain F3," *Journal of Phytopathology*, Vol. 161, No. 1, 2013, pp. 43-48. http://dx.doi.org/10.1111/jph.12015

[81] Y. Li, M. P. Zhou, Z. Y. Zhang, L. J. Ren, L. P. Du, B. Q. Zhang, H. J. Xu and Z. Y. Xin, "Expression of a Radish Defensin in Transgenic Wheat Confers Increased Resistance to *Fusarium graminearum* and *Rhizoctonia cerealis*," *Functional & Integrative Genomics*, Vol. 11, No. 1, 2011, pp. 63-70. http://dx.doi.org/10.1007/s10142-011-0211-x

[82] P. Faccio, C. Vazquez-Rovere, E. Hopp, G. Gonzalez, C. Decima-Oneto, E. Favret, A. D. Paleo and P. Franzone, "Increased Tolerance to Wheat Powdery Mildew by Heterologous Constitutive Expression of the *Solanum chacoense* Snakin-1 Gene," *Czech Journal of Genetics and Plant Breeding*, Vol. 47, 2011, pp. S135-S141.

[83] J. D. Smith, K. K. Kidwell, M. A. Evans, R. J. Cook and R. W. Smiley, "Evaluation of Spring Cereal Grains and Wild *Triticum germplasm* for Resistance to *Rhizoctonia solani* AG-8," *Crop Science*, Vol. 43, No. 2, 2003, pp. 701-709. http://dx.doi.org/10.2135/cropsci2003.0701

[84] G. Kovacs, L. Sagi, G. Jacon, G. Arinaitwe, J. P. Buso-

goro, E. Thiry, H. Strosse, R. Swennen and S. Remy, "Expression of a Rice Chitinase Gene in Transgenic Banana ('Gros Michel', AAA Genome Group) Confers Resistance to Black Leaf Streak Disease," *Transgenic Research*, Vol. 22, No. 1, 2013, pp. 117-130. http://dx.doi.org/10.1007/s11248-012-9631-1

[85] S. Ignacimuthu and S. A. Ceasar, "Development of Transgenic Finger Millet (*Eleusine coracana* (L.) Gaertn.) Resistant to Leaf Blast Disease," *Journal of Biosciences*, Vol. 37, No. 1, 2012, pp. 135-147. http://dx.doi.org/10.1007/s12038-011-9178-y

[86] S. Alamri, M. Hashem and Y. S. Mostafa, "*In Vitro* and *In Vivo* Biocontrol of Soil-Borne Phytopathogenic Fungi by Certain Bioagents and Their Possible Mode of Action," *Biocontrol Science*, Vol. 17, No. 4, 2012, pp. 155-167. http://dx.doi.org/10.4265/bio.17.155

[87] S. Vijayan, N. K. Singh, P. Shukla and P. B. Kirti, "Defensin (TvD1) from *Tephrosia villosa* Exhibited Strong Anti-Insect and Anti-Fungal Activities in Transgenic Tobacco Plants," *Journal of Pest Science*, Vol. 86, No. 2, 2013, pp. 337-344. http://dx.doi.org/10.1007/s10340-012-0467-5

[88] R. C. Foley, C. A. Gleason, J. P. Anderson, T. Hamann and K. B. Singh, "Genetic and Genomic Analysis of *Rhizoctonia solani* Interactions with *Arabidopsis*; Evidence of Resistance Mediated through NADPH Oxidases," *PLoS ONE*, Vol. 8, No. 2, 2013, Article ID: E56814. http://dx.doi.org/10.1371/journal.pone.0056814

[89] E. E. Helliwell, Q. Wang and Y. N. Yang, "Transgenic Rice with Inducible Ethylene Production Exhibits Broad-Spectrum Disease Resistance to the Fungal Pathogens *Magnaporthe oryzae* and *Rhizoctonia solani*," *Plant Biotechnology Journal*, Vol. 11, No. 1, 2013, pp. 33-42. http://dx.doi.org/10.1111/pbi.12004

[90] H. Bouzenna and L. Krichen, "*Pelargonium graveolens* L'Her and *Artemisia arborescens* L. Essential Oils: Chemical Composition, Antifungal Activity against *Rhizoctonia solani* and Insecticidal Activity against *Rhysopertha dominica*," *Natural Product Research*, Vol. 27, No. 9, 2013, pp. 841-846. http://dx.doi.org/10.1080/14786419.2012.711325

[91] C. H. Liu, W. X. Zou, H. Lu and R. X. Tan, "Antifungal Activity of *Artemisia annua* Endophyte Cultures against Phytopathogenic Fungi," *Journal of Biotechnology*, Vol. 88, No. 3, 2001, pp. 277-282. http://dx.doi.org/10.1016/S0168-1656(01)00285-1

[92] F. J. Chen, X. H. Long, M. N. Yu, Z. P. Liu, L. Liu and H. B. Shao, "Phenolics and Antifungal Activities Analysis in Industrial Crop Jerusalem Artichoke (*Helianthus tuberosus* L.) Leaves," *Industrial Crops and Products*, Vol. 47, 2013, pp. 339-345. http://dx.doi.org/10.1016/j.indcrop.2013.03.027

[93] S. Pietr and G. Oros, "Antifungal Properties of Polypeptide Antibiotics Produced by Some Strains of *Bacillus subtilis*," *Tagungsbericht der Akademie der Landwirtschafts-Wissenschaften DDR*, Vol. 253, 1987, pp. 261-266.

[94] L. Zheng, H. Q. Liu, M. L. Zhang, X. Cao and E. X. Zhou, "The Complete Genomic Sequence of a Novel Mycovirus from *Rhizoctonia solani* AG-1 IA Strain B275," *Archives of Virology*, Vol. 158, No. 7, 2013, pp. 1609-1612. http://dx.doi.org/10.1007/s00705-013-1637-3

[95] S. McGinley, "Rott Border Cells Defense Plants." ag.arizona.edu/pubs/general/resrpt2000/bordercells.pdf

[96] M. C. Hawes, U. Gunawardena, S. Miyasaka and X. Zhao, "The Role of Root Border Cells in Plant Defense," *Trends in Plant Science*, Vol. 5, No. 3, 2000, pp. 128-133. http://dx.doi.org/10.1016/S1360-1385(00)01556-9

[97] M. C. Hawes, L. A. Brigham, F. Wen, H. H. Woo and Y. Zhu, "Function of Root Border Cells in Plant Health: Pioneers in the Rhizosphere," *Annual Review of Phytopathology*, Vol. 36, 1998, pp. 311-327. http://dx.doi.org/10.1146/annurev.phyto.36.1.311

[98] S. A. Deepak, K. R. Kottapalli, K. G. Rakwal, G. Oros, K. S. Rangappa, H. Iwahashi, Y. Masuo and G. K. Agrawal, "Real-Time PCR: Revolutionizing Detection and Expression Analysis of Genes," *Current Genomics*, Vol. 8, No. 4, 2007, pp. 234-251. http://dx.doi.org/10.2174/138920207781386960

[99] F. O. Amulaka, J. N. Maling'a, M. Cakir and R. M. S. Mulwa, "Development and Characterization of Wheat Germplasm with Combined Resistance to Russian Wheat Aphid and Stem Rust (Race 'Ug99') in Kenya," *American Journal of Plant Sciences*, No. 4, 2013, pp. 767-773. http://dx.doi.org/10.4236/ajps.2013.44095

[100] S. L. Zeller, O. Kalinina, S. Brunner, B. Keller and B. Schmid, "Transgene 6 Environment Interactions in Genetically Modified Wheat," *PLoS ONE*, Vol. 5, No. 7, 2010, Article ID: E11405. http://dx.doi.org/10.1371/journal.pone.0011405

[101] G. K. Agrawal, R. Rakwal and A. Sarkar, "'Cost of Knowledge' and 'Quality of Knowledge': Looking toward Future," *International Journal of Life Sciences*, Vol. 7, No. 1, p. i. http://dx.doi.org/10.3126/ijlf.v.7i18167