Pathogens Associated with Agropyron repens (L.) Beauv. in Eastern Canada

by

N. G. Sampson

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Short Title

Pathogens of *Agropyron repens* (L.) Beauv.

Sampson

ABSTRACT

M.Sc. M.G. Sampson Dept. Plant Science

Plant Pathogens Associated with Agropyron repens (L.) Beauv. in Eastern Canada

The specificity of pathogens collected on Agropyron repens (L.) Beauv. (quack grass) in Eastern Canada and their role in the epidemiology of cereal diseases on Prince Edward Island was studied. Field surveys were conducted to determine which pathogens were present on quack grass and on cereal crops. Eleven pathogens were tested for host specificity on 52 grass species. Cross-inoculation studies were conducted with both quack grass and cereal pathogens. Thirty pathogens were isolated from quack grass, all of which have previously been reported as cereal pathogens. Three were host specific: Puccinia recondita Rob. ex. Desm. var. agropyri, Rhynchosporium secalis (Oud.) J.J. Davis and Urocystis agropyri (Preuss) Schroet. Studies are recommended to evaluate the potential of these pathogens in a biological control program. Quack grass may play a role in increasing or maintaining inoculum levels of Cochliobolus sativus (Ito & Kurib.) Drechs., Fusarium species, Bipolaris sorokiniana (Sacc.) Arx and Oliver and Septoria nodorum Berk. infecting cereal crops. Studies are recommended to consider various factors which influence disease levels in quack grass and cereal crops.

Résumé

M.Sc. M. G. Sampson Dept. Plant Science

Microbes pathogènes associés à l'Agropyron repens (L.) Beauv. dans l'Est du Canada.

Des microbes pathogènes ont été collectés sur l'Agropyron repens (L.) Beauv. (chiendent) dans l'Est du Canada pour étudier leur spécificité et leur rôle dans l'épidémiologie des maladies des céréales dans l'île du Prince Edouard. Des études de terrain ont été menées pour déterminer quels microbes pathogènes étaient présents sur le chiendent et les céréales. La spécificité de onze microbes pathogènes a été testée en regard de 52 espèces d'herbes. Des ètudes d'inoculations croisées ont éte conduites sur le chiendent et les céréales. Trente microbes pathogènes ont été isolés à partir du chiendent et la totalité d'entre eux ont été reconnus antérieurement comme étant également des microbes pathogènes vis à vis des céréales. Trois d'entre eux sont spécifiques: Puccinia recondita Rob. ex. Desm. var agropyri. Rhynchosporium secalis (Oud.) J. J. Davis et Urocystis agropyri (Preuss) Schroet. Il est recommandé d'effectuer des recherches pour évaluer la fonction potentielle de ces microbes pathogènes dans un programme de contrôle biologique. Le chiendent peut jouer un rôle important dans l'accroissement et le maintien des niveaux d'inoculum du Bipolaris sorokiniana(Ito & Kurib.) Drechs., des espèces de Fusarium, Gaeumannomyces graminis (Sacc.) Arx. et Oliver et Septoria nodorum Berk. qui infectent les céréales. Il

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est également recommandé d'entre prendre des études qui analyseront les différents facteurs qui influencent les niveaux de maladie dans le chiendent et les céréales.

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I. INTRODUCTION

1.1. Agropyron repens (L.) Beauv.

1.1.1. Distribution, habitat and economic importance

Agropyron repens (L.) Beauv. (Triticum repens L., Elytriga repens (L.) Nevski.) is an aggressive perennial grass that competes strongly with many cultivated crops. A. repens, commonly known as quack grass, couch grass, twitch grass, chiendent, is present in all major agricultural areas of the temperate regions of the world (King 1966). It is found throughout Europe, Australia, New Zealand and the temperate zones of Asia and North and South America. In North America, quack grass is in every state in the United States and in all areas of Canada (Werner and Rioux 1977).

Quack grass is a plant of open areas, mainly occurring where the native vegetation has been disturbed (Werner and Rioux 1977). The species may be found in arable land, roadsides, waste areas and along margins of rivers. It is found on dunes and/or alluvial soils, in salt and fresh water marshes and on tidal flats in maritime zones (Werner and Rioux 1977). Although tolerant of many soil types and a pH range of 4.5-8.0 (Holm et al. 1977), it is reported that most vigorous growth occurs in heavy soils and in neutral to alkaline soils (Dale et al. 1965).

Quack grass is a major weed in most areas where it occurs. It is considered as one of the world's ten worst weeds (Linscott 1970). Holm et al. (1977) also consider quack grass as one of the world's worst weeds, but ranked it as number 18. It occurs as an important weed from as far north as the limits of cultivation in the Arctic down to north temperate Africa (Pooswang et al. 1972). It is a serious weed in cereals and corn in both Europe and North America. It also competes strongly with potatoes, soybeans, vegetables, coffee, tobacco and various forage crops and can be a problem species in fruit crops and orchards (Holm et al. 1977).

Yield reduction from quack grass interference has been attributed to competition for nutrients and water and to the exudation of toxic inhibitors from roots and rhizomes (Bandeen 1966). Welbank (1961) reported that competition appeared to involve both nitrogen and water, but water was the most important factor under normal conditions. There was no evidence of interference by toxins secreted from underground parts of quack grass. However, Grümmer and Beyer (1960) isolated several phenolic substances exuded from decaying rhizomes and roots which were toxic to other plants. Similar substances were reported by Kommedahl et al. (1959), Ohman and Kommedahl (1960), Welbank (1960) and Toai and Linscott (1979). Many of these studies failed to demonstrate that toxic substances were produced from living roots and rhizomes and it may be that only dead and decaying roots and rhizomes produced an inhibitory effect (Werner and Rioux 1977).

Quack grass also interferes with crop growth indirectly by serving as a reservoir for plant pathogens which adversely affect crops. Quack grass, a perennial species, may act as a site for reproduction and an overwintering host for many crop pathogens. The role of quack grass as a source of inoculum has been reported for Cercosporella herpotrichoides Fron. (Cunningham 1965), Cochliobolus sativus (Ito. & Kur.) Drechs. (Kosyreva 1958), Fusarium species (Padwick and Henry 1933), Guaemannomyces graminis (Sacc.) Arx & Oliver (Padwick and Henry 1933), Puccinia graminis Pers. (Luig and Watson 1977), Rhynchosporium secalis (Oud.) J.J. Davis (Sprague 1950), Septoria nodorum Berk. (Harrower 1977), Septoria tritici Rob ex Desm. (Brokenshire 1975) and wheat streak mosaic (Slykhuis 1952).

In addition quack grass serves as an overwintering site for certain insects such as Mayetiola destructor (Sag.), Contarina tritici Kirby, W., Haplodiplosis equestris Wagner, B., Oscinella frit L., Chlorops pumilionis Bjerkander and Zabrus tenebriodes Goeze, which also attack crop species (Barnes 1956, Bjegovic 1957, Smith 1957).

Beneficial effects of quack grass have also been reported (Werner and Rioux 1977). It is of acceptable quality both as pasture and conserved feed (hay or silage). Total crude protein content of quack grass compares favorably with timothy (Phleum pratense L.) (Werner and Rioux 1977). The plant is one of the most effective plants for

reclaiming nutrients from municipal sewage effluent sprayed on vegetation. This greatly increases the nitrogen content of the grass, improving its use as a feed (Reed and Stephenson 1977). The vigorous, rhizomatous habit of quack grass makes it an efficient soil binder on slopes, embankments and on sandy soil. It also provides cover for wildlife.

Quack grass has also been useful as a medicinal plant, particularly the rhizomes. It is listed in most herbals and it is reported that some 250,000 pounds of rhizomes were imported annually into the United States from Europe for medicinal purposes (Henkel 1904). The rhizomes, when dried and ground, may provide a source of flour (Fernald and Kinsey 1958). A methanol extract prepared from quack grass has been effective against mosquito larvae (Aedes aegyptii L.) even in low concentrations (Supavarn et al. 1974).

1.1.2. Biology of Agropyron repens (L.) Beauv.

1.1.2.1. Botanical description

Quack grass is a herbaceous perennial, spreading by seeds and rhizomes. Rhizomes are long (up to one meter), slender, and smooth with many nodes and a scale leaf, bud or branch and fine root system at each node (Palmer and Sagar 1963). Culms are 3-12 cm tall with three to five nodes. The flat leaves may be 5-10 mm wide and 6-30 cm long, usually with scattered hairs above. Sheaths are rounded on the back, with short, clasping auricles at the apex. The ligule is membraneous and sometimes ciliated. The spike is erect, 5-30 cm long with numerous oblong, elliptic or wedge-shaped, overlapping spikelets. Spikelets are 3-8 flowered alternating in two rows on opposite sides of the axis with the broader side appressed to it. The glumes are lanceolate to oblong, blunt or pointed, 7-12 mm long and 3-7 nerved. The caryopsis is tightly enclosed by a hard lemma and palea. The lemma is 5-nerved and the palea has two rough keels (Gleason and Cronquist 1963; Palmer and Sagar 1963; Werner and Rious 1977).

1.1.2.2. Variability

Quack grass is a hexaploid with 2n = 42 (Peto 1930). However, counts of 2n = 21 and 63 (Dewey 1974) and 2n = 28, 34 and 35 (Palmer and Sagar 1963) have been reported.

Quack grass is very variable in growth and morphology. This variability has resulted in description of a large number of forms and varieties. Fernald (1933, 1950) recognized two varieties and eight forms. Jansen (1951) and Neuteboom (1975) also recognized six varieties and various forms reflecting the variation within the species with regard to a number of morphological characters. Bowden (1965) recognized only two forms, A. repens (L.) Beauv. f. repens and A. repens (L.) Beauv. f. aristatum (Schum.) Holmb. Forma repens is awnless or possesses very short awns and f. aristatum has awns 2-9 mm long. Forma repens is more common than f. aristatum in Canada (Bowden 1935).

In Canada, quack grass is usually recognized as one species (Agropyron repens) with no forms or varieties (Alex et al. 1980). Additional studies are required to determine if the species should be considered as one or if different forms or varieties should be assigned.

Plants established from seed exhibit great genetic variation due to cross pollination. Such variation is not evident within populations established from rhizomes. Bulcke et al. (1974) found clonal differences for various characters such as hairiness of plant parts, awn length, plant habit, leaf and inflorescence colour and production of dry matter by rhizomes and aerial parts. Similar differences were also reported by Raleigh et al. (1962), Palmer and Sagar (1963), Pooswang et al. (1972) and Neuteboom (1980). Raleigh et al. (1962) also reported variation among clones in the quantity of growth inhibitors produced and in the number of seed produced. Variation in response of clones to herbicides was also reported. Westra (1981) reported differential reaction of various quack grass clones to glyphosate.

Williams (1973) stated that the amount of variation in seedlings from different areas and within the study areas reflected the heterozygosity of the parent clones. Differences between genotypes demonstrated much genetic variation within the species for many morphological and physiological characters. Neuteboom (1980) reported that the

variability of quack grass on agricultural soils may also be affected by soil type or the geographical location. The combination of genetic diversity and environmental plasticity may aid in adaptation of quack grass to a wide range of conditions and may account for its success as a weed (Williams 1973).

1.1.2.3. Biology and life cycle of quack grass

Reproduction of quack grass is by seed and by rhizomes. Seed is produced but it is not considered to be an important factor in persistence of the weed (Williams 1971). It may, however, play an important role in introducing the weed into areas previously uninhabited by quack grass (Williams and Attwood 1971).

Seeds and rhizome buds germinate in the early spring. Seedlings and young shoots from rhizome buds begin to produce tillers at the 4- to 6- leaf stage and rhizomes in the 6- to 8- leaf stage (Palmer and Sagar 1963). Rhizomes start to develop at the 3- to 4- leaf stage in plants that have developed from rhizome buds (Fiveland et al. 1972). The primary rhizomes may branch and rebranch in the early part of the growing season. The rhizomes grow horizontally below the soil surface developing most rapidly during June, July and August (Evans and Ely 1935). In late summer, the tip of the rhizome becomes erect to form a primary shoot which may form a mature shoot the following season or may die from winter conditions in cold climates (Akhavein 1971). Rhizome growth ceases by the end of September (Palmer 1958). After initiating new rhizomes, the mature rhizomes deteriorate rapidly during the summer and fall months and the few that overwinter are of no consequence the following year (Johnson and Buchholtz 1962).

Over 95% of the buds remain inactive during the entire life of the rhizomes unless the rhizome apex has been removed or when the rhizome is severed from the parent plant (Johnson and Buchholtz 1962). Dormancy may be of two types: 1. apical dominance in which most of the buds along an intact rhizome do not initiate any growth (Akhavein 1971) and 2. seasonal dormancy (Johnson and Buchholtz 1962) which may be due to some gradual physiological changes taking place in the rhizome (Akhavein 1971) or due to nutrient deficiency (McIntyre 1965).

The latter type of dormancy has not been demonstrated in Britain (Ahhavein 1971) and no information is available for other areas.

Raleigh et al. (1962) reported that one plant produced 14 rhizomes which had a diameter of spread of over three meters and a total length of 154 meters. Two hundred and six shoots were produced from those rhizomes with 232 additional growing points.

The amount of rhizome produced is dependent upon the length of the photoperiod, with a greater rhizome biomass being produced under longer photoperiods (Williams 1971, Palmer 1958). A reduction in the level of light results in an increase in shoot production from rhizome buds but no increase in rhizome growth (McIntyre 1970). Rhizome growth is decreased by shading (Palmer 1958), repeated defoliation (Dexter 1938) and nitrogen deficiency (McIntyre 1965).

Flowering occurs in late June to July. Some shoots flower and set seed during the growing season while others remain entirely vegetative. Quack grass is wind pollinated and self sterile (Palmer and Sagar 1963). The amount of seed produced is highly variable and reports range from 15 to 400 seeds per plant stem, with 25 to 40 most common (Werner and Rioux 1977). This variability in seed production is probably due to the spatial isolation of single clones imposed by vegetative reproduction (MacKay 1964). Seeds ripen in August to early September and drop from the parent plants by October. The seeds possess no special morphological adaptation for dispersal and fall passively from the parent plant (Werner and Rioux 1977).

Seeds from quack grass have limited innate dormancy and germinate immediately after harvest if conditions are favorable (Sagar 1961). Chepil (1946), however, found that, under experimental conditions, seeds may be dormant for three or more years while Toole and Brown (1948) reported that two percent of seeds sown at a depth of 100 cm survived for ten years. In parts of the United States and Great Britain, some seeds germinate in autumn while in areas with colder temperatures, most seeds germinate in the spring (King 1966). Few seedlings are found in the field and it is therefore assumed that seeds are mainly important in establishing the species in a previously non-infested area (Neuteboom 1975).

In open habitats, an individual plant, in its first growing season, may form a clump as a result of subtillering of primary tillers with concomitant extensive development of the rhizome system. In the second season, patches develop from erected tips of the rhizomes of the first growing season and adjacent patches may coalesce to form a continuous stand unless the patch is contained by other vegetation (Palmer and Sagar 1963). The temporal birth pattern in natural stands in Great Great Britain displays peaks of recruitment in late spring and autumn each year with low points in mid-summer at flowering and in mid-winter (McMahon and Mortimer 1980).

This seasonal development is frequently changed by agricultural practices. The supply of nutrients and competition for them may be modified by fertilizer application. Low nitrogen levels can change the basic pattern of development by suppressing tillering which in turn may result in a significant reduction in the number of secondary rhizomes (McIntyre 1965). Increasing the nitrogen supply may result in a reduction in apical dominance (McIntyre 1965).

Decreased light intensity within the crop may have a suppressive effect on quack grass followed by rapid growth as the crop ripens (Williams 1970). The growth cycle of cereals shows that shoot emergence is fairly slow and maximum growth of shoots and new rhizomes occurs from July onwards (Carpenter 1972). The spring period of recruitment to the population may be absent in a crop due to cultivation (McMahon and Mortimer 1980). In such an agro-ecosystem, an individual plant consists of a primary shoot with two to three primary tillers and from two to four rhizomes. Clump formation does not occur in this system (Palmer 1958, Palmer and Sagar 1963).

1.1.4. Quack grass control

Quack grass is difficult to control because of its ability to propagate by rhizomes. Effective control depends upon limiting new rhizome production and increasing the death rate of existing rhizomes (Elliott 1972). The costs of quack grass control are relatively high and substantial yield responses are needed to repay the cost of treatment (Scragg 1980). Most control measures have utilized chemicals

and cultivation, but other measures including smother crops, mulching, hand digging, grazing, mowing and burning have been used (Kephart 1928).

1.1.4.1. Control by cultivation

The control of quack grass by cultivation depends upon depletion of food reserves below a critical level so that it is unable to regenerate from rhizomes or to survive winter conditions.

Shallow cultivation may bring rhizomes to the surface where they can dry out during summer days (Muhlethaler 1958). Fail (1954, 1959) showed that three to six rotary cultivations at 21-day intervals gave complete eradication. Rhizome pieces were reduced to one to three bud segments so that they could escape the effect of apical dominance. Subsequent regrowth by these rhizome buds was destroyed by the next cultivation. Shallow cultivation after harvest to encourage regrowth from fragments and thereby reducing carbohytrate reserves, will expose regrowth to the killing effects of frost (Evans 1957).

Rhizomes of quack grass buried to a depth of more than 29 cm are incapable of sending up a shoot to the surface and will eventually die (Evans 1957). Death was attributed to suffocation (Muhlethaler 1958). Permin (1980) reported that shallow ploughing followed by one or more thorough harrowings and then by deep winter ploughing reduced quack grass infestations by 65%.

1.1.4.2. Control by herbicides

In 1982, six herbicides, all of which are translocated within the plant, were recommended for control of quack grass in eastern Canada in 1982 (Anonymous 1982). Trichloroacetic acid (TCA) is a soil or foliar applied herbicide that is recommended as a pre-emergent or directed spray for grass control in sugarbeets and red beets (Anonymous 1982). Most effective control is obtained with autumn application just prior to frost (Bylterud 1958). Dalapon (2,2-dichloropropionic acid) is a foliar applied herbicide that is recommended as a spring or fall application followed by cultivation or plowing for quack grass control in a number of crops (Anonymous 1982).

Pronamide (3,5 dichloro (N-1, 1-dimethyl-2-propynl) benzamide) is a selective herbicide for quack grass control in forage legumes, blueberries, caneberries, sugarbeets, christmas trees and woody ornamental trees and shrubs (Anonymous 1979). Best control is obtained with fall application when soil temperatures are low and sufficient time is allowed before ground freeze for penetration into the soil and when the herbicide is placed with the rhizomes (Carlson et al. 1975).

Amitrole (3-amino-s-triazole), especially when combined with ammonium thiocyanate, is effective for control of quack grass (Anonymous 1982). It is recommended as a preplant application in corn, soybeans, and beans, as a post harvest application in small grains, as a directed spray in asparagus and orchards and for perennial weed control in non-crop areas. Most crops, if contacted by spray are sensitive to the herbicide (Anonymous 1979).

Atrazine (2-chloro-4-ethylamino-6-isopropylamino-s-triazine) is a widely used selective herbicide for control of broadleaf and grassy weeds in corn, sorghum, rangeland, sugarcane, turfgrass seed and conifers. It is also used for non-selective control of vegetation in chemical fallow and non-cropland (Anonymous 1979). It is applied as a preplant incorporated, preemergent or postemergent spray. Most effective control is obtained with a split application. Atrazine may be used in combination with aminotriazole/ammonium thiocynate for more effective control.

Glyphosate (N-(phosphonomethyl) glycine) is a broad spectrum, non-selective, systemic herbicide that provides a high degree of control of quack grass without residual effects on crops (Baird and Begman 1972). It is recommended as a preplant, post harvest, or directed spray in many crops and in industrial and non-crop land. The herbicide is rapidly absorbed by foliage and is translocated extensively to the rhizomes and untreated shoots (Sprangle et al. 1975).

Three new postemergent herbicides for quack grass control are in the final registration stages. BAS9052 or sethoxydim (proposed common name) (2-[1 ethoxyimino) butyl]-5-[2-ethylthio)-propyl]-3-hydroxy-2-cyclohexene-1-one] is being evaluated for selective grass control in broadleaf crops as is fluazifop-butyl (proposed common name) (Butyl 2-[4-(5-trifluoromethyl-2-pyridyloxy) phenoxy] propionate.

Both herbicides are highly active and have a wide margin of safety to a wide range of broadleaf crops (Anonymous 1981a; Anonymous 1981b). Dowco 453 [2-(4-(3-chloro-5-(trifluoromethyl)-2-pyridinyl)oxy)-phenoxy] propanoic acid is a new post emergent herbicide which is highly active, systemic herbicide with selectivity for a number of broadleaf crops. This herbicide is also still undergoing further field evaluations (Smith et al. 1982).

1.1.4.3. Other methods of control

Use of another crop has been found to be effective in eradicating quack grass infestations. Those crops found to be most effective are buckwheat, hemp, rapeseed, spring barley, peas or vetch with oats, sorghum and sunflowers (Kephart 1928). Dyke and Barnard (1976) found that growth of quack grass in barley can be lessened by a factor of two or more by undersowing the barley with ryegrass or red clover at or soon after barley sowing. The practice of undersowing spring cereals may serve as a means of slowing the spread of quack grass and provide a safeguard against its rapid spread after harvest if cultivation or spraying is delayed. Cussans (1973) stated that even with a greatly reduced annual increment of growth, it would take several years for control to be achieved. If competition is removed, conditions are ideal for quack grass to exploit its environment.

Intensive defoliation and grazing may lead to control of quack grass. Courtney (1980) found that a defoliation period of less than four weeks may give control of this weed. Hakansson (1972) found that at intervals of two weeks or longer, quack grass was likely to survive. Intensive defoliation is not in itself sufficient to control this weed and it is likely that control will only occur when there is effective additional competition from sown species. Kephart (1927) reported that sheep, hogs and geese can be an effective means of clearing out quack grass rhizomes. Other methods of control consist of mulching, hand digging, raking and burning (Kephart 1928, Lods 1931, 1932). These methods are useful in localized areas but are not treated further here.

1.1.4.4. Integrated control

Quack grass is a difficult weed to control and complete control is seldom obtained from a single method. Control is expensive (Scragg 1980) and complete eradication may never be obtained. Therefore, unless control measures continue, there can be extensive recovery during the next year (Valgardson and Corns 1974). A combination of two or more control methods may be required to obtain adequate control (Jenkinson 1977). Jenkinson (1977) recommended that, in addition to a combination of a number of control measures, detailed records and a plan of all fields be kept to pinpoint where problems may occur. He also recommended that weeds coming in from the borders of the fields be controlled.

Mortimer et al. (1978) and McMahon and Mortimer (1980) have utilized predictive models of the life history of quack grass to study the population dynamics of quack grass. It is their view that an understanding of the natural and man-managed factors that regulate the size of a weed population throughout its life cycle is necessary for long term success of an integrated control program. A totally integrated control program requires an understanding at the population level and, also, an understanding of crop/weed interactions (Mortimer 1983). An understanding of weed-crop interactions and interactions within a weed population may provide an all encompassing view of weed regulation and its practical applications.

1.2. Role of plant pathogens in the population dynamics of weeds

The impact of plant diseases on cultivated crops has been well documented. Data on disease development on weeds in natural and agro-ecosystems are scarce, except in relation to the role of weeds as reservoirs of pathogens attacking economic crops. Other information has dealt mainly with incidental reports of the occurrence of pathogens on weeds. The increasing interest in biological control of weeds with plant pathogens has resulted in an increasing effort to identify and study weed pathogens.

Most plant pathologists assume that the impact of disease in natural ecosystems is less than in agroecosystems. Plant species existing in a natural ecosystem would be expected to be genetically diverse. Wilson (1969) maintains that since weeds are so plentiful and adaptable, they possess a more elastic genetic base than other plants. As a result of this genetic diversity, these plants are expected to be sufficiently resistant to local pathogens and outbreaks are suppressed (Leppik 1974). Therefore, populations of wild plants exist in equilibrium with pathogens and possess an effective protective mechanism which enables them to survive and reproduce. Howard and Morrall (1975) found that the progression of disease with time on native prairie grasses was always very slow and most lesions did not grow significantly after the initial infection. This was probably due to the initiation of a host resistance response. There was also evidence of genetic diversity with respect to host response, as might be expected in a natural ecosystem.

Underpopulation may also partly explain why epidemics do not develop in natural ecosystems (Morrall and Howard 1975). Communities of plants in undisturbed habitats are often made up of unrelated species and a pure stand is the exception rather than the rule (Holm 1969). In such a system, the pathogen is in contact with a diversified plant population and, as a result, the chances of a pathogen coming in contact with a susceptible host is greatly reduced in comparison to a genetically uniform crop. Therefore, this would limit disease development in the community.

Many North American weeds are introduced from their native range and are far more aggressive in their new habitat than in their indigenous range. In most instances, these weeds are introduced without many of their natural enemies. These weeds may, therefore, have a fewer number of pathogens attacking them or they may be weakly parasitized by other pathogens (Wilson 1950). This is in contrast to indigenous weeds which have co-evolved with their pathogens and as such may have adapted to tolerate the presence of such pathogens. Introduced weeds, however, may not be as genetically diverse as weeds in their native habitat. This possible lack of genetic diversity and the ability of the weed to form dense populations makes introduced weeds similar to

cultivated crops in their vulnerability to ephiphytotics (Ohr 1974). As such, these weeds make suitable targets for biological control (Watson 1977).

An agroecosystem differs from a naturally occurring, undisturbed situation in that its continued existence depends upon manipulation of energy flows directed by man. The biotic environment is drastically reduced in diversity and a few selected energy paths are maximized to produce the greatest output from a single species of plant (Doutt and Smith 1969). Fewer pathogens may attack weeds in such a system. Disease development may be delayed in a cropping system. The crop may alter weed growth to affect disease development or the crop may effectively screen the weed from pathogen inoculum.

The impact of plant diseases on weeds is still largely unknown. Harper (1969) questioned whether or not epiphytotics were less frequent in natural habitats or if they have been documented only in agriculture and forestry. It is not yet known what pathogen complexes attack plants in undisturbed habitats or what conditions are responsible for disease development and the development of inherited resistance (Leppik 1970). In addition, the role in plant population regulation that these pathogens may play is unknown. They do occur in natural habitats, although their duration is shortened and the area affected is relatively small (Harper 1969). The pathogen may also be ineffective because it is too weak, sedentary or poorly disseminated, it may overwinter poorly, be restricted by climate, be suppressed by its own natural enemies, or require higher host densities for buildup to lethal levels (Templeton and Smith 1977). If a change occurs to favor the pathogen, an epiphytotic may occur.

There are few examples to illustrate the impacts of diseases on weeds. The most dramatic examples involve biological control of weeds. These examples are visual assessments of the impacts of diseases on weed populations and not on genetics or bioenergetics of weed populations (Ohr 1974). Colletotrichum xanthii Halst. causes a seedling blight or stem anthracnose on Xanthium spinosum L. (Spiny cocklebur). Infections of up to 80% in dense stands and 20% in sparse stands have been reported (Butlar 1951). Inman (1969) showed that infection of Rumex crispus L. (curled dock) by the rust Uromyces rumicis (Schum.)

Wint. significantly reduced seed weight, seed numbers, root weight and survived at the root stock until the following spring. Puccinia punctiformis (Str.) Rohl is a rust that attacks Cirsium arvense (L.) Scop. (Canada thistle). Watson and Keough (1981) reported that the effect of this disease can be very dramatic in reducing vigour and causing the ultimate death of its host. It was proposed that the complex of natural enemies present in eastern Canada is providing effective natural control in some habitats and as a result the species is not a serious weed.

The epiphytotic of Puccinia chondrillina Bub. and Syd. on Chondrilla juncea L. (Skeletonweed) in Australia provides an example of a man-induced epiphytotic created as a result of an intentional introduction of a pathogen. The rust had spread over the major part of the skeleton weed-infested area within a year after its introduction (Cullen et al. 1973). P. chondrillina appears to have a substantial impact on the growth and reproduction of its host in the field, and has apparently led to a reduction in the density of the narrow-leaved form of the weed (Burden et al. 1981).

The use of Colletotrichum gleosporioides (Penz.) Sacc. f.sp. aeschynomene as a mycoherbicide for the control of Aeschynomene virginica (L.) B.S.P. (northern jointvetch) in rice provides another example of a man-induced epiphytotic. Under natural conditions, this pathogen attacked late in the season and caused little damage. However, when sprayed onto the weed at an early growth stage, 99% of the plants had been killed within 46 days (Daniel et al. 1973).

Little is known about how these pathogens function in an ecosystem, how they affect a weed's distribution in its natural community or how they affect the evolutionary development of the plant (Ohr 1974). A more complete understanding of weed-pathogen relationships, particularly from an epidemiological point of view, would provide useful information for the development of a more integrated and ecological approach to control of these and other weeds.

1.3. Objectives

The objectives of this study were to identify and record pathogens that occur on Agropyron repens (L.) Beauv. (quack grass, chiendent),

to determine the effect of these pathogens on quack grass and associated crop species, and to determine the host range of some of these pathogens. Realization of these objectives should provide information on the existence of suppressive or regulatory effects of these pathogens on quack grass and, also, their potential for use in a biological control program.

Other objectives of this study were to determine the level of infestation of quack grass in Prince Edward Island and to determine if this grass may serve as a reservoir for pathogens attacking cereal crops in that province.

II. WEED SURVEYS AND DISEASE SURVEYS

2.1. Introduction

2.1.1. Weed surveys

The purpose of a weed survey is to determine the population density and distribution of an individual weed species or a mixture of weeds in a crop or specific area at a given time without reference to damage or loss. Weed surveys are an essential component of weed management programs (Thomas 1979).

Brown (1954) gives four main parameters by which weed distribution may be measured in a weed survey: (a) frequency of occurrence, (b) number of individuals per unit area (density), (c) cover, and (d) weight.

Frequency of occurrence is defined as the presence or absence of a species within a sampling unit without reference to the number of individuals that may be present (Klingman 1971). The sampling unit (quadrat) most often used is square, rectangular or circular in shape which may vary in size depending upon the desired accuracy. Frequency determinations are subject to error from three major sources: (1) quadrat size, (2) individual plant size and, (3) spatial distribution of individuals (Shimwell 1971).

The number of individuals per unit area or density may be determined by visual estimation or by actual counts of individuals in a series of randomly placed quadrats. Visual estimates may be of low accuracy and subject to bias, but are rapid, simple and allow data from a large number of plots to be obtained at a low cost (Klingman 1971). Actual counts may be time-consuming but are extremely accurate, allowing direct comparisons of quadrats in different areas (Shimwell 1971).

Area covered or cover is the area occupied by the vertical projection of the above ground plant parts onto the ground surface (Klingman 1971). Cover may be determined by visual estimates or by the line intercept method (Canfield 1941), point-quadrat (Levy and Madden 1933), step-point sampling (Evans and Love 1957), or leaf area index (Williams 1954, Sestake et al. 1971).

The above ground biomass or yield is the best single measure of the ecological effects of herbaceous species in that it combines plant density and plant size (Klingman 1971). This parameter is usually recorded as dry weight (g. or kg.) per unit area (m^2 or ha.) The disadvantages of this harvest method are that it is destructive and it is time-consuming.

The earliest weed surveys in Canada were conducted by Mr. H. Groh of the Canada Department of Agriculture who travelled extensively throughout Canada by train, listing all species of weeds he could see. Four thousand six hundred and sixty-six survey lists were assembled for all Canada between the years 1922 and 1947. Frequency of occurrence data was recorded for 1200 species across Canada in mimeographed Canada Weed Survey Reports (Alex 1979). Since that time, no country-wide weed surveys have been conducted, although recent surveys on a regional and/or provincial basis have been conducted (Alex 1964, Deschênes and Doyon 1982, Friesen and Shebeski 1960, Ivany 1980 and Thomas 1976, 1978a, 1978b, 1979).

Weed surveys have, in the past, had a low priority among researchers in many areas. However, they are an essential aspect of weed research in that they 1) provide data for identifying problem species, 2) can be used to identify long-term trends of weed populations and 3) provide an early indication of the appearance of potential problem weeds in an area where they were previously not reported.

2.1.2. Quack grass distribution in Canada

No extensive surveys of quack grass have been reported in Canada other than data recorded in more general weed surveys. Frankton and Mulligan (1970) reported that quack grass was common in agricultural areas of all provinces. Thomas (1976) reported that quack grass ranked 35 in frequency of occurrence on the list of species recorded in a

province-wide survey of Saskatchewan. It occurred in 4.6% of the fields in 1978 and 5.7% of fields surveyed in 1979 in Saskatchewan (Thomas 1978a, 1979). In Manitoba, quack grass ranked 17 in importance with a frequency of 12.5% (Thomas 1978b).

Quack grass is common in most areas of Ontario. However, no regional survey data are available. Alex (1964) found quack grass to be widespread in tomato and sweet corn fields in counties along the north shore of Lake Ontario and the north west shore of Lake Erie.

In Quebec, quack grass is the most important weed in cereal crops, first year pastures and small fruit crops (Deschenes and Doyon 1982). In 1980, quack grass occurred in 97-99% of the oat fields surveyed and 90-100% of the barley fields surveyed (Deschênes and Doyon 1982). Rioux (1981) reported that when quack grass occurred it infested more than 30% of the area in oat and corn fields in Kamouraska County, Quebec.

Quack grass is widespread throughout the Atlantic Provinces but, with the exception of Prince Edward Island, no extensive survey data are available. Ivany (1980) found that quack grass was the most common grass weed in cereal crops in Prince Edward Island. The weed occurred in 89.1% of the fields surveyed and ranked tenth on the list of most common weeds encountered.

2.1.3. Evaluation of disease incidence and intensity

Disease measurement is conducted to obtain quantitative data on the occurrence and development of diseases and is a vital requirement to assess the relative importance of different diseases (James 1971a). Disease incidence is the most popular paremeter measured (Horsfall and Cowling 1978). Incidence is defined as the number of plant units infected, expressed as a percentage of the total number of units assessed. Disease intensity or severity may give more information on the impact of various diseases and so may be a more meaningful parameter to measure. Disease intensity is defined as the area or volume of plant tissue that is diseased (James 1974). It may be measured by counting individual lesions, but this is time-consuming and often not considered worth the effort (Horsfall and Cowling 1978). A visual method has much more practical applications.

The degree of accuracy desired in disease assessment will vary according to the objectives of the research program. Therefore the disease assessment method utilized will not be the same in all situations (James 1971a) and will depend upon the type of data desired, the sample size, and the particular disease being assessed.

James (1974) stated that a percentage scale should be adopted as a standard because the upper and lower limits of such a scale are always defined, the scale is flexible and can be divided and subdivided according to specific needs, it is universally known and can be used to record both incidences and disease severity. Standard diagrams based upon a percentage scale (Cobb 1892; James 1971a, 1971b; Melchers and Parker 1922) are widely used for disease assessment. Diseases on cereals are assessed according to the percentage area affected by disease on individual leaves, sheaths or spikes. Roots and subcrown internodes may be assessed according to a percentage scale. Separate assessments are made if there is more than one disease present.

Horsfall and Barratt (1945) devised a disease assessment scale based upon a log scale since the grades detected by the human eye are approximately equal divisions on a log scale and follow the Weber-Fechner Law which states that visual acuity is proportional to the logarithm of the intensity of the stimulus. Horsfall and Barratt (1945) also noted that the eye actually reads diseased tissue below 50% and healthy tissue above 50%. Therefore, they established their grading system with a midpoint of 50%. Grades above or below this point were increased or decreased by a ratio of two (Horsfall and Cowling 1978). James (1974) has suggested that this method be used in conjunction with standard area diagrams and this suggestion has been widely accepted.

Whenever disease assessments are recorded, the growth stage of the crop should also be noted according to a published growth stage key. A key for cereals has been published by Large (1954) and Zadoks et al. (1974). In addition to growth stage, the plant organ assessed and the method of sampling should be recorded (James 1971a).

Sample size is determined by the variability of the disease present and the accuracy desired. It has been suggested that for areas larger than 0.004 ha, up to 50 primary tillers should be selected

at random along a path of predetermined shape (James 1971a). Lin et al. (1979) compared sampling methods and found that an X-shaped or W-shaped path covering the entire field was most accurate, particularly if disease distribution was clustered.

2.1.4. Diseases commonly occurring on cereals in the Maritime Provinces

Cereal crops account for approximately 21.5% of field crop production in the Maritime provinces (Statistics Canada 1977). The cereal crops grown, barley, wheat, oats and rye, are susceptible to a number of diseases which periodically become epidemic, causing severe yield decreases (Johnston 1969). The variation in disease levels from year to year may be related to factors such as the seed source and seed treatment (Nass et al. 1974, Sterling et al. 1977), seeding date (Sterling et al. 1977), crop management practices (Clough and Sanderson 1979), and weather variables (Clough and Johnston 1978a; Couture and Sutton 1978). Resistance to specific diseases is also present in some cultivars and may produce variations in disease intensity and distribution (Johnston 1969).

2.1.4.1. Barley

The most widespread disease occurring on barley in the Maritime provinces has been common root rot, incited by Cochliobolus sativus (Ito & Kurib.) Drechs. ex Datur. and various Fusarium species. This disease is usually present in all fields to varying degrees of severity (Clough and Johnston 1978a).

The most significant leaf diseases on barley are net blotch, caused by Pyrenophora teres (Died.) Drechsl. and spotblotch, caused by C. sativus. However, leaf scald, caused by Rhynchosporium secalis (Oud.) Davis, appears to be increasing in severity in the Maritime provinces. The disease is often favored by cool wet weather in June and July (Clough and Johnston 1978a).

Barley yellow dwarf virus infections may be severe from year to year but severe losses are usually restricted to a particular grower and it is believed that cultural practices, especially late seeding and

lack of insecticide applications may be the cause of the excessive yield loss (Johnston 1969).

Other diseases which are found but do not appear to cause damage are: speckled leaf blotch (incited by Septoria passerinii Sacc.), leaf stripe (incited by Pyrenophora gramineum (Died.) Ito. & Kurib.), leaf rust (incited by Puccinia hordei Otth.), stem rust (incited by Puccinia graminis Pers.), loose smut (incited by Ustilago nuda (Jens.) Rostr.), covered smut (incited by Ustilago hordei (Pers.) Lagerh.), and ergot (incited by Claviceps purpurea (Fr.) Tul.) (Johnston 1969). Also included in this list are powdery mildew (incited by Erysiphe graminis DC ex Merat.f.sp. hordei Marchall) (Clough and Johnston 1978a) and Selenophoma leafspot (incited by Selenophoma donacis var stomaticola Bauml.) (Sprague, A.G. Johnson) (Sampson and Clough 1979). A physiological, non-parasitic brown spot has also been reported on barley, but neither the cause nor the effect of these symptoms has been determined (Clough and Johnston 1978a).

2.1.4.2. Wheat

Powdery mildew of wheat, incited by Erysiphe graminis DC ex Merat. f. sp. tritici Marchall, is one of the major diseases occurring on both spring- and fall-seeded wheat in the Maritime provinces. Yield losses may be severe on spring wheat only in areas of winter wheat production or where high levels of nitrogen fertilizer were used (Clough and Johnston 1978a, b; Johnston 1974).

Leaf and glume blotch , incited by Septoria nodorum Berk, has been reported to occur at moderate to severe levels on spring and winter wheat. The disease is favored by frequent rains in mid-summer which provide ideal conditions for splash dispersal of conidia. It may also be severe in fields where wheat had been grown previously (Clough and Johnston 1978a).

Root rots are considered to be among the most important diseases occurring on wheat (Johnston 1969). Common root rot, incited by C. sativus and Fusarium species, is the most widespread of the root rots. Take-all, incited by Gaeumannomyces graminis (Sacc.) Arx and

Oliver var tritici Walker, is of increasing concern, particularly where growers are producing wheat each year.

Various Fusarium species have been reported to cause some culm rot, moderate to severe head blight and, in some instances node breakage. The disease appears to be of increasing importance in recent years (Clough and Johnston 1979b). Sooty moulds often occur on blighted heads (Clough and Johnston 1978b).

A number of other diseases occur but are of lesser importance. Loose smut, incited by Ustilago tritici (Pers.) Rostr., is common in most fields of "Opal" wheat. All other wheat varieties grown in the Maritime provinces are resistant to this pathogen (Johnston 1969). Leaf rust, incited by Puccinia recondita Rob. ex Desm. and stem rust, incited by P. graminis Pers. f. sp. tritici Eriks. & Henn., usually do not occur until late in the season after flowering is completed and it is not known how serious these diseases are under these conditions (Johnston 1969). Ergot (incited by C. purpurea) and barley dwarf virus may also occur on wheat but are usually at very low levels.

2.1.4.3. Oats

The most prevalent disease of oats is leaf blotch incited by Septoria avenae Frank f. sp. avenae. This disease appears to be less affected by weather patterns than other cereal leaf diseases. Overall yield loss attributed to leaf blotch may be substantial (Clough and Johnston 1978a). Drechslera avenacea (Curt. ex Cke.) Shoem. is a common pathogen isolated from oat seed (Clough and Johnston 1978b) and occurs, in most instances, concurrently with septoria leaf blotch. The overall yield reduction by this disease is unknown and on oat leaves may account for an unknown but probably considerable proportion of symptoms classified locally as "Septoria" (Clough and Johnston 1978b).

Red leaf, caused by the barley yellow dwarf virus, may cause severe losses but this is dependent on the presence and time of occurrence of the aphid vectors. The disease tends to be more severe in fields which were seeded late (Clough and Johnston 1978a).

Crown rust, incited by Puccinia coronata Cda, usually occurs on oats late in the season, especially on late-seeded oats. The effect of this disease is not known.

2.1.4.4. Rye

Rye is not extensively grown in the Maritime Provinces and diseases are generally not a problem on this crop (Clough and Johnston 1978a). Ergot, incited by Claviceps purpurea and leaf spotting incited by Cochliobolus sativus, are the most commonly occurring diseases (Johnston 1969). Other diseases that have been reported are scab (head blight), incited by Fusarium species (Johnston 1969) and sooty moulds, caused by Cladosporium and Alternaria species (Clough and Johnston 1978a).

2.1.5. Previous reports of pathogens on quack grass

Many of the pathogens occurring on quack grass have not been fully investigated because of their association with a weed species. The majority of reports, therefore, have been incidental reports or reported as part of a mycological survey. However, in some instances, those diseases which also attack economic crops have been studied in greater detail as to host range and the role of quack grass as a source of inoculum for these pathogens.

One hundred forty-three pathogens have been reported to occur on quack grass worldwide and are recorded in Tables 1, 2 and 3. Table 1 records only those pathogens reported on quack grass in the Maritime Provinces, Table 2 records all pathogens reported on quack grass in Canada excluding those listed in Table 1 and Table 3 records all those pathogens reported on quack grass outside of Canada.

Thirty-eight pathogens have been reported on quack grass in Canada and, of those, only nine have been reported to occur on quack grass in the Maritime Provinces. It should be noted that, of those pathogens reported in the Maritimes, all but two, L. anisomeres and P. trichostoma, have been reported in other areas of Canada. Most pathogens of quack grass reported in Canada have also been recorded in other areas of the world.

The low numbers of pathogens reported for the Maritime Provinces is primarily due to the lack of information available, rather than an actual low number of different pathogens present. No extensive surveys have

TABLE 1: Fungi previously reported on quack grass in the Maritime Provinces

Pathogen	Disease
Claviceps purpurea (Fr.) Tul.	Ergot
Erysiphe graminis DC ex Merat.	Powdery mildew
Leptosphaeria anisomeres Wehm.	Leaf spot
L. herpotrichoides de Not.	Leaf spot
Phyllachora graminis (Pers. ex Fr.) Fckl.	Tar spot
Puccinia coronata Cda.	Crown rust
Puccinia graminis Pers.	Stem rust
Puccinia recondita Robex Desm.	Leaf rust
Pyrenophora trichostoma (Fr.) Fckl.	Leaf spot

(Wehmeyer 1950)

TABLE 2: Fungi previously reported on quack grass in Canada excluding the Maritime Provinces

Pathogen	Disease	Reference
Agropyron mosaic virus	mosaic	Slykhuis & Baylis 1957
Barley yellow dwarf virus	red leaf	Conners 1967
Cochliobolus sativus (Ito & Kurib) Drechs.	Root rot, spot blotch	Padwick & Henry 1933
Erysiphe graminis f. sp. agropyri Marchal	powdery mildew	Cherewick 1944
Fusarium poae (Pk.) C.E. Lewis	silvertop	Berkenkamp & Meeres 1975
Gaeumannomyces graminis (Sacc.) Arx & Oliver	take-all	Russell 1930
Lagena radicola Vanterpool & Ledingham	root necrosis	Vanterpool & Ledingham 1930
Ligniera pilorum Fron. & Gaillat	virus transmission	Barr 1979
Low temperature basidiomycete	snow mold	Cormack 1948
Passalora graminis (Fckl.) Hohn	brownstripe	Conners 1967
Physoderma graminis (Busgen) de Wild	Physoderma disease	Childers 1948
Polymyxa graminis Ledingham	Virus transmission	Barr 1979
Puccinia coronata f. sp. secalis Pet.	crown rust	Arthur 1934
P. montanensis Ellis.	brown leaf rust	Cummins & Green 1966
P. striiformis West.	stripe rust	Sanford and Broadfoot 1933
Pyrenophora tritici-repentis Died.	leaf spot	Conners 1967
Pythium arrhenomanes Drechsl.	browning root rot	Conners 1967
P. graminicola Subram.	browning root rot	Conners 1967
Ramularia pusilla Unger	leaf spot	Sprague 1955
Rhizophydium graminis Schenk.	virus transmission	Barr 1973

Pathogen	Disease	Reference
Rhynchosporium secalis (Oud.) Davis	scald	Conners 1967
Selenophoma donacis (Pass) Sprague	halo spot	Conners 1967
Septoria agropyri Ell. & Ev.	leaf spot	Conners 1967
S. elymi Ell. & Ev.	leaf spot	Conners 1967
Urocystis agropyri (Pruess.) Schroet	flag smut	Conners 1967
Ustilago agrestis Syd.	stem smut	Conners 1967
U. hypodytes (Schlecht.) Fr.	stem smut	Beck 1934
U. macrospora Desm.	stripe smut	Conners 1967
Xanthomonas translucens (Jones, Johns & Reddy (Dawson) f. sp. cerealis Haborg	bacterial blight	Conners 1967

TABLE 3: Pathogens reported on quack grass exluding those reported in Canada

Pathogen	Locality	Reference
Fungi		
Alternaria tenuis auct senso. Wiltshire	U.S.A.	Anonymous 1960
Apiocarpella agropyrii Sprague	Wisconsin	Green 1950
A. graminicola (Sacc.) Mass.	U.S.A.	Anonymous 1960
A. sorghi Sacc.	U.S.A.	Sprague 1950
A. utahensis Sprague	U.S.A.	Sprague 1950
Cephalosporium gramineum Nisikada & Ikata	U.S.A., Europe	Bruehl 1957, Moore & Thurston 1970
Cladochytrium caespitis Griff & Mantil.	Europe	Sampson & Western 1941
Cladosporium graminum Pers ex. Lk.	Iowa, U.S.S.R.	Anonymous 1960, Dorokhova 1970
Colletotrichum graminicola (Ces.) Wilson	U.S.A., Europe	Bruehl, and Dickson 1950
Coniosporium rhizophilum (Preuss) Sacc.	U.S.A.	Seymour 1929
Coniothyrium sp.	U.S.S.R.	Dorokhova 1970
Curvularia geniculata (Tracew & Earle) Boed.	N. & S.Dakota	Anonymous 1960
Cyathicula furva Gradden	United Kingdom	Graddon 1977
Didymella agrostidis Dearn & House	New York	Anonymous 1960
Diccoccum asperum Corda	U.S.S.R.	Dorokhova 1970
Dothidea glumarum B & C.	U.S.A.	Seymour 1929
Drechslera halodes (Drechs) Subram & Jain	U.S.A.	Sprague 1950
Epichloe typhina (Pers. ex Fr.) Tul.	Europe	Moore 1959
Fusarium acuminatum Ell. & Ev.	U.S.A.	Sprague 1950
F. avenaceum (Fr.) Sacc.	U.S.A.	Butler & Jones 1961
F. culmorum (W.G. Sm.) Sacc.	U.S.A.	Butler & Jones 1961

Pathogen	Locality	Reference
F. equiseti (Cda.) Sacc.	U.S.A.	Anonymous 1960
F. nivale (Fr.) Ces.	U.S.A.	Sprague 1950
F. oxysporum Schlecht. ex Fr.	U.S.A.	Sprague 1950
F. scirpi Lambotte & Fautr. var acuminatum (Ell. & Ev.) W.R.	U.S.A.	Anonymous 1960
Gibberella zea (Schw.) Petch.	U.S.A.	Seymour 1929
Gloeosporium bolleyi Sprague	U.S.A.	Sprague 1948
Helicobasidium purpureum Pat.	Europe	Baudyš 1929
Helminthosporium giganteum Heald & Wolfe	U.S.A.	Drechsler 1923b
Hendersonia culmicola Sacc.	U.S.A.	Sprague 1950
Hendersonia rostrupii Lind.	U.S.A.	Sprague 1950
Leptosphaeria eustomoides Sacc.	England	Webster and Hudson 1957
L. luctosa Niessl.	England	Webster and Hudson 1957
L. michotii (Westend) Sacc.	England	Webster 1955
L. microscopica Karst.	England	Webster 1955
L. nodorum Muller	U.S.A., Europe	Sprague 1950, Becker 1957
L. pontiformis (Fuckel.) Sacc.	England	Webster and Hudson 1957
Marasmus graminum (Lib.) B.	Europe	Vigorov 1961
Marasmus tritici P.A. Young	U.S.A.	Young 1925
Mycosphaerella tulasnei (Jancz.) Lindau	Alaska	Conners 1967
Nigrospora oryzae (Berk & Br.) Petch	U.S.A.	Kommedahl & Ohman 1960
Oidium monilioides L.K.	U.S.A.	Seymour 1929
Oospora nivea Dor.	U.S.S.R.	Dorokhova 1970
Ophiobolus herpotrichus (Fr.) Sacc.	U.S.A., Europe	Anonymous 1960; Webster & Hudson 1957
Papularia sphaerosperma (Pers. ex L.K.) Hoehn.	U.S.A.	Anonymous 1960
P. sphaerosperma (Pers. ex L.K.) Hoehn. var. inquinans (Dur. & Mont.) Grove	U.S.A.	Anonymous 1960

Pathogen	Locality	Reference
Phaeoseptoria festucae var. andropogonis Sprague	U.S.A.	Sprague 1962
Phleospora graminearum Sprag. & Hard.	Michigan	Sprague 1950
Phoma terrestris Hansen	U.S.A.	Sprague 1944
Phomatospora dinemasporium Webster	England	Webster 1955
Physoderma gerhardti Schroeter	artificial inoculation	Sparrow and Griffin 1964
P. maydis Miyabe.	artificial inoculation	Sparrow and Griffin 1964
P. palustris Sparrow	artificial inoculation	Sparrow and Griffin 1964
P. vagans Schroeter	artificial inoculation	Sparrow and Griffin 1964
Phytophthora species	U.S.A.	Anonymous 1960
Pleospora vagans Niessl.	England	Webster 1950
Pseudocercosporella herpotrichoides (Fron.) Deighton	Ireland	Cunningham 1967
Puccinia coronata var. rangiferina (Ito.) Cumm.	Europe	Azbukina 1956
Puccinia persistens Plowr. *	Europe	Markova and Urban 1977
Pyrenochaeta terrestris (Hans) Goreng. J.C. Walker & Larson	N. Dakota	Sprague 1950
Pyrenophora teres Drechs.	artificial inoculation	Singh 1962
Pythium aristophorum Vanterpool	U.S.A.	Middleton 1943
P. debaryanum Hesse	U.S.A.	Anonymous 1960
P. perilium Drechs.	U.S.A.	Sprague 1950
P. ultimum Trow.	U.S.A.	Anonymous 1960
Rhizoctonia solani Kuehn.	Ubiquitous	Sprague 1962
Sclerotium elymi Sprague	U.S.A.	Sprague 1950
Selenophoma donacis var. stomaticola (Bauml.) Sprague & Johnson	U.S.A.	Sprague & Johnson 1950
Septoria affinis Sacc.	U.S.A., Europe	Anonymous 1960 Palmer & Sagar 1963

Pathogen	Locality	Reference
Septoria avenae f. sp. triticea T. Johnson	U.S.A.	Sprague 1962
Septoria hispanica Babayan & Bokhyan	U.S.S.R.	Teterevnikova-Babayan & Bokhyan 1970
Septoria phyllostictoides Golovin	Denmark	Frandsen 1943
Septoria tritici Rob.& Desm.	Europe	Brokenshire 1975
Stagonospora arenaria Sacc.	U.S.A.	Sprague 1950
Stagonospora simplicor Sacc. & Berl.	U.S.A.	Sprague 1962
Stysanus medius Sacc.	U.S.S.R.	Dorokhova 1970
Tilletia brevifaciens Fischer	Europe	Della Torre 1962
Tilletia caries (DC) Tul.	Europe	Palmer & Sagar 1963
Tilletia controversa Kuhn. in Rab.	Europe	Meiners & Hardison 1957
Tilletia earlei Griff.	U.S.A.	Brenckle 1918
Typhula incarnata Fr.	Scotland	Gray 1963
Ustilago bullata Berk.	Europe	Palmer & Sagar 1963
U. calamagrostidis (Fuckel.) Clinton	Europe	Zundel 1953
Ustilago elytrigae Golovin	U.S.S.R.	Anonymous 1963
U. jamalainenil Liro	Europe	Zundel 1953
U. longissima var. dubiosa Liro	Europe	Zundel 1953
U. striiformis (Westend) Niessl.	U.S.A., Europe	Thirumalachar & Dickson 1949, Kucmierz 1977
U. trebouxi H. & P. Sydow	U.S.S.R.	Zundel 1953
Wojnowicia graminis (McAlpine) Sacc. & D. Sacc.	U.S.A., Europe	Anonymous 1960, Mäkelä 1979

Hudson and Webster (1958) have described succession of fungi on decaying stems.

Pathogen	Locality	Reference
Bacteria		
Pseudomonas angulata F. & M.	Kentucky	Anonymous 1943
Pseudomonas coronafaciens (Elliot) P.L. Stevens var. *atropurpurea* (Reddy & Godkin) Stapp.	U.S.A.	Reddy and Godkin 1923
Xanthomonas campestris pv. *cerealis* Haborg	Japan	Miyajima & Tsuboki 1980
Nematodes		
Anguina species	Europe	Molliard 1904
Ditylenchus dipsaci (Kuehn) Filip.	New York	Anonymous 1960
Ditylenchus radicicolus (Greef) Filip.	Europe	Palmer & Sagar 1963
Heterodera major Schmidt	Europe	Palmer & Sagar 1963
H. schachtii	Europe	Palmer and Sagar 1963
Meloidogyne incognita (Koford & White) Chitwood	Europe	Palmer & Sagar 1963
Paranguina agropyri Kirjanova	U.S.S.R.	Kirjanova 1955
Parasitic higher plants		
Cuscuta gronovii Willd.	U.S.A.	Anonymous 1960
Viruses		
Brome mosaic virus	Europe	Milicic *et al.* 1968
Potato virus Y	U.S.S.R.	Akhatova *et al.* 1979
Tobacco rattle virus	Scotland	Cooper & Harrison 1973
Wheat chlorotic streak mosaic virus	France	Leclant & Signoret 1975; Signoret *et al.* 1977
Wheat streak mosaic virus	U.S.A.	Staples & Brakke 1933

* *P. persistens* may be considered synonymous to *P. recondita*. (see text, page 32).

been conducted of quack grass populations in this region and therefore, many pathogens may have been overlooked. This may also apply to other areas of Canada.

The pathogenicity of several of these organisms is unclear. The damage caused by some organisms is difficult to assess, particularly those attacking roots and rhizomes. Since many reports are incidental reports and the result of isolation experiments, no pathogenicity tests were conducted. Therefore, there is no indication if these organisms were parasites or saprophytes. They were reported as pathogens, however, since they have been reported as pathogens on other crops. Dorokova (1970) reported the presence of five fungi on quack grass but no indication of pathogenicity was given.

Another factor that may add confusion is the unsettled taxonomy of several of these organisms. Many pathogens have, in the past, been known under different names. For example, 51 names have been listed as synonymous with Puccinia recondita Rob. ex Desm. (Cummins 1971). The name P. persistens Plowr. is still commonly used in Europe (Markova 1976) and Markova (1976) described the subspecies P. persistens Plowr. ssp. persistens var. persistens Urban and Markova and P. persistens Plowr. ssp. agropyrina (Eriks) Urban and Markova, both of which occur on quack grass and P. persistens Plowr. var. triticina Urban and Markova which occurs on wheat. Cummins (1971) considers the species P. persistens to be synonymous with P. recondita. However, Markova (1976) differentiated 49 taxonomic units on their ecologic, physiologic and morphologic features. The reason for the controversial taxonomy of this species is the extreme variability in morphological features in this species. Distinctive segments of the population may exist regionally and, therefore, may receive separate names (Cummins 1971). Similar situations may exist for other pathogens.

Tilletia brevifaciens G.W. Fischer has been listed here as a distinct species. Duran and Fischer (1961) consider this name to be a synonym of T. controversa Kuhn. in Rab. However, Della Torre (1962) retains the name T. brevifaciens, referring to it as a mutant of T. caries or a segment of T. caries x T. foetida (Wallr.) Liro.

Pyrenophora teres Drechs., although reported here as a pathogen, has never been found on quack grass in nature (Shipton et al. 1973). The pathogen does, however, infect quack grass with artificial inoculation (Singh 1962). In view of this, it is possible that naturally infected quack grass may eventually be found. A similar situation exists with Xanthomonas campestris pv. cerealis Haborg., Physoderma gerhardti, P. maydis, P. palustris and P. vagans.

Three organisms, Polymyxa graminis Ledingham, Ligniera pilorum Fron. and Gaillat, and Rhizophydium graminis Schenk., although minor parasites of quack grass, may be more important as virus transmitters. They have all been implicated in transmission of wheat spindle streak mosaic virus (Barr 1973, 1979).

Therefore, the lists presented here may be incomplete due to the lack of research and knowledge on plant pathogens of quack grass. The controversy surrounding the taxonomy of various pathogens and the lack of pathogenicity tests of many pathogens provide an unclear picture which can only be clarified by additional studies on taxonomic and host-pathogen relationships.

There is little indication of the distribution and prevalence of many pathogens. There is also little indication of how much damage these pathogens are causing to quack grass. This is an important factor to consider since it must be acknowledged that some of these pathogens may serve to regulate population sizes or to alter the competitive ability of the weed.

2.2 Materials and Methods

Weed and disease surveys were conducted during the summers of 1979 and 1980 to provide information on: 1) the distribution and level of infestation of quack grass and 2) the incidence and severity of diseases on quack grass, wheat, oats and barley in Prince Edward Island. One hundred and twenty-one fields in 1979 and hor fields in 1980 were randomly selected from a list of farmers, provided by the Soils and Crops branch of the Prince Edward Island Department of Agriculture and Forestry. The distribution of fields selected was determined by a

stratified random sampling procedure. The province is divided into five extension districts (Fig. 1) and the number of fields surveyed in each district was based on the proportion of land in grain in each district in relation to the total hectarage of land in grain in the province (Ivany 1980). The number of fields in each district and the number of fields of each crop surveyed are listed in Table 4. Locations of survey fields are indicated in Fig. 1.

Surveys began on June 11, 1979 and June 12, 1980 after herbicide applications and establishment of quack grass. Questionnaires were circulated to farmers to obtain information dealing with past history and management practices for each field being surveyed. A sample questionnaire is included in Appendix 1.

2.2.1. Weed surveys

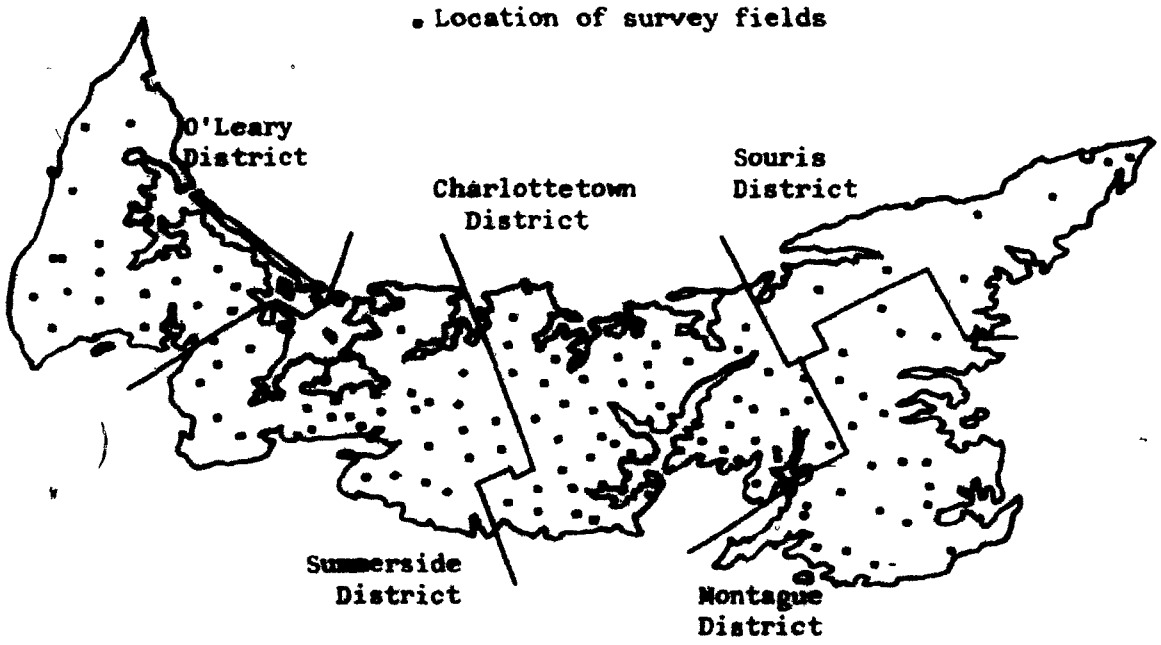
The survey procedure used was similar to that of Thomas (1976). The first sampling site in each field was selected by walking fifty paces along the edge of the field, making a 90^{o} turn, walking fifty paces into the field and selecting a site ten paces from this point at a 45^{o} angle. A 0.25 m^{2} (50 cm x 50 cm) was randomly placed at this point and the number of quack grass shoots in the quadrat was recorded. Sampling was continued as shown in Fig. 2 until twenty samples were obtained. In addition to these twenty samples, an additional ten samples were taken along the sides of each field for a total of thirty samples per field. This procedure was modified to fit small or odd-shaped fields.

2.2.2. Disease surveys

Disease ratings were recorded during the quack grass survey. Ten crop tillers and ten quack grass shoots were selected from each quadrat in the field and rated for disease. However, disease ratings were not done for plants in the quadrats along the outer edges of the fields due to possible interference of inoculum from outside the field.

Fig. 1: Extenaion districts and location of survey fields on Prince Edward Island

1979:



1980:

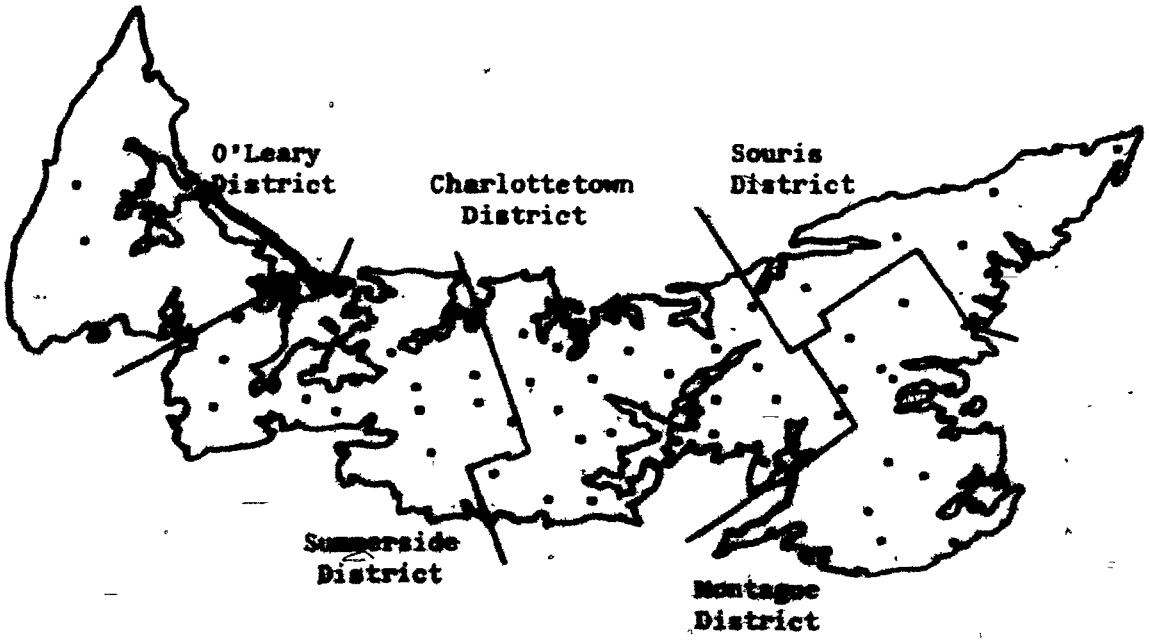
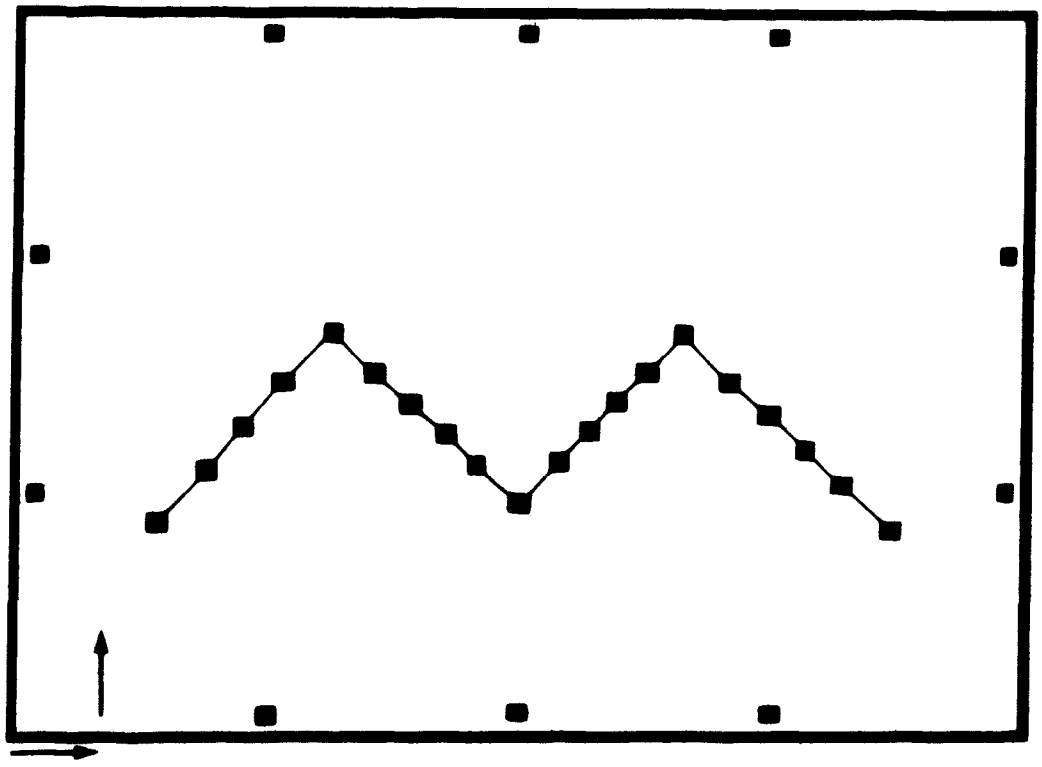


TABLE 4: Number of fields surveyed in each extension district

DISTRICT	WHEAT		OATS		BARLEY		MIXED		TOTAL	
	1979	1980	1979	1980	1979	1980	1979	1980	1979	1980
O'Leary	2	-	5	1	5	1	6	-	18	2
Summerside	6	1	4	2	10	3	7	6	27	12
Charlottetown	8	3	8	6	12	4	19	8	47	21
Montague	4	2	7	3	3	3	8	2	22	10
Souris	2	-	1	1	3	1	1	2	7	4
Total	22	6	25	13	33	12	41	18	121	50

Fig. 2 Sampling procedure for weed survey



■ Location of sampling units

The system of disease rating depended on the disease being observed. Foliar spotting diseases were rated according to the system of Horsfall and Barratt (1945). Percentage leaf area affected by disease was determined by visual observation and the rating placed in an appropriate grade. The grade number was converted to a grade formula percent (Table 5) and the mean grade formula percent was the estimated percent disease The flag leaf and second leaf of the cereal crops were assessed at growth stages 10.5.1 - 11.1(beginning of flowering to milky ripe) according to the Feekes growth stage key (Large 1954). Lower leaves were also observed for the presence of disease but were not rated. All leaves of quack grass plants were rated for disease at all growth stages of the plants. Quack grass plants along the edges of the fields were observed for disease but ratings were not recorded. Leaf samples of both the weed and the crops were collected and taken back to the laboratory for isolation to identify those diseases which could not be recognized in the field.

Diseases affecting the spike of the plants were assessed by determining the percentage of plants infected in each field. Ten tillers were selected at random from ten of the quadrats. For diseases affecting individual spikelets of the infloresence, the percentage of infected spikelets on one hundred randomly selected tillers was assessed.

Root rot of crop plants was assessed according to the method of Russell and Sallans (1940). One hundred plants were randomly selected at growth stage 11.1 (milky ripe). The roots of these plants were washed and the subcrown internode assessed for discoloration according to the following classes: slight (up to 25% discolored), moderate (25%-50% discolored) and severe (50% or more discolored). Root rot ratings were determined by the following formula: $\frac{a + 2b + 4c}{10}$ where a, b and c are the percentages of plants in the classes slight, moderate and severe. Root rot ratings were not done on roots and rhizomes of quack grass since it was rare to find root or rhizome sections without lesions. Instead, incidence of root rotting organisms was determined by isolation from the rhizomes.

TABLE 5: The Horsfall-Barratt Grading System

GRADE	% DISEASED	% HEALTHY	GRADE FORMULA %
0	0	100	1.17
1	0-3	97-100	2.34
2	3-6	94-97	4.68
3	6-12	88-94	9.37
4	12-25	75-88	18.75
5	25-50	50-75	37.50
6	50-75	25-50	62.50
7	75-88	12-25	81.25
8	88-94	6-12	90.63
9	94-97	3-6	95.31
10	97-100	0-3	97.66
11	100	0	98.82

(Horsfall and Barratt 1945)

2.2.3. Isolation from plant material

Isolations were made from diseased leaves, stems, seeds and rhizomes of plants collected in New Brunswick, Nova Scotia, Prince Edward Island and Quebec. Isolations were made immediately after plant tissues were collected or, if isolations could not be made at this time, tissue was stored in a refrigerator at 5°C for a maximum of one week. Prior to surface sterilization all tissue was rinsed in distilled water to remove soil and debris. Plant tissue was then surface sterlized by dipping it in 70% ethanol and immediately transferring it to a solution of 2% sodium hypochlorite for 5, 10 or 15 seconds for leaf tissue, stems and seeds, or for 45, 60, 90 seconds for rhizomes. The plant tissue was aseptically transferred to sterile distilled water and cut into smaller sections. End sections were discarded and the remaining sections were transferred aseptically onto potato dextrose agar plates. The petri plates were incubated at 20°C for one week. Pure cultures were obtained on potato dextrose agar slants and maintained for identification. Those organisms which could not be identified, or those for which confirmation of identity was required, were sent to the Biosystematics Research Institute, National Identification Services, Central Experimental Farm, Ottawa, Ontario. Organisms which could not be grown on artificial media were maintained on live plants or as dried spores stored at 5°C. Those organisms used for host specificity tests and subsequent experiments were maintained as agar slant cultures in a refrigerator at 5°C. Cultures were sub-cultured at three week intervals if they were not used for tests within that time period.

2.3. Results and Discussion

2.3.1. Quack grass densities in cereal crops in 1979 and 1980

This survey provided information on the current distribution and abundance of quack grass in wheat, oats, barley and mixed grain in Prince Edward Island (Table 6). The frequency values indicate that quack grass occurred at least once in 82% and 98% of the fields sur-

TABLE 6: Occurrence of quack grass in cereal crops on Prince Edward Island

	1979	1980
Frequency	82.0	98
Field Uniformity	25.9	60.3
Density (all fields)	22.6	48.9
Density (occurrence fields)	27.6	49.1
Density range	0.1 - 144.8	0.2 - 298.5

Where:

Frenquency = the percentage of fields surveyed in the province or district in which quack grass occurred.

Field Uniformity = the percentage of sampling units in the province or district in which quack grass occurred.

Density (all fields) = density (m^{-2}) of quack grass shoots averaged over all fields surveyed.

Density (occurrence fields) = density (m^{-2}) of quack grass shoots averaged over only those fields in which quack grass occurred.

Density range = minimum and maximum density recorded throughout the survey (shoots 1 m^2).

veyed in 1979 and 1980 respectively. Frequency, however, does not indicate what proportion of surveyed land was infected with quack grass or how often the weed occurred in each field (Thomas 1978a, Ivany 1980). This is indicated by field uniformity and values for 1979 and 1980 respectively indicate that approximately 25.9% of the area in cereals in 1979 and 60% of the area in cereals in 1980 were infested with quack grass.

Density was recorded as shoots m^{-2} rather than plants m^{-2}. It was difficult to identify single plants without excavation since a single plant may produce a number of shoots from its rhizome system. Tillers were not counted as separate shoots. Tillers were counted as one with the associated main shoot. The mean density of quack grass for all fields in 1979 and 1980 was 22.6 shoots m^{-2} and 48.9 shoots m^{-2} respectively. To give a better indication of the level of infestation in fields in which quack grass occurred, the mean densities of occurrence fields were calculated and found to be 27.6 shoots m^{-2} and 49.1 shoots m^{-2} for 1979 and 1980, respectively. The minimum and maximum density recorded, excluding those fields in which the weed did not occur, was 0.1 and 144.8 shoots m^{-2} for 1979 and 0.2 and 298.5 shoots m^{-2} for 1980. This wide range in density recorded for both years demonstrates the extreme variability that can occur from one field to the next. Such variability may be due to differences in crops sown, crop management practices, soil type, control applications and previous field history. The higher densities observed in 1980 were most likely a reflection of the fact that different fields were surveyed in that year.

Table 7 classifies density of quack grass according to extension district. In 1979, quack grass occurred most frequently in the Montague district (86.4%) but occurred in the largest proportion of samples in the O'Leary district (35.9%). The highest mean density was also recorded in the O'Leary district (51.1 shoots m^{-2}). The lowest mean density was recorded in the Montague district (10.2 shoots m^{-2}). This could be due to the fact that many of the fields surveyed were previously sown to tobacco. Weed control in tobacco fields

TABLE 7 : Occurrence of quack grass in small grain fields in each extension district on Prince Edward Island

	Frequency[1]		Field Uniformity		Density (all fields)		Density (occurrence fields)	
	1979	1980	1979	1980	1979	1980	1979	1980
O'Leary	83.3	100	35.9	27.5	51.1	37.4	61.4	37.4
Summerside	74.1	100	20.3	69.6	20.8	49.3	28.1	49.3
Charlottetown	80.9	100	18.0	69.5	12.0	45.3	14.8	45.3
Montague	86.4	90	23.2	54.0	10.2	59.2	11.8	60.4
Souris	85.7	100	32.1	81.0	18.9	53.2	22.1	53.2
Total Province	82.0	98	25.9	60.3	22.6	48.9	27.6	49.1

[1] see page 41 for definition of frequency, field uniformity, density (all fields), and density (occurrence fields).

was generally very good. The same did not apply to other fields in other districts which may have been sown to cereals, potatoes, hay or pasture in previous years. Quack grass may be a problem in these fields.

Frequency of quack grass in 1980 was significantly higher than in 1979 in all districts. Field uniformity and mean density were also significantly higher for all districts except O'Leary where results were significantly lower than 1979. The lower results for O'Leary are probably due to the fact that only two fields were surveyed in 1980 compared with 18 fields in that district in 1979. The highest mean density was recorded in the Montague district (59.2 shoots m^{-2}) and the lowest density (37.4 shoots m^{-2}) was recorded in the O'Leary district. The highest proportion of land infested with quack grass was in the Summerside district but similar results were recorded for the Charlottetown district. The lowest proportion of land infested was in the O'Leary district. However, this may not be truly indicative of quack grass infestation in this district as only two fields were surveyed.

Table 8 classifies density of quack grass according to the individual crops surveyed. In 1979, quack grass densities, frequency and field uniformity were highest in oats. The lowest mean density, frequency and field uniformity were recorded in wheat. In 1980, quack grass frequency was 100% for wheat, oats and mixed grain and 92.3% for barley. There was no significant difference in field uniformity for wheat, oats and mixed grain but field uniformity for barley was significantly less than for the other crops. Density was also lowest in barley. Cussans (1968) reported lower densities of quack grass in barley than in wheat, field beans and rape seed. He suggested that barley was a much more effective competitor with quack grass than the other crops.

Mean density of quack grass in wheat in 1980 was 94.6 shoots m^{-2}. Two fields in the Montague district, with densities of 298.5 and 272.3 shoots m^{-2} probably account for this high density. However, even if the densities of these two fields are left out of the calculation, the mean density of wheat is still higher than the mean densities of the other crops.

TABLE 8 : Occurrence of quack grass in barley, oats, wheat and mixed grain fields on Prince Edward Island

	Frequency[1]		Field Uniformity		Density (all fields)		Density (occurrence fields)	
	1979	1980	1979	1980	1979	1980	1979	1980
Barley	80.4	92.3	22.8	49.5	18.8	20.2	20.5	21.1
Oats	88.6	100	31.3	61.0	31.8	38.8	36.4	38.8
Wheat	74.2	100	20.7	62.9	14.4	94.6	22.9	94.6
Mixed	84.8	100	28.9	67.9	25.4	41.9	30.5	41.9

[1] see page 41 for definition of frequency, field uniformity, density (all fields), and density (occurrence fields).

The significant difference in results between 1979 and 1980 may be due to the fact that less than one half the number of fields were surveyed in 1980 as were surveyed in 1979. Also, the same fields could not be surveyed the second year due to the rotation system and other farm management decisions on many farms. The crop management practices the previous year (for example fall ploughing or fall applied herbicides) could have a significant effect on the succeeding year's population and result in significantly altered densities. Also, winter and spring weather conditions could also influence densities.

The densities recorded in all districts in 1979 and 1980 would be classified as a severe infestation by Cussans (1980). Cussans (1980) defined a severe infestation as one in which densities are in the range of 10 to 100 shoots m^{-2}. In such an infestation yield loss is usually, but not always, greater than cost of control. With densities higher than 100 shoots m^{-2}, yield lost is always higher than cost of control.

Pearson product-moment correlation (Steel and Torrie 1980) was used to determine if there was any correlation between the density of quack grass and the total area in cereals in each district. The total area in each district under cultivation to small grains in 1976 is presented in Table 9. Correlation coefficients of 0.2395 and -0.2679 for 1979 and 1980 respectively indicate that there was no strong correlation between the total area in cereals and quack grass density. Actually, in 1980, there was a tendency for high densities in the districts with less grain. Therefore, there was no tendency for quack grass to occur more in the districts with a larger proportion of land in cereals.

Pearson product-moment correlation was also used to determine if there was any correlation between quack grass density and crop and also between quack grass density and the area under cultivation to each individual crop in each district. Correlation coefficients of -0.1442 and 0.2027 for 1979 and 1980 respectively indicated no strong correlation existed between density and crop. A much stronger correlation was apparent in 1979 between quack grass density and the area sown to each individual crop in each district (correlation coefficient 0.4212). Therefore, in 1979, there appeared to be a tendency for quack grass to occur in those crops which were grown on the greater proportion of land. This correlation was not apparent in 1980 (correlation coefficient -0.0887).

TABLE 9: Area of land in each extension district in cereal production on Prince Edward Island in 1976

District	Wheat	Oats	Barley	Mixed	Total
O'Leary	497	3 472	3 532	3 841	11 342
Summerside	1 610	4 711	4 444	9 068	19 833
Charlottetown	1 966	4 692	2 042	14 350	23 050
Montague	1 570	2 785	2 224	3 012	9 591
Souris	859	1 679	728	3 390	6 656
Total	6 502	17 339	12 970	33 661	70 472

(Statistics Canada 1978)

2.3.2. Disease incidence on quack grass - 1979 and 1980

A total of 30 pathogens were recorded on quack grass in the areas surveyed, including Quebec (Table 10). Three of these, Drechslera biseptata (Sacc. & Roum.) Richardson & Fraser, Pyrenophora bromi (Died) Drechsl. and P. japonica Ito & Kurib are pathogens that have not been reported previously on quack grass. Fourteen of these pathogens or varieties or races of them have been reported elsewhere as pathogens of economic crops.

A total of 24 pathogens were found on quack grass in the Maritime Provinces, twenty of these being previously unreported on quack grass in this area. This increase in the number of pathogens reported and the common occurrence of pathogens identified in this survey in other areas of Canada is most likely indicative of the lack of survey work previously conducted on quack grass.

2.3.2.1. Pathogens occurring on leaves and stems

Powdery mildew was the most prevalent leaf disease on quack grass in the Maritime Provinces in 1979 and 1980. Signs of the causal organism, Erysiphe graminis, consisting of a white powdery fungal growth on the upper and lower surface of the leaves, were evident in late May and persisted throughout the season. The disease occurred on quack grass in all areas surveyed in both cultivated fields and undisturbed areas. This disease appeared to be the most damaging disease occurring on quack grass in the Maritime Provinces. This disease also occurred in Quebec, but its occurrence was not as widespread as in the Maritime Provinces, especially in cultivated fields.

Crown rust was prevalent on quack grass throughout eastern Canada. In Quebec, reddish-orange uredia of the causal organism, Puccinia coronata, could be found in early May on newly emerged shoots. The presence of the uredial stage on quack grass so early in the season, indicates that the rust may overwinter on quack grass, possibly as

TABLE 10: Pathogens identified on quack grass in Eastern Canada[1]

Pathogen	Disease	Location[2]	Specificity[3]
Barley yellow dwarf virus (BYDV)	Red leaf	P.E.I.	2
Cochliobolus sativus (Ito & Kur.) Drechs.	Root rot & spot blotch	E. Canada	2
Cladosporium sp.	Leaf mold	Que.	2
Claviceps purpurea (Fr.) Tul.	Ergot	E. Canada	2
Colletotrichum sp.	Anthracnose	P.E.I.	?
Drechslera biseptata (Sacc. & Roum.) Richardson & Fraser	Root rot	E. Canada	2
Erysiphe graminis D.C. exMerat	Powdery mildew	E. Canada	2
Fusarium acuminatum Ell. & Ev.	Head blight	Que.	3
F. avenaceum (Fr.) Sacc.	Root rot & Head blight	E. Canada	3
F. culmorum (W.G.Sm.) Sacc.	Root rot & Head blight	E. Canada	3
F. equiseti (Cda.) Sacc.	Root rot & Head blight	E. Canada	3
F. graminearum Schw.	Root rot & Head blight	E. Canada	3
F. oxysporum Schlecht.	Root and rhizome discoloration	E. Canada	3
F. poae (Pk.) Wr.	Head blight, Silvertop	N.S. and P.E.I.	2
F. sporotrichoides Sherb.	Root rot & Head blight	E. Canada	3
Gaeumannomyces graminis (Sacc.) Arx & Oliver	take all	N.S. and P.E.I.	2
Gliocladium roseum (Lk.) Bainier	Root necrosis	P.E.I.	2
Passalora graminis (Fckl.) Hohn.	Brown stripe	Que.	2
Phyllachora graminis (Pers.ex Fr.) Fckl.	Tar spot	E. Canada	2
Puccinia coronata Cda.	Crown rust	E. Canada	2

Pathogen	Disease	Location	Specificity
P. graminis pers.	Stem rust	E. Canada	2
P. recondita Rob.ex Desm. var. agropyri	Leaf rust	E. Canada	2
Pyrenophora japonica Ito & Kuribi	Leaf spot	P.E.I.	2
P. bromi (Died.) Drechsl.	Leaf blotch	Que.	2
Pythium sp.	Seedling blight and root rot	E. Canada	3
Rhizoctonia solani Kuhn	Root rot	P.E.I.	3
Rhynchosporium secalis (Oud.) J.J. Davis	Scald	P.E.I	2
Septoria nodorum Berk.	Leaf blotch and glume blotch	P.E.I.	2
Septoria sp.	Leaf blotch	Que.	?
Urocystis agropyri (Preuss) Schroet.	flag smut	Que.	2

[1]Eastern Canada refers to New Brunswick, Nova Scotia, Prince Edward Island and Quebec. Newfoundland was excluded due to travel constraints during surveys.

[2]Location - the pathogen was isolated or identified at least once in the provinces mentioned.

[3]Specificity refers to the specificity reported in the literature.

1 - specific to quack grass

2 - restricted to Poaceae

3 - not restricted to Poaceae

? - pathogen unidentified, specificity unknown

resting mycelium in infected tissue or as uredospores Mehta (1923) has reported successful overwintering of uredospores of P recondita and P striiformis Green (1963) reported that P graminis f sp secalis Erikss & Henn could overwinter on quack grass in Manitoba Butler and Jones (1961) reported that resting mycelium of P striiformis is capable of surviving unfavorable climatic conditions in infected tissue that uredospores could not tolerate. The presence of these uredospores early in the season could provide a source of primary inoculum from which a more extensive and widespread development of the disease may occur

Maximum development of this disease occurred late in July in Quebec and appeared to cause significant damage for the remainder of the season In the Maritime provinces, the disease did not appear until early August and disease development was not as extensive as in Quebec As a result, damage to quack grass did not appear to be as significant as in Quebec

Two other rust organisms, P graminis and P recondita var agropyri occurred on quack grass, but did not appear until late July-early August These rusts were found on quack grass not protected by a crop canopy and did not occur on quack grass found in cereal fields P graminis infections were rarely found on the leaves of the plant but were restricted to the leaf sheaths, stems and spikes In areas surveyed in Quebec, stem rust development was extensive on quack grass by mid-August and appeared to be causing extensive damage, particularly in communities dominated by quack grass. In the Maritime provinces, extensive stem rust development was found only in the Annapolis Valley region of Nova Scotia and, although it occurred in other areas, it appeared to cause little damage other than in the Annapolis Valley region

P recondita occurred at a much lower incidence than P graminis or P coronata and disease intensity was also much less. The levels of disease were higher in those areas and on those plants where P coronata did not occur. It is possible that P coronata, when present at high levels on leaves, may be effectively excluding P

recondita from the leaves by reducing the number of infection sites available Therefore leaf rust was not as damaging as either crown rust or stem rust

One other leaf disease found in all provinces surveyed was tar spot incited by Phyllachora graminis Symptoms appeared as spindle-shaped, black, shiny lesions on the leaf surface The disease was common throughout Prince Edward Island in 1979 but was only recorded occasionally in 1980 The disease was first noticed in Quebec in 1981 on quack grass collected on the Macdonald Campus of McGill University This disease was never found on quack grass in cultivated fields but was common along the borders and headlands of many fields and in undisturbed areas The disease was also never found on cultivated cereal crops although it had been noted on other wild grasses

Leaf scald, incited by Rhynchosporium secalis, was common on quack grass throughout Prince Edward Island and occurred occasionally in areas surveyed in Nova Scotia Early symptoms were characterized by dark bluish-gray colored lesions with a water-soaked appearance These lesions later appeared a light grey to ivory color The margins of the lesions assumed a dark-brown color Lesions generally measured 1-2 cm x 0.5-1 cm although lesions often coalesced. Occurrence of the disease was restricted to the margins of fields and undisturbed areas and was rarely found in cultivated fields It did not occur on quack grass found in barley which was heavily infected with the disease This indicates a possible host specialization of this organism. The disease appeared on quack grass in early-to mid-June in both years in which the survey was conducted.

Spot blotch, incited by Bipolaris sorokiniana, was often found infecting quack grass leaves in cereal fields in Prince Edward Island, particularly as the crop matured and after harvest. Symptoms appeared as small, oval to circular brown lesions which may or may not have been surrounded by a chlorotic margin. The pathogen was never isolated from plants in areas away from a cereal field and only occasionally from along field margins. The pathogen was also a serious pathogen of cereals and other grasses, causing, in addition to spot blotch, root rot and seedling blight. The increase in the incidence of the pathogen on quack grass

late in the season, indicates that the inoculum source may be from the cereal crop.

Septoria nodorum, the causal organism of septoria leaf blotch and glume blotch, was often found on quack grass associated with wheat on which the disease was a problem Spores of the pathogen are dispersed by splashing rain (Scharen 1963) and spores can be easily splashed onto quack grass growing within an infected wheat crop Isolated lesions appeared as necrotic diamond-shaped lesions, but often the lesions coalesced and large areas of necrotic tissue occurred, particularly along the tip of the leaves The disease was generally restricted to lower leaves, although upper leaves occasionally showed isolated lesions

Pyrenophora japonica was isolated on only two occasions from plants collected in Prince Edward Island Symptoms resembled those of Cochliobolus sativus on leaves of quack grass This is the first time this pathogen has been recorded on quack grass and also the first time the pathogen has been reported in eastern Canada. The taxonomy of this organism is unclear. Kenneth (1962) considers this name to be a synonym of P. teres. However, Shoemaker (1962) accepts both as valid species.

P japonica was tentatively identified on barley Symptoms consisted of dark elliptical lesions surrounded by a chlorotic zone, similar to leaf symptoms caused by C. sativus. Similar symptoms were observed by Clough and Sanderson (1979) and were attributed to P. teres. However, confirmation of the existence of P. japonica on barley may explain the "atypical symptoms" attributed to P. teres.

Barley yellow dwarf was noted only once on quack grass in 1979. Leaves of infected plants appeared red in color. The plants were found adjacent to a field of mixed grain which was heavily infected by this disease in Elmira, Prince Edward Island.

Four pathogens occurring on leaves of quack grass were found only in the areas surveyed in Quebec. A Cladosporium species was found on leaves of quack grass in April, 1980. Signs of the fungus were present as a dark green-brown fungal growth on necrotic tissue at the leaf tips. It is possible that this organism was colonizing tissue that had been

damaged by frost and weakly parasitizing adjacent healthy tissue.

A species of Septoria was isolated from leaves of quack grass collected at the Emile A. Lods Research Center of Macdonald Campus of McGill University in 1981. There was extensive necrosis on infected plants and symptoms resembled those of other septoria diseases on grasses. Exact identity of the organism could not be obtained because of bacterial contamination of the original spore material.

A pathogen thought to be Passalora graminis was identified on quack grass collected in research plots at the Emile A. Lods Research Center in 1981 and again in 1982. Symptoms of the disease appeared as a brown stripe originating at the base of the leaf where the blade meets the leaf sheath. The disease was not common and did not appear to progress further than the second or third leaf. The disease, also, did not appear later in the season and caused no significant damage.

Flag smut, incited by Urocystis agropyri, was a disease commonly found on quack grass in the area surrounding the Macdonald Campus of McGill University. Diseased plants usually occurred in patches since the organism most likely infects through the rhizomes. Symptoms appeared as elongated grayish or dull white stripes which were slightly raised above the surface of the leaf. These sori, which were usually interveinal, remained unbroken for several weeks. Eventually the epidermis ruptured and exposed the black mass of spores within. Secondary shoots and tillers arising within 10 to 15 cm of an infected plant were also infected. These infected secondary shoots may have arisen from hyphae already inside the tissues of the host plant (Griffiths 1924). Spikes were rarely produced and, when present, were generally sterile or had few mature seeds. Often the glumes of infected plants also showed signs of the organism.

The damage caused by this pathogen did not appear to significant. Infected plants persisted throughout the summer and survived the winter as mycelium in infected rhizomes. Patches were not large and there appeared to be no extensive areas of damage. Infected plants appeared to withstand the effects of regular mowing when they occurred in lawns. No information is available on the effect of the organism on rhizome growth, but

preliminary results of greenhouse studies indicate that rhizome growth of infected plants was severely restricted after eight months. There was rhizome growth during the initial stages of disease development which continued for six to eight weeks, until the appearance of symptoms. This initial rhizome growth eventually decayed, and, instead of new rhizome growth, there was an increase in the number of shoots originating at the crown. Therefore, it appears that this organism has the potential to restrict vegetative reproduction of quack grass, which is this species' most important method of reproduction.

2.3.2.2. Pathogens occurring on spikes and seed

Three diseases were recorded on the spikes. Flag smut, described earlier, occurred only in Quebec. The other two diseases, ergot and fusarium head blight, were found extensively throughout eastern Canada.

Ergot, incited by Claviceps purpurea, was found on 93% of all spikes examined. Signs of the organism consisted of hardened black sclerotia which replaced the seed in individual florets. Sclerotia of C. purpurea were commonly seen emerging from infected florets. However, many additional sclerotia were found following dissection of the florets. The number of sclerotia per floret varied with the maximum number recorded from a single floret being four. The number of infected spikelets per spike also varied with a maximum of 21 sclerotia found in 26 spikelets on one spike. The main damage caused by this organism was a reduction in the number of seed produced. Losses were estimated at 12-15% of total seed produced.

Fusarium head blight occurred in two forms. The first form, which was the most damaging, was blighting of the whole spike. Infection apparently occurred at the first node or just above it. The entire spike above this point appeared bleached in contrast to the healthy plant parts below. Spikes were found to be sterile. Stems were often broken and fungal growth could be seen at the initial point of infection. Seven Fusarium species were isolated from the blighted spikes but the one most consistently found was F. poae. However,

F. avenaceum was also often isolated from the stems. This form of the disease was seen only in Prince Edward Island and Nova Scotia. The symptoms of the disease were similar to a disease called silver top, described by Berkenkamp and Meeres (1975) and both diseases, in this case are considered to be the same. Berkenkamp and Meeres (1975) attributed the disease to F. poae, but they also stated that mites or insects may also be involved. It is possible that a wound from an insect could serve as an infection court for a pathogen such as F. poae.

The second form of fusarium head blight that was found was blighting of individual spikelets. Only one or two spikelets on each spike were affected and no seed were produced in these spikelets. Infected spikelets appeared whiter than noninfected ones and often an orange or pink fungal growth could be seen on infected spikelets. The disease is similar to fusarium head blight occurring on cereal crops. The disease was found in all areas surveyed and again different Fusarium species were also isolated from infected spikelets.

The damage caused by diseases reducing seed production is not considered to be significant in regulating population numbers, since seed may not be considered important in persistence of the weed (Williams and Attwood 1971). Quack grass may, however, be more important in serving as a source of inoculum for these diseases on economic crops.

2.3.2.3. Pathogens isolated from rhizomes

The pathogenicity of several organisms isolated from the rhizomes was difficult to assess. The fungal flora of the rhizomes was quite varied and in most cases two or more fungi were isolated from each rhizome piece. Diseased rhizomes are often very quickly colonized by secondary invaders and it is difficult to isolate the actual pathogen (Kommedahl and Ohman 1966). Another difficulty is that rhizomes of uninoculated control plants also developed disease symptoms or several organisms that were isolated from diseased rhizomes were also isolated from apparently healthy tissue. Therefore,

the pathogenicity of some of these fungi could not be established and it is suggested that saprophytic strains of these organisms may exist that are part of the natural saprophytic flora of the rhizomes. Fungi whose pathogenicity is uncertain are Fusarium acuminatum, F. equiseti, F. oxysporum, F. sporotrichoides, Gliocladium roseum and Pythium species. Pathogenic strains of these oganisms, however, may also exist.

The pathogenicity of Bipolaris sorokiniana and Drechslera biseptata were easily confirmed as these pathogens also cause a leaf spot. The most notable symptom of take-all, incited by Gaeumannomyces graminis, is a blackening of the rhizome and stem base, and the presence of this symptom was used as confirmation of the pathogenicity of this organism. No other organism reported on quack grass causes a similar symptom. Colletotrichum causes a characteristic sunken lesion on the rhizome from which the fungus will sporulate profusely and its identity is easily confirmed. F. avenaceum, F. culmorum and F. graminearum resulted in a seedling blight when soil was infested with these organisms and reisolation confirmed their pathogenicity. Rhizoctonia solani caused a browning of the rhizome and, in some cases, characteristic oval, ivory colored eyespot lesions on the base of the stem. Reisolation was also used to confirm the pathogenicity of this organism.

These organisms have the potential to cause serious damage to quack grass. However, the significance of their effect under field conditions is unknown. Germination studies indicated that diseased rhizomes collected from the field showed percent germinations of 90-100% and little shoot death was noticed in the field. More intensive studies of quack grass populations under field conditions are required to determine the effect of pathogens on rhizome growth.

2.3.2.4. Other fungi isolated from plant parts

Several saprophytic fungi were also isolated from quack grass. Some of these fungi are listed in Table 11. Species from two of these genera have been previously reported as pathogens on quack grass (Alternaria and Acremonium (Cephalosporium)). However, organisms listed in Table 11 were not associated with any symptoms of

TABLE 11: Non-pathogenic fungi isolated from quack grass

Species	Plant Part
Acremonium (Cephalosporium) sp.	rhizomes
Alternaria sp.	rhizomes, stem, leaves, seeds
Aspergillus sp.	rhizomes
Epicoccum purpurascens Erenb. ex Schlect.	leaves and seed
Microdochium bolleyi (Sprague) de Hoog and Hermanides - Nijhof	rhizomes
Penicillium sp.	rhizomes
Trichoderma hamatum (Bon.) Bain aggr.	rhizomes
Trichoderma viride Pers. ex Fr.	rhizomes

disease on the plant.

Six of the fungi listed in Table 11 have been reported to be antagonistic to other fungi. Species of Trichoderma have been reported to be antagonistic to Cochliobolus sativus (Prasad et al. 1978) and Aspergillus species, Trichoderma species and Epicoccum purpurascens have been shown to be antagonistic and possibly parasitic on C. sativus, Fusarium culmorum and Rhizoctonis solani (Wu 1977), all of which are important cereal pathogens. Microdochium bolleyi has been linked to a possible antagonistic effect on Fusarium species. Reinecke (1978) found that there was a negative correlation between the presence of M. bolleyi and the number of wheat plants infected with Fusarium species. Acremonium boreale Smith and Davidson may play a significant role in determining the nature and intensity of damage in snow mold complexes of cereals and grasses (Smith and Davidson 1979). Therefore, in addition to the possibility of quack grass carrying potential pathogens of economic crops, it may also carry a complement of antagonistic fungi which may limit pathogen development on this plant and thereby reduce its importance as a source of inoculum. The importance or extent of this phenomenon, however, is unknown.

2.3.3. Disease occurrence on cereal crops - 1979 and 1980

Disease surveys of cereal crops were conducted to establish the type and severity of cereal diseases on Prince Edward Island in 1979 and 1980. Cereal grain production constitutes an important part of Prince Edward Island agriculture. Cereal disease is one factor which can greatly affect crop quality and yield. The type and severity of these diseases may vary from one year to the next.

The summer weather pattern may explain, to some extent, the disease situation which occurred each year. Weather summaries were obtained from the Technical Services Branch of the Prince Edward Island Department of Agriculture and Forestry. Above normal rainfall during the latter part of May 1979 resulted in a delay of spring sowing to late May and early-to-mid June. Above normal temperatures for May and June and below normal rainfall in June resulted in rapid growth

and development of early sown fields. As a result of dry conditions, disease levels remained low in these fields. Record high rainfall in July and August, and above average air temperatures in July and early August, encouraged high disease levels to develop in some crops, particularly in late sown fields.

In 1980, dry conditions allowed many farmers to complete spring planting operations by the end of May. Cool, dry conditions during May and early June resulted in delayed germination and slow crop development. Disease development was delayed also, however. Above normal rainfall in late June and July resulted in rapid development of many diseases which was intensified by high rainfall in early August. Conditions were very good for harvesting cereals and, as a result, over 40% of the cereal crop was harvested by the end of August (Anonymous, 1980). Crops harvested late were seriously damaged by fusarium head blight however, which was promoted by well above normal rainfall.

2.3.3.1. Diseases occurring on wheat

Winter wheat does not constitute an important part of wheat hectarages grown on Prince Edward Island. Winter kill was severe in 1979 and as a result, a large hectarage had to be plowed down, with remaining fields showing 30-60% winter mortality. The most widespread diseases occurring on winter wheat in 1979 were Septoria leaf blotch (incited by Septoria nodorum), powdery mildew (incited by Erysiphe graminis f. sp. tritici), and fusarium head blight (incited by various Fusarium species) (Table 12). Leaf disease ratings ranged from two to ten percent of the flag leaf diseased and from three to ten percent of the second leaf diseased. Yield losses from foliar pathogens were not considered to be significant. Fusarium head blight occurred in all fields surveyed but serious losses occurred only in one field (13% spikelets destroyed). Low levels of take-all, incited by Gaeumannomyces graminis, were present in most fields surveyed, but represented a yield loss of only one to three percent. Glume blotch, incited by S. nodorum, also occurred but disease levels were generally low.

TABLE 12: Mean disease ratings for diseases occurring on wheat

Disease	Mean	Standard error	Minimum-Maximum
Powdery mildew[1]			
1979	1.96	0.4	1.2 - 10.7
1980	1.3	.1	1.2 - 3.0
Septoria leaf blotch[1]			
1979	11.7	4.9	1.2 - 94.5
1980	21.7	4.6	1.2 - 43.9
Fusarium head blight[2]			
1979	22.2	2.9	0 - 51.0
1980	14.4	6.2	0 - 63.0
Take-all[3]			
1979	0.2	0.1	0 - 3.0
1980	12.1	2.2	1 - 24.0
Smut[3]			
1979	2.0	0.9	0 - 20.0
1980	7.5	2.1	0 - 24.0

1 - % leaf area diseased

2 - % spikelets diseased

3 - % population diseased

Only three farmers were known to have grown winter wheat in 1980. Results of disease surveys of these fields are not presented due to the severe winter kill which occurred in this region.

Fusarium head blight caused the most serious losses in spring wheat in 1979 and 1980. This disease has been increasing in importance on Prince Edward Island in recent years (Clough and Johnston 1978b). Warm temperatures and high rainfall in late July provided very favorable conditions for disease development. In 1979, the mean percentage of spikes infected was 36.2%. Approximately 22.2% of the spikelets were affected. Maximum disease level recorded was 70% of the spikes and 51% of the spikelets affected.

The high levels of disease present in 1979 provided high levels of inoculum for 1980. Disease ratings ranged between 12% and 41% of the plant population affected and between 22% and 63% of the spikelets affected. Fields which were harvested late were the most severely damaged. Fusarium head blight can cause serious damage as long as the crop remains standing in the field, and, therefore, the longer the crop is exposed to pathogen inoculum, the greater is the potential for serious yield loss. This disease was also responsible for reduced germination of seed used in 1980 and 1981 and a similar situation was expected for certified seed stocks for 1982 (Stirling, personal communication). In addition to these losses, there is also a danger from mycotoxins produced by various Fusarium species, which may have serious consequences for livestock which consume contaminated grain.

Powdery mildew was more severe on wheat in 1979 than in 1980. Mean disease ratings were 1.6% of the flag leaf diseased and 2.4% of the second leaf diseased in 1979. In 1980 powdery mildew was found in only 50% of the fields surveyed. Disease levels in both years were not considered to be great enough to cause serious yield loss. This may have been due to the extremely high rainfall in July. High disease levels may be favoured by low moisture levels (Cherewick 1944). Disease severity is also influenced by high nitrogen fertility (Clough and Johnston 1978a), nearness to other wheat fields (Johnston 1974), and the use of fungicides. These factors may explain variation in disease levels found in one field to the next and from one season to the next.

Septoria leaf blotch was the foliar pathogen occurring at the highest levels in 1979 and 1980. The disease occurred at low levels during the first part of the season in both years and only trace amounts of the disease were found. Frequent rains in July and August provided ideal conditions for splash dispersal of conidia, and by early August disease levels were high in all fields surveyed. There was a strong positive correlation in 1979 and 1980 between septoria ratings and seeding rate (r_s = 0.5441 in 1979 and r_s = 0.7374 in 1980, both significant at α = 0.0005). This was to be expected since high density and close association of plants would make splash dispersal of conidia very effective. Higher planting density would also make the canopy microclimate more favorable for disease development. No correlation was found between disease ratings and date of seeding. There was also no correlation between disease ratings and successive years in grain in 1979. In 1980 however, there was a positive correlation between septoria leaf ratings and successive years in wheat (r_s = 0.5642, significant at α = 0.0005). Mean septoria rating was 11.7% in 1979 and 21.7% in 1980. Although the 1980 mean was higher than that of 1979, there was less variation in disease ratings.

Loose smut, incited by Ustilago tritici, caused some losses, particularly in Opal and Vernon wheat. In both years, losses were highest in fields where untreated seed was used. Losses in these fields ranged from 6% to 20% in 1979 and 8% to 24% in 1980. Losses of 12% to 15% however, were recorded in fields in which fungicide-treated seed was used in 1980.

Common root rot, incited by Bipolaris sorokiniana and various Fusarium species, occurred in all fields surveyed, although disease levels varied greatly, ranging between 7% to 30% in 1980. Mean root rot rating was 18%. Take-all, also a root rot, was a problem only in isolated fields. In 1979, maximum disease rating was 14% and the mean was 3.0% of the plants infected. In 1980, the minimum and maximum disease ratings were 1.0% and 24% respectively, with a mean of 12.2%.

Other diseases which occurred on wheat but which had a low incidence included spot blotch (incited by B sorokiniana), sclerophoma

leaf spot (incited by Selenophoma donacis), leaf rust (incited by Puccinia recondita f. sp. tritici), barley yellow dwarf (incited by barley yellow dwarf virus (BYDV)), and ergot (incited by Claviceps purpurea)

2 3.3.2 Disease occurrence on rye

On Prince Edward Island, rye is grown primarily as a rotation crop, prior to tobacco. Therefore, yield loss due to disease on this crop is not as important as for other cereals. The most common diseases occurring on rye, in 1979, were leaf scald (incited by Rhynchosporium secalis and take-all (Table 13). The mean scald rating was 10% Maximum disease rating for any field was 45.14%. Mean take-all rating was 4% of the population in the field affected. Maximum percent disease recorded was 21%. With both diseases, there was a positive correlation between successive years in rye and mean disease ratings For scald, r_s = 0.7218 and was significant at α = 0.002. For take-all, r_s = 0.8539 and was significant at α = 0.002. This indicates that both diseases were more severe in those fields in which rye was grown for two or more years.

Ergot was common in most fields surveyed but was frequently restricted to those plants occurring along the edges of the field. Fusarium head blight appeared late in the season, as weather conditions at this time were favorable for disease development. Septoria leaf blotch, spot blotch, common root rot, and powdery mildew were also present.

Rye was not included in surveys in 1980.

2.3.3.3. Disease occurrence on oats

The most prevalent disease occurring on oats in 1979 and 1980 was septoria leaf blotch incited by Septoria avenae (Table 14). This disease was present at low levels in all fields during the first half of the season. By late July to early August, however, the disease occurred at high levels in most fields.

TABLE 3 Mean disease rating for diseases surveyed on rye 1979

Disease	Mean	Standard error	Minimum	Maximum
Scald	[illegible]	[illegible]	[illegible]	[illegible]
Take-all	4		[illegible]	0

1 - Rye was surveyed only in 1979

- Percent leaf area diseased

3 - % population diseased

TABLE 4 Mean disease ratings for diseases [illegible]

Disease	Mean	Standard error	Minimum	Maximum
Septoria leaf blotch				
1979	18.6	4	-	89.4
1980	10.8	3.1	-	5[illegible]

[illegible] - Percent leaf area diseased

The mean disease rating in 1979 was 6.5%. The minimum and maximum values were 2% and 80.4% respectively. In 1980 the mean was 8%. The minimum and maximum values were 1% and 37% respectively. Therefore, disease level appeared to be lower in 1980. The late sowing in 1979 may have been responsible for this. Septoria leaf blotch is present on oats every year and although disease levels may vary significantly from one field to the next, overall disease levels from one year to the next do not vary significantly (Clough and Johnston 1978a). Most cultivars of oats are susceptible to this disease (Clark and Zillinsky 1960). Overall yield loss attributed to this disease on Prince Edward Island has been considered to be substantial in previous years (Clough and Johnston 1978a), and may be as high as 50%. A positive correlation was found between seeding date and disease rating (r_s = 0.4425, significant at α = 0.03) in 1979. This suggests that the disease was more severe in later sown fields. No such correlation was found in 1980. No correlation was found between disease ratings and successive years in oats. Only three of the fields surveyed in 1979 and two fields in 1980 had been sown to oats the previous year, however. Disease ratings in these fields tended to be higher than was found in those fields not sown to oats the previous year. This was to be expected as the causal organism survives from one year to the next on crop debris.

Drechslera leaf spot, incited by Drechslera avenacea, occurred at low levels in most fields in 1979 and 1980 but actual levels of the disease were not ascertained. Symptoms of this disease may be easily confused with those of septoria leaf blotch. Therefore both of these diseases were assessed together. Isolations to confirm the identity of the causal organisms were conducted in only a few cases. This pathogen has been reported to occur at high levels on oat seed in the Maritime provinces (Clough and Johnston 1978b), but the importance of this disease on oats is unknown.

Red leaf of oats, incited by BYDV, was found in many fields but was more serious in those fields which had been sown late. This disease is transmitted by aphids and has the potential to be very damaging if it occurs early in the season. In 1979 and 1980 however, aphids were

first detected late in the season which probably accounted for the low incidence of this disease.

Other diseases which were also recorded in 1979 and 1980 at low levels on oats were grey speck (a manganese deficiency), covered smut (incited by Ustilago kolleri Wille), crown rust (incited by Puccinia coronata f. sp. avenae), powdery mildew, and fusarium head blight.

3.4 Disease occurrence on barley

The most widespread disease found on barley, and probably the most damaging, was common root rot, incited by Cochliobolus sativus, Fusarium species, and possibly a number of other root infecting pathogens (Table 15). This disease was found in all fields surveyed in 1979 and 1980 and tended to be more severe in fields in which grain was grown for two or more years. There was a positive correlation found in 1979 between the number of successive years in grain and percent root rot. No such correlation was found in 1980. The mean root rot percent found in 1979 was 32.1% with a minimum and maximum rating of 14% and 59% respectively. The mean percent root rot rating in 1980 was 24.8% with a minimum and maximum value of 8.0% and 42.0% respectively. The decrease in root rot noted in 1980 may have been due to the early sowing and cool, dry conditions of late May and early June in 1980.

Spot blotch (incited by C. sativus), and net blotch (incited by Pyrenophora teres) were the most prevalent leaf diseases occurring on barley in 1979 and 1980. Net blotch was found in all fields surveyed. Spot blotch was recorded in most fields surveyed, but only later in the season. Net blotch ratings remained low for June and most of July in both years. However, there was a dramatic increase in net blotch ratings following a period of wet weather in late July. As a result, disease ratings varied extensively over the season. In 1979, mean ratings ranged between 1.2% and 97.4% with a mean of 8.7%. Ratings ranged between 1.2% and 48.1% with a mean of 6.8% in 1980. Pyrenophora teres was consistently isolated from diseased tissue for most of the season. Isolation of C. sativus became more common as the crop matured. C. sativus was often isolated from lesions similar to those associated

TABLE 15 Mean disease ratings for diseases occurring in barley

Disease	Mean	Standard error	Minimum		Maximum
Net blotch[1]					
1979	9	[illegible]	[illegible]	-	97.4
1980	6.8	[illegible]	[illegible]	-	48.1
Scald[1]					
1979	[illegible]	[illegible]	[illegible]	-	[illegible]
1980	3.4	1.6	1.2	-	[illegible]
Leaf blotch[1]					
1979	trace	-		-	
1980	2.5	0.9	1.2	-	21.4
Root rot[2]					
1979	32.1	2.3	14.0	-	59.0
1980	24.8	2.1	8.0	-	42.0
Smut[3]					
1979	1.1	0.4	0	-	15.0
1980	0.2	0.1	0	-	2.0

1 - Percent leaf area diseased

2 - Percent root rot = $\frac{a + 2b + 4c}{10}$

where a,b,c = percent of plants in the categories slight, moderate and severe disease respectively

3 - Percent population affected

with net blotch In this situation, C sativus was probably a secondary invader of these lesions.

Leaf scald, incited by Rhynchosporium secalis, also commonly occurred in many fields. This disease was found in all early seeded fields but remained at low levels for June and early July. Disease levels rose dramatically however, following the period of wet weather in mid-July in both 1979 and 1980. Scald was not as prevalent on the top two leaves of barley in 1980 as was found in 1979 although traces could be found on lower leaves in most fields surveyed. This disease did, however, develop to a serious level in fields which had a previous history of scald and had been sown to barley for two or more years Since this disease overwinters on straw and debris and its spread is limited by rain splash dissemination (Ayesu-Offei and Cartier 1979), the role of the infected debris in increasing disease levels is obvious. Mean scald ratings were higher in 1980 (3.4%) than in 1979 (1.5%), even though the disease did not occur as often. Although mean disease ratings were low for both years, mean scald ratings for individual fields ranged to a high of 37.6% in 1980.

Other leaf spotting diseases which occurred on barley in 1979 and 1980 included powdery mildew (incited by Erysiphe graminis f. sp. hordei), leaf rust (incited by Puccinia hordei), selenophoma leaf spot (incited by Selenophoma donacis var. stomaticola), and barley yellow dwarf (incited by BYDV). These diseases developed to high levels only in isolated fields, particularly in fields which were sown late. Overall yield loss attributed to these diseases was low however.

Four diseases affecting spikelets were recorded. Loose smut, incited by Ustilago nuda, was quite common in fields in which home-grown seed and/or no fungicide seed treatments were used. In these fields, levels of 15% to 20% of the plant population was affected. In fields where certified and fungicide-treated seed was used, smut levels of only 1% was recorded.

Fusarium head blight, incited by various Fusarium species, was prevalent and caused serious losses in most fields, particularly in 1980. The high disease levels were probably the result of favorable

weather conditions for disease development and the presence of high inoculum levels from 1979 and from other infected crops, especially wheat. In addition to the actual yield loss, there was reduced germination of barley seed used for the 1981 cropping season. The Fusarium species occurring on barley were known toxin producers which caused additional problems with feed barley (Johnston, personal communication).

Two other diseases found to occur on spikes, but only at low levels, were seed blight (incited by Bipolaris sorokiniana), and ergot (incited by Claviceps purpurea).

2.3.3.5. Diseases occurring in mixed barley and oat fields

Mixed grain, mainly oats and barley, constitutes the largest hectarage of land sown to grain crops on Prince Edward Island. The diseases recorded in mixed fields were the same as those found in pure stands (Table 16). Disease levels were generally lower than were found in pure stands of these crops. In 1979, many fields were sown late and, as a result, mean disease ratings for net blotch on barley for the season in these fields were higher than in pure stands. A similar situation also existed for septoria leaf blotch on oats. The late sowing of these fields may also have been responsible for the trace amounts of leaf scald found on barley as this disease is more favored by early spring conditions (Clough and Johnston 1978a).

2.3.4. Conclusion

Quack grass was found in the marjority of cereal fields in 1979 and 1980. Although densities varied greatly from field to field, survey results indicated that quack grass often occurred at levels which could severely interfere with crop yields. In addition, because this species is the most important grass weed in cereal fields on Prince Edward Island (Ivany 1980), and is associated with many cereal pathogens, it may also be an important source of inoculum for cereal diseases.

TABLE 16: Mean disease ratings for diseases occurring on mixed grain

Crop	Disease	Mean[1]	Standard error	Maximum - Minimum
Barley	Net blotch			
	1979	22.9	8.3	1.4 - 91.9
	1980	2.7	0.6	1.2 - 15.9
	Scald			
	1979	trace	-	-
	1980	1.7	0.4	1.2 - 12.2
	Leaf blotch			
	1979	trace	-	-
	1980	2.7	1.0	1.2 - 28.7
Oats	Septoria leaf blotch			
	1979	18.2	5.1	1.3 - 90.7
	1980	3.4	1.4	1.2 - 40.9

1 - Ratings expressed as percent leaf area diseased

Thirty pathogens were identified on quack grass in eastern Canada during this survey. This list of pathogens may however, be incomplete With a survey covering such a wide area, it would be easy to overlook some pathogens. The most intensively surveyed area was Prince Edward Island. It should be noted that the area surveyed in Quebec was the region surrounding Macdonald Campus of McGill University. Only small areas of Nova Scotia and New Brunswick were surveyed. No data is available for other areas of these provinces with the exception of incidental reports. Surveys were based primarily on visible symptoms of disease. As a result, organisms causing no apparent symptoms but still pathogenic on the plant would be overlooked. Variations in levels of pathogens, as a result of weather or other factors, from one year to the next may also have influenced which species were found during any survey period in question. Phyllachora graminis, for example, was commonly found at high levels on quack grass in most areas of Prince Edward Island in 1979. In contrast, this pathogen was found only occasionally in 1980 and could have easily been overlooked in some instances. More intensive surveys of quack grass in eastern Canada covering a wider area and including more cropping systems and undisturbed areas would therefore provide a more comprehensive list of pathogens occurring on this species.

In cereal disease surveys, several diseases were found to be widespread in all cereal growing areas. Septoria leaf blotch and fusarium head blight were the most commonly found diseases of wheat. The most serious disease of oats was septoria leaf blotch. Common root rot and net blotch were the most serious diseases found on barley during this survey. Other diseases found on these crops occurred only at low levels or, if serious, were problems in isolated areas only. A great variation in levels of many diseases was found from one year to the next or from one area to another. This is an indication of the importance of annual regional surveys to determine which diseases are likely to cause problems. The pathogens responsible for these diseases were also found on quack grass, with the exception of septoria leaf blotch of oats. There is a need to establish the extent of the role which quack grass may play in the epidemiology of cereal pathogens.

III. HOST SPECIFICITY OF SELECTED FUNGI ISOLATED FROM PLANT MATERIAL

3.1. Introduction

The concept of host specificity of pathogens occurring on quack grass is important in two ways. First, it is essential in understanding the role of this host in the perpetuation or establishment of any disease occurring on an economic grass crop. Second, host specificity studies are a critical phase of a biological control strategy against this weed. Before any pathogen can be seriously considered as a potential biological control agent, the specificity of the organism must be clearly understood.

Most pathogens show a high degree of specificity, particularly the rusts and mildew, as noted by Brian (1976). The evolution of such specialized pathogens is thought to be strictly bound to that of their hosts (Ciccarone 1976). Eshed and Dinoor (1981) have suggested that specificity of host/parasite relationships reflects many random events that accompany host development in a region in seasons where the parasite is very active. Adapted phenotypes, particularly if specialized, may be subjected, through the host, to an ecological isolation followed by genetic barriers and speciation (Ciccarone 1976). Therefore, the longer a pathogen and a particular host evolve together, the greater is the chance of a more specialized host-pathogen relationship developing.

The specificity of organisms found on quack grass has not been studied to any extent, with the exception of physiologic races of pathogens occurring on economic crops and in respect to the ability of quack grass to serve as a source of inoculum for pathogens of economic crops.

Table 17 lists those pathogens of quack grass which have a restricted host range or for which physiological races of a limited host range are known. This list does not include those pathogens, such as Fusarium species or Bipolaris sorokiniana for which variation in virulence or aggressiveness may exist but have not been assigned the status of a special physiological form. In such cases, the variation reported

TABLE 17 Pathogens of quack grass reported to have a restricted host range

Pathogen	Disease	Reference
Apiocarpella agropyri	leaf spot	Green 1950
Erysiphe graminis f sp *agropyri*	powdery mildew	Marchal 1902
Leptosphaeria anisomeres	leaf spot	Wehmeyer 1942
Phleospora graminearum	leaf spot	Hardison and Sprague 1943
Physoderma graminis	brown spot	Thirumalachar and Dickson 1947
Puccinia coronata	crown rust	Eriksson 1909
Puccinia graminis	stem rust	Eriksson 1894
Puccinia recondita var. *agropyrina*	leaf rust	Eriksson 1894
Puccinia striiformis	stripe rust	Eriksson 1894
Pythium perilum	root rot	Sprague 1950
Rhynchosporium secalis	leaf scald	Caldwell 1937
Tilletia earlie	smut	Brenckle 1918
Ustilago bullata (*U. agropyri*)	stem smut	Zundel 1953
U. elytrigae	smut	Anonymous 1963
U. spegazzini var. *agrestis*	stem smut	Fischer and Hirschorn 1945

may be a result of pathogen variation rather than a special host parasite interaction

Only two pathogens have been reported to be specific to quack grass Apiocarpella agropyri Sprague (Green 1950) and Leptosphaeria anisomeres Wehm. (Wehmeyer 1942) Little information is available on these two organisms Both pathogens induce leaf spotting and the former has been reported to be quite parasitic on leaves of quack grass (Sprague 1950) A lack of research on these organisms may explain their limited known host range

There is some confusion concerning the specificity of the other pathogens listed in Table 17 Host specific forms have been reported for Puccinia species (Eriksson 1894), Erysiphe graminis DC f sp agropyri Marchal (Marchal 1902) and Rhynchosporium secalis (Caldwell 1937). However, several reports indicate that the host ranges of these organisms may not be as strict as previously reported Hardison (1944) reported isolates of E graminis from quack grass that could attack wheat, barley and Elymus species. Peturson (1954) reported the existence of Puccinia coronata f. sp. secalis on quack grass which had a much wider host range than previously reported forms. Schwinghamer (1955) has also reported a form of P. coronata in North Dakota similar to P coronata f sp secalis.

The specificity of forms of P. striiformis was placed in doubt by reports of Straib (1935, 1937) who reported that isolates of P. glumarum (P. striiformis) from quack grass in Germany were highly pathogenic to American kanred red wheat, even though German wheats were resistant. Popov (1979) also reported isolates of P. striiformis from quack grass could attack wheat. However, distinct biological races of P. striiformis have been reported by Straib (1935, 1937), Mehta (1924) and Manners (1950). Since Rudorf (1929) has reported distinct physiological differences between European and North American isolates, further investigations may reveal forms which display higher levels of specificity.

There are few reports on the existence of host specific forms of P. graminis and it is generally assumed that races of P. graminis,

although greatly restricted in host range, are not restricted in host range to one species (Eriksson 1894, Stakman and Piemeisel 1917, Mehta 1923 and Batts 1950). Prasda (1947) however, reported the existence of P. graminis Pers. f. sp. agropyri Mehta and Prasda, which differed physiologically from any other form of P. graminis and was restricted in host range to three Agropyron species. Therefore, sufficient contradictions exist which suggest the necessity of additional work on the host specificity of various isolates of P. graminis.

Puccinia recondita, one of the most highly specialized of the cereal rusts, is the only graminaceous rust for which a specific forma speciales is reported on quack grass (Samborski, personal communication). However, there is still controversy regarding the taxonomy of P. recondita and Cummins (1971) treated the species as a "species complex" encompassing over 50 synonyms for the species. There has been extensive subdivision of the species mainly based upon host specialization (Mains 1933). Host specific forms from quack grass have been reported by Mains (1933), Wilson and Henderson (1966) and Markova (1976). Quack grass isolates of P. recondita lacking this strict host specialization were reported by Fischer (1935), Guyot (1944) and Markova (1976). Markova (1976) used the name P. persistens which may be considered synonymous with P. recondita.

Controversy also surrounds the specificity of Rhynchosporium secalis, the causal organism of scald of grasses. Caldwell (1937) reported a high degree of specialization, each race being restricted to its own host. This conclusion was supported by results of Müller (1953), Reed (1957), Owen (1958) and Kline (1960). However, conflicting results were reported by Bartels (1928), Smith (1937), Sarasola and Campi (1947), Schein (1958) and Kay and Owen (1973). Ali (1972) attributed the conflicting results reported to variability in environmental conditions between the field and glass house and suggested that R. secalis can possibly develop in the tissue of many host genera only under favorable conditions. Further work is required to establish if the reported specificity is a result of a failure to recognize optimum infection conditions or if the lack of specificity is an artifact of prevailing environmental conditions and not important under field conditions (Shipton et al. 1974).

Eight species causing various smuts of quack grass and other grasses have been reported to have a restricted host range. They are 1. Tilletia earlie Griff. (Brenckle 1918), 2. T. tritici-repentis DeCandolle and Liro (Zundel 1953), 3. Ustilago agropyri Bisby Buller (U. bullata Berkeley) (Zundel 1953), 4. U. agropyrina Lavrov (U. longissima var. dubiosa Liro, U. spegazinii Hiesch.) (Zundel 1953), 5. U. calamagrostidis (Fuckel) Clint., 6. U. jamalainei Liro (Zundel 1953), 7. Ustilago spegazzinii var. agrestis (Syd.) Fisch. and Hirsch (Fischer and Hirschorn 1945), 8. U. trebouxi (Zundel 1953). One other smut organism (U. elytrigae) has been reported on quack grass, but no other information is available (Anonymous 1963).

The taxonomy of several of these organisms is unclear and the validity of them as species has been disputed by a number of authors (Fischer 1937; Fischer and Hirschorn 1945, Fischer 1953, Duran and Fischer 1961). Therefore, the specificity of these pathogens is unclear. U. spegazzinii, U. bullata and T. earlie are currently accepted as valid species. Only one reference to U. elytrigae has been found (Anonymous 1963) and, therefore, the status of this species is unclear.

Little information is available on the specificity of Phleospora graminearum and Pythium perilum and this lack of information and research on these two organisms may be responsible for the limited host range reported in the literature. Although Physoderma graminis was originally reported to be host specific (Thirumlachar and Dickson 1947), Sparrow and Griffin (1964) have found that the epibiotic stage of this organism could be produced on various grasses. The organism has not been reported to occur on grasses other than quack grass in nature. The host specificity of this organism, therefore, merits further investigation.

Therefore, the actual number of pathogens restricted in host range to quack grass and a few related species is still unknown. Reasons for this are varied. Checklists and indices may have been published classifying pathogens according to hosts without following Koch's postulates for each pathogen on each host (Dinoor 1974). Also, many reports are the results of tests with one particular isolate of a pathogen without regard to the existence of other physiologic forms

or races. Extensive variation has been reported for pathogens such as Puccinia graminis (Stakman and Piemeisel (1917), P. recondita (Mains 1933, Cummins 1971) and Rhynchosporium secalis (Ali and Boyd 1974). Ali and Boyd (1974) have also reported the existence of intra-isolate variability in both host reaction and isolate pathogenicity of R. secalis. Yet, little information is available on the reaction of quack grass to pathogen variation in different isolates.

Host range lists are also incomplete in respect to variation of the host. Variation in quack grass has been reported to be extensive (Jansen 1951, Palmer and Sagar 1963, Williams 1973, Neuteboom 1975, 1980). No information, however, exists on the reaction of different populations or different clones to one or more pathogen isolates. The use of different varieties with different disease reactions may also explain the apparent discrepancies in results of host range tests of pathogens such as Rhynchosporium secalis and Erysiphe graminis.

Therefore the existence of specific varieties of many pathogens is a distinct possibility as exemplified by the specificity of cereal rusts. This possibility is also supported by physiologic specialization of other pathogens occurring on grass species such as Fusarium species (Tu 1930), Claviceps purpurea (Stager 1903, Mantle and Shaw 1977), Urocystis agropyri (Fischer and Holton 1943), Ustilago striaeformis (Davis 1935). However, isolates of pathogens from quack grass may have a broader host range than isolates occurring on economic crops as reported for R. secalis from barley and quack grass (Kay and Owen 1973) and various pathogens on wild grasses in Israel (Dinoor 1974, Eshed and Wahl 1970, 1975).

More extensive research is required to establish, in more detail, host-pathogen relationships. Research should be extended to account for host and pathogen variation. If pathogens prove to be specific, then studies may be directed to evaluating their use in biological control programs. Pathogens proving to be non-host specific may be sources of inoculum for diseases of economic crops and studies oriented to crop protection would follow.

3.2. Materials and Methods

Ten pathogens isolated from quack grass and one from barley were used for host specificity tests. Criteria for selection of those pathogens of quack grass tested were based on the known host range of the pathogen, on the existence of known races of the pathogen, or if the pathogen represented the first record of its occurrence on quack grass. In some cases, it was not possible to obtain inoculum for these tests (for example Phyllachora graminis), so they were not included in tests. Erysiphe graminis was not used in tests because of its ability to be widely disseminated and the threat of it contaminating other research material or subsequent experiments. One pathogen from barley, Pyrenophora teres was used because of the taxonomic confusion with P. japonica (Shipton et al. 1973) and because it has only been found on Hordeum species and Bromus diandrus Roth. in nature (Shipton et al. 1973).

A total of 51 species in the family Poaceae were screened for their susceptibility to Bipolaris sorokiniana. Not all 51 species were available for testing with the other 10 pathogens. Therefore, the number of plant species tests varied between 44 and 48 for the other pathogens. All tests were conducted in growth cabinets with a daylight period of 14 hours and day-night temperatures of 20°C and 15°C respectively. Each treatment was replicated four times and the experiments were repeated three times, unless otherwise stated. All pathogens were inoculated onto foliage with the exception of Urocystis agropyri which was inoculated by four different techniques.

3.2.1. Foliar inoculation

3.2.1.1. Non-obligate pathogens

Spray inoculation onto leaves of plants were used for Cochlibolus sativus, Drechslera biseptata, Pyrenophora bromi, Pyrenophora japonica, Pyrenophora teres, Rhynchosporium secalis and Septoria sp.. Single spore isolates, grown on potato dextrose agar for two weeks at 21°C

were used for all inoculations. Spore suspensions were prepared by pouring 10 ml of 0.5% solution of gelatin into five cultures of each organism. Gelatin was used as a surface wetting agent to obtain better adherence of spores to the leaf surface. The cultures were scraped with a flame-sterlized needle and the resulting suspension was poured into a sterlized blender jar. The suspension was blended for 15 seconds and then filtered through three layers of cheesecloth to remove unwanted pieces of agar and large pieces of mycelium. The pathogens used, the culture media they were grown on and the final spore concentrations used for inoculation are presented in Table 18.

Plants were inoculated at three weeks of age by spraying the suspension onto the plants with an atomizer until the spray was dropping off the plants. Plants were incubated for 24 hours in plastic bags and then placed in growth cabinets. Isolations were made from plants showing disease symptoms to confirm the identity of the causal organisms.

3.2.1.2. Obligate pathogens

Three rust fungi were used for host specificity tests. Inoculum was produced on quack grass plants grown in growth cabinets. Inoculum was collected by suction of spores from infected leaves. To inoculate leaves, plants were first sprayed with a 0.5% solution of gelatin to wet the leaves. Spores were then applied to leaf surfaces by placing spores on the tips of fingers and rubbing the leaves between these fingers. Plants were placed in the dark for 24 hours at room temperature and in plastic bags. Plants were then transferred to growth cabinets. Rust reaction was determined according to the method of Stakman et al. (1962). The rating system consisted of six infection types: 0,0;, 1, 2, 3, 4. Infection types 0, 0;, 1 and 2 were considered resistant reactions.

3.2.2. Inoculation of Urocystis agropyri (Preuss) Schroet.

Urocystis agropyri was inoculated by four different techniques: soil infestation, seed infestation, rhizome inoculation and seedling inoculation.

TABLE 18: Pathogens used for foliar inoculation

Pathogen	Culture media	Spore concentration (spores/ml.)
Cochliobolus sativus	PDA	50 000 - 60 000
Drechslera biseptata	PDA	75 000 - 90 000
Pyrenophora bromi	V-8 juice agar	50 000 - 60 000
Pyrenophora japonica	V-8 juice agar	only mycelium used
Pyrenophora teres	V-8 juice agar	50 000 - 60 000
Rhynchosporium secalis	Lima bean agar	500 000 -550 000
Septoria	Czapec-Dox-V-8 juice agar	1 000 000

3.2.2.1. Soil infestation

Ten seeds of each species screened were sown in "Promix" in 10 cm diameter pots. The spore suspension was prepared in the following manner. Quack grass leaves infected with U. agropyri were crushed in sterile distilled water with a mortar and pestle. The resulting suspension was filtered through three layers of cheesecloth. The spore concentration was adjusted to 50 000 - 60 000 spores/ml. Twenty-five millilitres of spore suspension were poured onto the soil at sowing, at plant emergence and at four weeks growth, at which time the plants were clipped to allow regrowth. Controls were established by crushing healthy quack grass leaves in sterile distilled water, filtering this through cheesecloth and pouring 25 ml of the filtrate onto the soil at times specified above. Results were recorded four weeks following clipping to allow for any growth of infected buds which may have been previously suppressed by apical dominance.

3.2.2.2. Seed infestation

A spore suspension of U. agropyri was prepared in the same manner as outlined in Section 3.2.2.1. Seeds were placed in the spore suspension in a filtering flask. The flask was then sealed and the air in the flask removed by means of a vacuum pump. Seeds were left in the suspension in the vacuum for five minutes. It was assumed that spores infiltrated the seed through cracks and crevices in the seed coat when the vacuum was released. Ten seeds of each species screened were sown in "Promix" in 10 cm diameter pots and placed in growth cabinets. Plants were clipped after four weeks growth and results were recorded as number of infected shoots after four weeks regrowth.

3.2.2.3. Rhizome inoculation

A spore suspension was prepared as outlined in Section 3.2.2.1. One bud rhizome pieces were soaked for 24 hours in the spore suspension in a filtering flask. Air was then removed from the flask with use of

a vacuum pump, and the rhizome pieces were left in the suspension in the vacuum for five minutes. Release of the vacuum allowed infiltration of the spores into the rhizomes. Controls consisted of rhizome pieces soaked for 24 hours in a filtrate from crushed healthy quack grass and then placed in a vacuum for five minutes.

A second rhizome inoculation method used involved injecting the spore suspension into the axil of the rhizome bud with a hypodermic syringe. Rhizome buds injected with the filtrate from crushed healthy leaves were used as controls.

Five rhizome pieces from each treatment were sown in "Promix" in 10 cm diameter pots and placed in growth cabinets. The plants were cut back after four weeks growth to allow for any growth of infected dormant buds.

3.2.2.4. Seedling inoculation

Seed of plants were germinated at 21°C in petri dishes on filter paper moistened with the spore suspension of U. agropyri. Germinated seeds were left in the petri dishes in the dark at room temperature for two days following germination. This would allow for infection of the emerging radicle and coleoptile. Ten germinated seeds of each species were sown in "Promix" in 10 cm diameter pots and placed in growth cabinets. Plants were clipped four weeks following emergence and results were recorded after four weeks regrowth.

3.3. Results and Discussion

Host specificity tests were conducted to determine: 1. if there were any potential biological control agents of quack grass; and 2. which crop pathogens were most likely to be influenced by the presence of quack grass. A pathogen with a broad host range would not be considered a potential biological control agent and, in this situation, quack grass might serve as a source of inoculum for this pathogen.

3.3.1. Bipolaris sorokiniana (Sacc. in Sorok.) Shoem.

B. sorokiniana (perfect stage: Cochliobolus sativus) had the widest host range of all pathogens tested (Table 19). All 51 grass species tested developed symptoms with some species more susceptible than others. More than 10% leaf area diseased was considered a susceptible reaction whereas plants with ratings of less than 10% were considered to be moderately resistant. Symptoms on oats and wild oats were small necrotic spots or flecks rather than the typical brown spot. These may have been a host reaction similar to the hypersensitive reactions reported for various pathogens and probably represented a resistance reaction to the pathogen. Similar flecking occurred on corn. Results presented here agreed closely with results reported by Berkenkamp (1971). Christensen (1922) reported an extremely wide host range, the pathogen attacking wheat, barley, rye and 83 species of wild grasses belonging to 37 different genera. Nelson and Kline (1962) also reported a wide host range for this organism, but reported marked variation in pathogenicity between different isolates. They attributed this variation to genetic difference among the isolates as a result of mutation and recombination between strains of this species. Hynes (1935) reported a total of 31 physiologic forms of this pathogen.

B. sorokiniana has been reported as a pathogen on quack grass in the United States (Drechsler 1923b), Canada (Padwick and Henry 1933), Great Britain (Sampson and Western 1941), Denmark (Anderson 1955), U.S.S.R. (Kozyreva 1958), New Zealand (Blair 1936) and possibly other countries. In view of the wide distribution of B. sorokiniana and quack grass and the broad host range of B. sorokiniana, it is unlikely that quack grass specific strains of this pathogen will be found. Therefore emphasis of study of this pathogen occurring on quack grass should be directed toward the influence of quack grass on disease levels on cereal crops.

TABLE 19: Host range of some leaf spotting fungi isolated from quack grass

Plant Species	Host reaction[1]						
	Bipolaris sorokiniana	Dreschslera biseptata	Pyrenophora bromi	Pyrenophora japonica	Pyrenophora teres	Rhynchosporium secalis	Septoria species
Agropyron cristatum (L.) Gaertn.	+++	++	++	+++	+++	0	+++
A. dasystachyum (Hook.) Scribn.	+++	++	0	+++	+++	0	+++
A. elongatum (Host) Beauv.	+++	++	0	+++	+++	0	+++
A. intermedium (Host) Beauv.	+++	++	++	+++	+++	0	+++
A. repens (L.) Beauv. (P.E.I.)	+++	+++	++	+++	+++	++	+++
A. repens (L.) Beauv. (Que.)	+++	+++	++	+++	+++	++	+++
A. repens (L.) Beauv. (Sask.)	+++	+++	++	+++	+++	n.t.	+++
A. riparium Scribn. & Smith	+++	++	0	+++	+++	0	+++
A. smithii Rydb.	+++	++	0	+++	+++	0	+++
A. trachycaulum (Link) Malte	+++	++	0	+++	+++	0	+++
A. trichophorum (Link) Richt.	+++	++	0	+++	+++	0	+++

TABLE 19 (cont.)	Bipolaris sorokiniana	Dreschslera biseptata	Pyrenophora bromi	Pyrenophora japonica	Pyrenophora teres	Rhynchosporium secalis	Septoria species
Agrostis alba L.	++	++	0	++	++	0	++
Alopecurus arundinaceus Poir.	++	++	0	++	++	0	0
Avena sativa L. (cv. Laurent)	+	+	0	++	++	0	0
Avena fatua L.	+	+	0	++	++	n.t.	n.t.
Bromis inermis Leyss (cv. Saratoga)	+++	++	+++	++	++	0	+++
Bromus species	+++	++	+++	++	++	0	+++
Dactylis glomerata L. (cv. Frode)	+++	++	0	++	++	0	++
Digitaria ischaemum (Schreber) Schreber ex Muhl.	++	++	0	0	0	0	0
Echinochloa crus-galli (L.) Beauv.	++	++	0	0	0	0	0
E. crus-galli var. frumentacea (Ruxb.) link	++	n.t.	0	0	0	n.t.	
Elymus angustus Trin.	+++	++	0	+++	++	0	+++
E. canadensis L.	+++	++	0	+++	++	0	+++
E. cinereus Scribn. and Merr.	+++	++	0	++	++	0	+++

TABLE 19 (cont.)	Bipolaris sorokiniana	Dreschslera biseptata	Pyrenophora bromi	Pyrenophora japonica	Pyrenophora teres	Rhynchosporium secalis	Septoria species
E. condensatus Presl.	+++	++	0	++	++	0	+++
E. junceus Fisch.	+++	++	0	++	++	0	+++
Festuca arundinaceae Schreb.	+++	++	0	++	++	0	0
F. longifolia Thuill.	+++	++	0	++	++	0	0
F. pratensis Hudson	+++	++	0	++	++	0	0
F. rubra L.	+++	++	0	++	++	0	++
F. rubra L. var. commutata Goud.	+++	++	0	++	++	0	++
F. tenufolia Sibth.	+++	++	0	++	++	0	++
Hordeum jubatum L.	+++	+++	0	+++	+++	0	++
Hordeum vulgare L. (cv. Loyola)	+++	+++	0	+++	+++	0	++
H. vulgare L.(cv. Laurier)	+++	+++	0	+++	+++	0	++
H. vulgare L.(cv. Volla)	+++	+++	0	+++	+++	0	++
Lolium multiflorum Lam.	+++	++	0	++	++	0	0
L. perenne L.	+++	++	0	++	++	0	0
Muhlenbergia species	++	++	0	++	++	0	0

TABLE 19 (cont.)

	Bipolaris sorokiniana	Dreschslera biseptata	Pyrenophora bromi	Pyrenophora japonica	Pyrenophora teres	Rhynchosporium secalis	Septoria species
Oryza sativa L.	+++	n.t.	n.t.	n.t.	n.t.	n.t.	n.t.
Panicum capillare L.	++	++	0	0	0	0	0
Panicum miliaceum L.	++	n.t.	n.t.	n.t.	n.t.	n.t.	n.t.
Pennisetum typhoides (Burm.) Stapf & Hubbard	++	n.t.	n.t.	n.t.	n.t.	n.t.	n.t.
Phalaris arundinaceae L. (cv. MCRI)	+++	+++	0	++	++	0	0
Phelum pratense L.	+++	++	0	0	0	0	0
Poa compressa L.	+++	++	0	0	0	0	++
Poa pratensis L.	+++	++	0	0	0	0	+++
Secale cereale L. (cv. Kustro)	+++	+++	0	++	++	0	+++
Setaria glauca Beauv.	++	++	0	0	0	0	0
S. italica (L.) Beauv.	++	++	0	0	0	0	0
S. viridis (L.) Beauv.	++	+++	0	0	0	0	0
Stipa viridula Trin.	++	+++	0	0	0	0	++
Triticum aestivum L. (cv. Glenlea)	+++	+++	0	++	++	0	+++
T. aestivum L. (cv. Frederick)	+++	+++	0	++	++	n.t.	+++

TABLE 19 (cont.)	Bipolaris sorokiniana	Dreschslera biseptata	Pyrenophora bromi	Pyrenophora japonica	Pyrenophora teres	Rhynchosporium secalis	Septoria species
Triticale	+++	+++	0	++	++	n.t.	+++
Zea mays L.	+	+	0	0	0	n.t.	0

1 - Host reaction

0 - immune no reaction

\+ - flecking, perhaps a hypersensitive response (resistant)

++ - small restricted leaf spots, pathogen not always re-isolated (moderately resistant)

+++ - leaf spots covering > 10% of leaf area, pathogen re-isolated from diseased tissue

n.t. - not tested

3.3.2. Drechslera biseptata (Sacc. & Roum.) Richardson & Fraser

The isolation of this pathogen is the first record of this pathogen occurring in Canada and the first record of occurrence of D. biseptata on quack grass. This organism has rarely been reported as a pathogen of cereal crops and, as a result, little information is available on the pathogenicity of D. biseptata. It has been reported as a pathogen of wheat and corn in the U.S.S.R. and is a known phytotoxin producer (Brynza et al. 1977).

D. biseptata was isolated from the rhizomes of quack grass and roots of barley. However, when artificially inoculated onto leaves, this organism causes leaf spotting. Because infection could apparently occur through the leaves, foliar inoculation was used for host specificity tests. Symptoms on Avena sativa, A. fatua and Zea mays were flecks similar to those recorded for B. sorokiniana and probably represented a resistance reaction. Disease ratings of greater than 10% leaf area affected were recorded only on quack grass, Hordeum jubatum, H. vulgare, Phalaris arundinaceae, Secale cereale, Triticum and Triticale (Table 19). Symptoms on these hosts consisted of small irregular brown spots and disease ratings were never higher than 20% of the leaf area. The restricted development of this pathogen on leaves, when compared to extensive development of B. sorokiniana suggests its more important role being a root pathogen. Root inoculation of this pathogen yielded no leaf symptoms and, in many cases, it was difficult to detect symptoms on the root system of various plants. Therefore, this pathogen may be a weak parasite on many grasses.

D. biseptata has also been isolated from barley and may therefore play a role as part of a complex of organisms causing root rot of barley and other cereals. The extent of damage on these crops, however, is still unknown.

3.3.3. Pyrenophora bromi (Died.) Drechsl.

P. bromi was isolated in April 1980 from quack grass growing in a stand of brome grass (Bromus species) which showed extensive lesion

development resulting from infection by P. bromi. This pathogen is almost identical in morphology to P. tritici-repentis. Therefore, confusion surrounds the identity of the isolate from quack grass. Unfortunately, confirmation of identification is not available and original cultures have subsequently been lost. The pathogen was assumed to be P. bromi, based upon its early occurrence in the field and the restricted host range, as recorded in host specificity tests.

Shoemaker (1962) reported that P. bromi appears to be restricted to Bromus species while P. tritici-repentis has the widest host range of all species in the genus Drechslera (imperfect stage of Pyrenophora). In host specificity tests reported here, disease development was restricted to Bromus species, Agropyron intermedium and quack grass (Table 19). Disease symptoms on quack grass and A. intermedium were characterized by small brown spots with a slight chlorotic margin surrounding the lesions. Symptoms on Bromus, however, were characterized by much larger circular brown lesions with prominent chlorotic halos. These symptoms closely resembled symptoms found on Bromus in nature.

P. bromi is probably not a common pathogen occurring on quack grass. Quack grass infected by this pathogen was found only in stands of Bromus species severely infected by this pathogen. This suggests that conditions in the stand were so favorable for disease development and inoculum levels were high enough that the pathogen could infect quack grass. Infection may not occur under other less favorable conditions. Wehmeyer (1961) considered P. bromi a physiologic form of Pleospora trichostoma (Fr.) Ces. & de N. (Pyrenophora trichostoma (Fr.) Fckl.), a pathogen previously recorded on quack grass in eastern Canada (Wehmeyer 1950). P. trichostoma has also been reported as a pathogen of several grasses including wheat (Conners 1967). If P. bromi is a physiologic form of P. trichostoma, then it is conceivable that, under particularly favorable conditions or under experimental conditions, P. bromi may infect quack grass.

3.3.4. Pyrenophora japonica Ito & Kurib.

P. japonica was not restricted in its host range, infecting all but 12 species tested (Table 19). Disease development was restricted on 23 species, while 18 species were considered susceptible to this organism. Prior to this, only rye had been reported as a host to this pathogen in Canada (Conners 1967). In these tests, wheat, barley, rye, all species of Agropyron tested, three species of Elymus and Hordeum jubatum were susceptible. If this species is considered to be synonymous with P. teres (Kenneth 1962), then this host range agrees closely with the reported host range of P. teres. However, as discussed earlier, Shoemaker (1962) considered P. japonica and P. teres to be separate and valid species.

The lack of specificity of this organism indicates that its potential use as a biological control agent is severely restricted. Quack grass may serve as a source of inoculum, but, since this organism is not a serious pathogen, quack grass probably has little influence on crop losses due to this organism.

3.3.5. Pyrenophora teres (Died.) Drechsl.

P. teres causes net blotch of barley and is probably confined to Hordeum species in nature (Shipton et al. 1973). Infection of quack grass has occurred only under experimental conditions (Singh 1962). Shipton et al. (1973) stated that it may be possible for P. teres to sporulate on the surface of lesions on resistant grasses in nature. For these reasons and also because of the confusion concerning the taxonomy of P. teres and P. japonica, this organism was included in host specificity tests.

The host range of P. teres was the same as the host range of P. japonica (Table 19), suggesting that the separation of these two as separate species may be questioned. The only difference noticed was that P. japonica developed more extensively on Elymus species than did P. teres. The host range reported here is broader than any previously reported host range for this organism. Agrostis and Muhlenbergia genera were not previously reported to be susceptible to this pathogen.

However, this host range is much broader than that found in nature. Therefore, the experimental host range may not resemble the host range of this pathogen under natural conditions which may be restricted to Hordeum species (Kenneth 1962; Shipton et al. 1973). However, in view of the broad experimental host range, it is possible that P. teres may attack genera other than Hordeum in nature if conditions are favorable.

3.3.6. Rhyncosporium secalis (Oud.) J.J. Davis

The isolate of R. secalis used in host specificity tests was host specific (Table 19). Clones of quack grass from Quebec and Prince Edward Island were susceptible to the isolate used. However, infection occurred only in the first trial. Subsequent trials failed to yield disease symptoms on any species including quack grass. Cultures lost their ability to sporulate very quickly, particularly after subculturing. Also, the cultures were very slow growing and often became contaminated before they could be used. Similar problems were encountered by Caldwell (1937) and Kay and Owen (1973). In addition, environmental conditions appear to be critical for infection and may vary between isolates obtained from different hosts. Also, infection of different hosts may require different environmental conditions. Inadequate environmental conditions or unfavorable culture conditions may have been responsible for the lack of infection. Ali and Boyd (1974) suggested that failure to recognize proper environmental conditions may have been responsible for reports of restricted host ranges. Conclusions concerning the host range of quack grass isolates of R. secalis cannot be made until further studies are conducted.

Under field situations in Prince Edward Island, R. secalis has been recorded on barley, rye and quack grass. However, quack grass infected with R. secalis was never observed within a crop, even within barley crops heavily infected with R. secalis. Therefore, in nature, R. secalis forms from different hosts may be restricted to the hosts on which they occur.

3.3.7. Septoria species

An unidentified Septoria species was isolated from quack grass growing in a wheat field in Prince Edward Island. The organism was suspected to be S. nodorum but the identification has not been confirmed. Agropyron species, Bromus species, Poa pratensis, Secale cereale, Triticum aestivum and Triticale were susceptible to this pathogen (Table 19). Symptoms on susceptible plants were necrotic spots or extensive tip burn. The development of pycnidia on leaf tissue and sporulation were considered positive confirmation of pathogenicity. Eleven species showed typical tip burn symptoms but did not support sporulation. Seventeen species showed no disease development. Brokenshire (1975) reported similar symptoms for S. tritici on various Graminaceous hosts. This pathogen is, therefore, unspecialized and capable of attacking and sporulating on a number of different grasses. Therefore, any grass which is susceptible to this pathogen and can support sporulation may play an important role as a source of inoculum for this pathogen.

3.3.8. Urocystis agropyri (Preuss) Schroet.

U. agropyri was found only in Quebec and results of host specificity tests, indicate that this pathogen was specific to quack grass (Table 20). No trace of infection was noted on any other species. The mean percentage infection in all tests was 23.0%. There was no significant difference in disease reaction of the various collections of quack grass. Percentage infection was low and four different inoculation techniques were used in attempts to increase infection levels. Maximum percent infection obtained was with soil infestation (25.5%) but this technique did not differ significantly from rhizome inoculation or seedling inoculation ($\alpha = 0.05$). It did differ significantly from seed infestation which had the lowest percent infection (18%). Rhizome inoculation and seedling inoculation, however, did not differ significantly from seed infestation. Results, therefore, indicate that inoculum in the soil probably contributes more to disease levels than does infected seed.

TABLE 20: Host range of Urocystis agropyri isolated from quack grass

Plant species	Inoculation technique	Percent of Plants Infected: soil infestation	seed infestation	rhizome inoculation	seedling inoculation
Agropyron cristatum (L.) Gaertn.		0	0	0	0
A. dasystachum (Hook) Scribn.		0	0	0	0
A. elongatum (Host) Beauv.		0	0	0	0
A. intermedium (Host) Beauv.		0	0	0	0
A. repens (L.) Beauv. (Seed, P.E.I.)		22.5	20.0	-[1]	25.5
A. repens (L.) Beauv. (rhizome P.E.I.)		30.0	-	21.0	-
A. repens (L.) Beauv. (seed, Que.)		24.0	15.5	-	25.5
A. repens (L.) Beauv. (rhizome, Que.)		31.0	-	26.5	-
A. repens (L.) Beauv. (seed, Sask.)		15.5	19.0	-	19.0
A. riparium Scribn. & Smith		0	0	0	0
A. smithii Rydb.		0	0	0	0

TABLE 20 (cont.)

	soil infestation	seed infestation	rhizome inoculation	seedling inoculation
A. trachycaulum (Link.) Malte	0	0	0	0
A. trichophorum (Link.) Richt.	0	0	0	0
Agrostis alba L.	0	0	0	0
Alopecurus arundinaceus Poir.	0	0	0	0
Avena sativa L. (cv. Laurent.)	0	0	0	0
Avena fatua L.	0	0	0	0
Bromus inermis Leyss (cv. Saratoga)	0	0	0	0
Bromus species	0	0	0	0
Dactylis glomerata L. (cv. Frode)	0	0	0	0
Digitaria ischaemum (Schreber) Schreber ex Muhl.	0	0	0	0
Echinochloa crus-galli (L.) Beauv.	0	0	0	0
Elymus angustus Trin.	0	0	0	0
E. canadensis L.	0	0	0	0

TABLE 20 (cont.)

	soil infestation	seed infestation	rhizome inoculation	seedling inoculation
E. cinereus Scribn. & Merr.	0	0	0	0
E. condensatus Presl.	0	0	0	0
E. junceus Fisch.	0	0	0	0
Festuca arundinaceae Schreb.	0	0	0	0
F. longifolia Thuill.	0	0	0	0
F. pratensis Hudson.	0	0	0	0
F. rubra L.	0	0	0	0
F. rubra L. var. commutata Gaud.	0	0	0	0
F. tenufolia Sibth.	0	0	0	0
Hordeum jubatum L.	0	0	0	0
Hordeum vulgare L. (cv. Loyola)	0	0	0	0
Lolium multiflorum Lam	0	0	0	0
L. perenne L.	0	0	0	0
Muhlenbergia species	0	0	0	0
Panicum capillare L.	0	0	0	0

TABLE 20 (cont.)

	soil infestation	seed infestation	rhizome inoculation	seedling inoculation
Phalaris arundinaceae L. (cv. MCRI)	0	0	0	0
Phleum pratense L.	0	0	0	0
Poa compressa L.	0	0	0	0
P. pratensis L.	0	0	0	0
Secale cereale L. (cv. Kustro)	0	0	0	0
Setaria glauca Beauv.	0	0	0	0
S. italica (L.) Beauv.	0	0	0	0
Stipa viridula Trin.	0	0	0	0
Triticum aestivum (cv. Glenlea)	0	0	0	0
T. aestivum L. (cv. Opal)	0	0	0	0
T. aestivum L. (cv. Frederick)	0	0	0	0
Triticale	0	0	0	0
Zea mays L.	0	0	0	0

[1] test not applicable

Symptoms of smut usually did not appear until after plants were clipped and allowed to regrow. A similar situation was reported by Griffiths (1924) for U. agropyri on wheat. The pathogen may be slow growing in its host and clipping may allow the pathogen mycelium to grow along with the growing point of the plant.

The host range of U. agropyri reported here is contrary to what is reported in the literature. Clinton (1908), Davis (1922), Fischer and Hoton (1943) and Fischer (1951) reported that species in the genera Agropyron, Agrostis, Bromus, Calamagrostis, Elymus, Hordeum, Koeleria, Phleum, Poa, Sitanion, and Triticum were susceptible to U. agropyri. Fischer (1953) listed 47 different species of Gramineae as susceptible to this pathogen. In Canada, the pathogen has been reported on quack grass, Agropyron trachycaulum, Bromus ciliatus L., Elymus species, Hordeum jubatum and Poa compressa L. (Anonymous 1975). In Nova Scotia, this pathogen has been found on rattlesnake grass (Glyceria canadensis (Michx.) Trin. (Sampson, unpublished). No investigations of the host specificity of Canadian isolates have been reported.

These results indicate that a physiologic race may occur on quack grass in Quebec. However, the existence of contradicting reports suggests that the host range of this isolate should be studied more closely. Infection may be influenced by factors such as plant health and environmental conditions and such factors were not studied in these tests.

Studies with other isolates from other hosts are required to determine the existence of physiological specialization. The existence of physiologic specialization was shown by Fischer and Holton(1943). They identified three physiologic races of U. agropyri but none of their races were specific to one host. The quack grass isolate they used attacked Agropyron semicostatum, Elymus canadensis and Hordeum jubatum var. caespitosum. The Quebec isolate reported here did not infect E. canadensis nor H. jubatum, and A. semicostatum was not available for testing. Also, seed of G. canadensis was not available for testing. Therefore, additional work is required to establish the host range of this pathogen. However, it is possible that the Quebec isolate is specific and future work is required to confirm this.

3.3.9. Puccinia coronata Cda.

P. coronata had the widest host range of all rust species tested (Table 21). All species of Agropyron except A. dasystachum were susceptible to P. coronata. In addition, all species of the genera Elymus and Hordeum were susceptible as were Festuca arundinaceae, a Muhlenbergia species, Secale cereale and Triticale. The host range is similar to that reported by Peturson (1954) and Schwinghamer (1955) for P. coronata f. sp. secalis Peturson. The one exception is the susceptibility of F. arundinaceae. Swinghamer (1955) reported that members of the genus Festuca were resistant to this forma of P. coronata. F. elatior var. arundinacea (F. arundinacea) has also been reported to be generally resistant to P. coronata (Anonymous 1960).

The susceptibility of barley, rye, Triticale and various forage grasses suggests a possible role of quack grass as a source of inoculum for this pathogen. Uredia have been noticed on quack grass at the one to two leaf stage in early May and, therefore, may provide an early primary source of inoculum. This also suggests that the rust overwinters as mycelium in the leaves or rhizomes of quack grass. Since foliage of quack grass is killed by frost, it is more likely that the pathogen overwinters as mycelium in the rhizome. Bruizgalova (1951) reported that P. graminis f. sp. secalis can overwinter in the rhizomes of quack grass. Additional studies would be required to establish if a similar situation exists for P. coronata.

3.1.10. Puccinia graminis Pers.

Quack grass was the only species which showed a susceptible reaction to P. graminis (Table 21). A hypersensitive reaction occurred on a number of species and lesions of infection type one occurred on A. cristatum, A. riparium, A. smithii, E. canadensis, E. cinereus and E. junceus. In both cases, these are considered resistant reactions.

The forma of P. graminis commonly occurring on quack grass is P. graminis f. sp. secalis although P. graminis f. sp. tritici is known to occasionally occur on quack grass in nature (Stakman and

TABLE 21: Host range of *Puccinia* species isolated from quack grass

Plant species	Host Response[1]		
	Puccinia coronata	*Puccinia graminis*	*Puccinia recondita*
Agropyron cristatum (L.) Gaertn.	4	1	1
A. dasystachum (Hook.) Scribn.	0	0;	0;
A. elongatum (Host) Beauv.	3	0;	0;
A. intermedium (Host) Beauv.	3	0;	1
A. repens (L.) Beauv. (P.E.I.)	4	3	4
A. repens (L.) Beauv. (Que.)	4	3	4
A. repens (L.) Beauv. (Sask.)	4	3	4
A. riparium Scribn. & Smith	3	1	1
A. smithii Rydb.	4	1	1
A. trachycaulum (Link) Malte	3	0;	0;
A. trichophorum (Link) Richt.	3	0;	0;
Agrostis alba L.	0	0	0
Alopecurus arundinaceus Poir.	0;	0	0
Avena sativa L. (cv. Laurent)	0;	0	0
A. sativa L. (cv. Garry)	0;	0	0

TABLE 21 (cont.)	Puccinia coronata	Puccinia graminis	Puccinia recondita
A. sativa L. (cv. Stormont)	0;	0	0
A. fatua L.	0	0	0
Bromis inermis (Leyss (cv. Saratoga)	0;	0	0
Bromus species	0;	0	0
Dactylis glomerata L. (cv. Frode)	0;	0	0
Digitaria ischaemum (Schreber) Schreber ex Muhl.	0;	0	0
Echinochloa crus-galli (L.) Beauv.	0	0	0
Elymus angustus Trin.	3	0;	0
E. canadensis L.	4	1	0
E. cinereus Scribn. and Merr.	4	1	0
E. condensatus Presl.	4	0;	0
E. junceus Fisch.	4	1	0
Festuca arundinacese Schreb.	4	0	0
F. longifolia Thuill.	0	0	0
F. pratensis Hudson	0	0	0
F. rubra L.	0	0	0
F. rubra L. var. commutata Gaud.	0	0	0

TABLE 21 (cont.)

	Puccinia coronata	Puccinia graminis	Puccinia recondita
F. tenufolia Sibth.	0	0	0
Hordeum jubatum L.	4	0	0
Hordeum vulgare L. (cv. Loyola)	4	0	0
H. vulgare L. (cv. Laurier)	4	0	0
H. vulgare L. (cv. Volla)	3	0	0
Lolium multiflorum Lam.	0	0	0
L. perenne L.	0	0	0
Muhlenbergia species	3	0	0
Panicum capillare L.	0	0	0
Phalaris arundinaceae L. (cv. MCRI)	0;	0	0
Phleum pratense L.	0	0	0
Poa compressa L.	1	0	0
P. pratensis L.	0	0	0
Secale cereale L. (cv. Kustro)	4	0;	0;
Setaria glauca Beauv.	0;	0	0
S. italica (L.) Beauv.	0;	0	0
S. viridis (L.) Beauv.	0;	0	0
Stipa viridula Trin.	0;	0	0

TABLE 21 (cont.)

	Puccinia coronata	*Puccinia graminis*	*Puccinia recondita*
Triticum aestivum L. (cv. Glenlea)	0;	0;	0
T. aestivum L. (cv. Opal)	0;	0;	0
T. aestivum L. (cv. Frederick)	0;	0;	0
Triticale	4	0;	0;
Zea mays L.	0	0	0

1 0 – no visible reaction

0; – hypersensitive reaction but no uredia

1 – minute uredia surrounded by a distinct necrotic or chlorotic area

2 – small to medium uredia usually surrounded by a necrotic or chlorotic area

3 – medium-sized uredia, no necrosis but chlorosis, coalescence of uredia infrequent

4 – large, often coalescing uredia, no necrosis but chlorosis may be present

0, 0;, 1, 2 – resistant reactions

3, 4 – susceptible reaction

Piemeisel 1917). The host range reported here is, however, narrower than that reported by Stakman and Piemeisel (1917) and Stakman et al. (1918). These results suggest the existence of a more restricted form of P. graminis. However, it was difficult at first to obtain infection on quack grass and only 36% of inoculations yielded a susceptible reaction. Poor infection rates may have been due to inappropriate environmental conditions, an inadequate after-ripening period of the spores, or non-viable spores. Further studies are required to investigate more closely the host range of this organism. Also, host range studies should be conducted for those isolates of P. graminis which develop on hosts other than quack grass, to determine if these forms can also attack quack grass. This would give some indication of the possibility of parasexual hybridization between two forms of P. graminis on quackgrass. The restricted host range of this pathogen suggests that a role of quack grass as a source of inoculum for the form of P. graminis is unlikely.

3.1.11. Puccinia recondita Rob. ex Desm.

The isolate of P. recondita used was specific to quack grass (Table 21). All Agropyron species, Secale cereale and Triticale showed a "0;" or "1" infection type and were considered resistant. These results agreed with those reported by Eriksson (1894), Mains (1933), Wilson and Henderson (1966) and Samborski (personal communication). The host range, however, does not agree with that of Fischer (1935), who reported that A. dasystachum, A. tenerum, E. glaucus Buckl., Hordeum gussoneanum, H. jubatum, and H. murinum were very susceptible and E. canadensis, E. virginicus, and H. pusillum were moderately susceptible to an isolate of P. rubigo-vera (P. recondita) from quack grass. Quack grass, however, was susceptible to only those forms isolated from quack grass (Fischer 1935). Marked intraspecific differences were noted for various collections of wild grasses to various isolates of P. rubigo-vera (Fischer 1935).

Therefore, results reported here agree with the strict specialization of P. recondita as reported in the literature and indicate the presence of a highly specialized form of P. recondita in eastern Canada.

This rust is the most promising rust as a potential biological control agent. This form of P. recondita var. agropyri is apparently restricted to quack grass and does significant damage to its host. However, reports of a less specialized form of this rust, its close relationship to forms of P. recondita attacking wheat, the possible hybridization of P. recondita var. agropyri with other forms of P. recondita (Shifman 1958) and the reported intraspecific variation in rust reaction (Fischer 1935) are possible deterrents to biocontrol studies. Additional studies are required to investigate the existence of intraspecific variation in P. recondita var. agropyri, variation in host reaction and the relationship of this variety to other varieties and forms.

3.1.12. Conclusion

Results of host specificity tests are still inconclusive as to the exact host range of these organisms. While specificity tests under experimental conditions may give some indication of the host range and what hosts one may expect to find as hosts under natural conditions, they may not reflect the true situation in the field. Exact field conditions cannot be duplicated under experimental conditions and results may be an artifact of the experimental conditions. This has been suggested by Ali (1972, 1974) for Rhynchosporium secalis and may be the reason for the wide host range of Pyrenophora teres in this study. More extensive surveys for specific pathogens, host specificity tests conducted under field conditions, a more comprehensive understanding of host-pathogen relationships and the determination of exact conditions required for infection may help dispel some of the confusion concerning host ranges of various pathogens.

The existence of variation within a pathogen species and variation in host reactions within various collections of a host species suggests that further studies along these lines are necessary. Such studies would give a more complete picture of the host range of a pathogen and host-pathogen relationships. To do this, it is necessary to study a number of different isolates of a pathogen and their reactions on a number of collections of a host species as well as on a number of different plant species.

Three organisms were reported as host specific: 1. *Puccinia recondita* var. *agropyri*, 2. *Rhynchosporium secalis*, and 3. *Urocystis agropyri*. Therefore, further studies are required to determine the potential of these organisms as biological control agents and determine their relationship to pathogens occurring on cereal and other grass crops.

IV. QUACK GRASS AS A SOURCE OF INOCULUM OF SELECTED CEREAL PATHOGENS IN PRINCE EDWARD ISLAND

4.1. Introduction

Weeds, apart from reducing crop yield may serve as alternative hosts for many plant pathogens, including soil-borne, seed-borne and foliar pathogens, of crop species. Therefore weeds occurring near or within the crop may serve to increase disease levels in the crop. Close botanical affiliation between the crop and associated weed species, although important, is not always a requirement since many pathogens have a broad host range.

The relative importance of wild plants serving as a source of inoculum varies widely. Pathogens may be associated with weed hosts in three ways. First, pathogens may occur mainly on agricultural crops but weed hosts may occasionally be found diseased during the main season. Second, weed hosts may play, at least the same role as the main crop host to harbour and perpetuate the pathogen from one season to the next. Third, weed hosts may be found diseased during the crop season but their cardinal role is in the absence of the crop when they maintain and produce active inoculum. These hosts are of major importance in maintenance of the pathogen within the agroecosystem (Dinoor 1974). In addition to production of inoculum, weeds may serve to preserve and recombine pathogen genotypes, a process that may lead to the development of new pathotypes which may eventually spread and initiate epidemics on new or otherwise resistant varieties (Dinoor 1974).

Weed species and wild plants, therefore, complicate problems presented by various plant pathogens. These hosts may help maintain pathogens that would otherwise die out after a year or two, and may negate the benefits expected from break crops or rotations designed for control (Moore and Thurston 1970). Weed control with the sole objective of elimination of a pathogen reservoir is seldom considered economic. However, this practice may serve a useful role in an integrated approach to control of these diseases.

Due to the close genetic inter-relationships between grasses, wild grasses may be secondary hosts for a wide range of pathogens exhibiting a broad range of pathogenic variation (Eshed and Wahl 1970). As such, they may serve as a source of inoculum for several pathogens attacking economic grass crops. Since quack grass is one of the most common weed grasses occurring in cereals in eastern Canada, this grass may cause increased disease problems in cereal crops in this area.

4.1.1. Quack grass as a source of inoculum for cereal pathogens

If quack grass is a host of a pathogen that also attacks cereal crops, it is generally assumed that it may then provide a source of inoculum for that pathogen attacking cereal crops. This may not always be true, however, and in some cases, it is possible that quack grass may help reduce crop losses due to disease. Quack grass may serve as a trap species, particularly with nematodes. In this situation, quack grass receives inoculum normally available to the crop. Shearer and Zadoks (1972) demonstrated that the passage of Leptosphaeria nodorum (imperfect stage: Septoria nodorum) through a host may result in some degree of specialization in the pathogen . Harrower (1977) and Ao and Griffiths (1976) also reported increased specialization of L. nodorum and Septoria tritici after passage through a number of grasses, including quack grass. This phenomenon may be due to selection within the pathogen isolate by the secondary host for the genotype best adapted to the secondary host (Ao and Griffiths 1976). This, in turn, would weaken inoculum for the next season, possibly stabilizing the virulence below that found in well-adapted crop isolates (Ao and Griffiths 1976).

Quack grass is suspected of contributing to increased crop damage from more than 25 pathogens (Table 22) and, through further studies, this number can probably be increased. Quack grass may serve as a source of inoculum in many ways. Quack grass may directly increase pathogen inoculum by providing additional plants for sporulation, resulting in a more rapid buildup of inoculum levels. Quack grass is known to contribute to inoculum buildup of Puccinia graminis f. sp. secalis (Klebahn 1931, Green 1963), P. striiformis (Popov 1979) and

TABLE 22: Pathogens of economic crops for which quack grass has been reported to serve as a source of inoculum

Pathogen	Disease	Crop attacked	Reference
Agropyron mosaic virus	Agropyron mosaic	wheat	Slykhuis 1962
Brome mosaic virus	Brome mosaic	grass crops	Slykhuis 1967
Claviceps purpurea	Ergot	cereals	Campbell 1957
Cochliobolus sativus	root rot, seedling blight	cereals	Blair 1936
Dilophospora alopecuri	Twist	grass crops	Rainio 1936
Erysiphe graminis	Powdery mildew	cereals	Hardison 1944, 1945
Fusarium species	root rot, head blight	cereals	Padwick and Henry 1933
Gaeumannomyces graminis	Take-all	wheat	Kirby 1922
Helicobasidium purpureum	violet root rot	carrots	Tinsley 1980
Helminthosporium giganteum	zonate eye spot	grass crops	Drechsler 1923a
Longidorus elongatus	needle nematode	various crops	Thomas 1969
Low temperature basidiomycete	snow mould	cereals, forage legumes	Cormack 1948
Paranguina agropyri	Stem gall nematode	cereals	Krall and Krall 1970
Physoderma graminis	Brown stripe	grasses	Sparrow and Griffin 1964
Pseudocercosporella herpotrichoides	eye spot	cereals	Cunningham 1965
Puccinia graminis f. sp. secalis	stem rust	rye	Klebahn 1931, Green 1963
Puccinia striiformis	stripe rust	wheat	Popov 1979
Rhizoctonia solani	root rot	various crops	Griesbach 1975
Rhynchosporium secalis	scald	forage grasses	Sprague 1955
Septoria nodorum	leaf blotch	wheat	Ao and Griffithr 1976

TABLE 22 (cont.)

Pathogen	Disease	Crop attacked	Reference
Septoria tritici	Leaf blotch	wheat	Brokenshire 1975
Tilletia controversa	dwarf bunt	wheat	Zakharova 1963
Ustilago spegazzinii	stem smut	forage grasses	Fischer 1945
Wheat streak mosaic virus	wheat streak mosaic	wheat	Slykhuis 1952
Xanthomonas transluscens	bacterial blight	grass crops	Boosalis 1952

Claviceps purpurea (Campbell 1957). These pathogens sporulate vigorously on quack grass and are disseminated readily by wind, rainsplash, or insects.

Quack grass may aid in maintaining pathogen inoculum in the absence of a susceptible crop host, such as overwinter or during a break in the cropping system due to crop rotation. The growing season of quack grass often extends longer than the growing season of the crop. Also, the rhizomes of quack grass may persist throughout the year. Quack grass has been reported to be responsible for maintaining damaging levels of pathogens such as Gaeumannomyces graminis (Kirby 1922, Russel 1930, Padwick 1935), Bipolaris sorokinianaand Fusarium species (Padwick and Henry 1933, Blair 1936) in the absence of a suitable crop host. In these instances, control of quack grass has been advocated for disease control (Russel 1930, Blair 1936, Ogilvie and Thorpe 1962).

The quack grass rhizome system may act as a bridge for spreading of some pathogens. Padwick (1935) reported that G. graminis spread very little in bare soil while, in soil supporting quack grass, the pathogen spread a considerable distance along the rhizome system.

Secretion from dead tissue and actively growing quack grass may be capable of stimulating the growth of pathogens in the soil (Padwick 1935, Kommedahl and Ohman 1960). Leaf and rhizome exudates of quack grass have been associated with an increase in problems associated with seedling blight of cereals and alfalfa, incited by Bipolaris sorokiniana, Fusarium species, and a number of the other soil-borne pathogens (Kommedahl and Ohman 1960).

Quack grass has also been reported to be a collateral host for vectors of plant pathogens, particulary viruses attacking economic crops (Bos 1981). Therefore, these vectors may obtain pathogen inoculum from quack grass and carry it to a crop host. In addition, virus-free quack grass may serve as a host for virus vectors, providing an opportunity for reproduction of the vector. Quack grass may serve to indirectly contribute to an increase in crop disease in this manner (Bos 1981).

The role of quack grass as a site for asexual recombination and hybridization of different isolates of pathogens considered by a

number of authors. Johnson (1947, 1949) reported successful crosses between P. graminis f. sp. tritici and P. graminis f. sp. secalis, both of which occur on quack grass. The hybrid rusts exhibited high fertility and had a wider range of pathogenicity than either of the parental varieties. Green (1971) reported similar results in crosses between formae speciales of P. graminis and Shifman (1958) reported hybridization between P. triticina (P. recondita f. sp. triticina) and P. agropyrina (P. recondita f. sp. agropyrina). Shifman (1958) recommended eradication of quack grass and other wild grasses as a means of limiting hybridization of these rusts. Hardison (1944) acknowledged the possibility of hybrids between different pathogenic races of Erysiphe graminis and Hiura (1965) successfully crossed forma speciales of E. graminis from wheat, barley, rye and Agropyron species. Hiura (1965) concluded that quack grass could serve as a site for hybridization.

It should be noted, however, that in all cases where hybrids were formed between different forms of P. graminis and between different forms of E. graminis, the resulting hybrids were either non-pathogenic or reduced in pathogenicity. It was concluded that the specialized pathogenic forms were prevalent and hybrids which were virulent on both wheat and quack grass rarely survived (Johnson 1949, Hiura 1978). Therefore these hybrids would not be of practical importance (Johnson 1949). This view, however, is not supported by the lack of specialization of E. graminis reported by Hardison (1945). Also, with the rusts, a variable pathogen population on a weed host would provide a source of variation during sexual recombination on the pycnial and aecial host, in addition to recombination during anastomosis during the uredial stage. Therefore it is conceivable that the survival of such a hybrid is possible on either the weed host or the crop host if the appropriate combination occurs.

4.2. Materials and Methods

4.2.1. Field surveys

4.2.1.1. Foliar pathogens

Data collected in field surveys outlined in section 2.2.2. were used to determine the relationship between diseases on quack grass and the level of diseases on grain crops. Due to the non-parametric nature of the data, Spearman rank correlation (Spearman 1904) was used to determine if there was a correlation between 1) the density of quack grass and the level of disease on the grain crop and 2) the disease ratings of quack grass and disease ratings of the grain crop.

4.2.1.2. Root rots

Twenty wheat fields were surveyed for the presence of take-all on the crop and quack grass. Data were analyzed by the Spearman rank correlation test to determine if there was a relationship between quack grass density and percent disease in wheat.

Twenty barley fields were randomly selected to study the influence of quack grass on the incidence of root rot of barley. The field history of each field and crop management practices were obtained from each farmer prior to surveying the field. Density of quack grass was recorded as shoots m^{-2} and root rot of barley was assessed according to the method of Russell and Sallans (1940). One hundred randomly selected barley plants from each field were assessed for root rot and brought back to the laboratory for isolations. Isolations were made from the subcrown internodes of the barley plants.

Quack grass rhizomes were collected from each field for isolation of pathogens. In addition, rhizomes were also collected from five potato fields in which potatoes had been grown for two or more years. Comparisons were made between isolations from the potato and barley fields.

4.2.2. Foliar inoculations

Isolations were made from leaves of wheat, oats and barley showing various disease symptoms. The organisms obtained were used for cross inoculation onto quack grass. The organisms used were Bipolaris sorokiniana, Drechslera biseptata, Pyrenophora japonica, Pyrenophora teres, Rhynchosporium secalis and Septoria nodorum. Cultures were used to inoculate quack grass in the same manner as described in Section 3.2.3.1. Puccinia recondita f. sp. triticina from wheat, P. hordei from barley and P. coronata from oats (f. sp. avenae) and from barley (f. sp. secale?) were also used for cross inoculations and inoculated in the same manner as outlined in Section 3.2.3.2. Wheat, oats, and barley were inoculated with quack grass isolates of these organisms.

Ten plants of each species were grown in a total of five pots each and inoculated with each organism. Plants were grown in growth cabinets and results recorded two weeks after inoculation. Following the development of symptoms, pathogens were re-isolated from the experimental host and reinoculated back onto their original host. Results were recorded one week following the appearance of symptoms as percent leaf area diseased, or, in the case of rust organisms as infection type.

4.2.3. Soil infestation of Bipolaris sorokiniana and Fusarium avenaceum

B. sorokiniana and F. avenaceum were grown in a soil-sand mixture containing cornmeal (40g soil: 5g sand: 5g cornmeal). This mixture was placed in 900 ml mason jars with 15 ml of distilled water and autoclaved at 120°C at 103 kilopascals for 30 minutes. A 0.5 cm^2 block of pathogen culture, grown on potato dextrose agar was placed in each jar. The jars were then incubated at room temperature for two weeks. Controls consisted of 0.5 cm^2 agar blocks placed in mason jars containing the soil:sand:cornmeal mixture. Following incubation, infested soil and control soil were mixed with greenhouse soil (4 parts loam:2 parts sand:½ part peat) and placed in 15 cm diameter pots. Ten seeds each of wheat, oats, barley, rye, quack grass and alfalfa were sown in the pots. Quack grass rhizomes were also sown. The experiment was con-

ducted on a greenhouse bench with four replicates in a completely randomized design. Plants were watered regularly, as required, and results recorded as percent emergence and percent diseased plants three weeks following emergence. Symptoms assessed were seedling blight, leaf spotting, abnormal growth and discoloration of subcrown internodes.

4.2.4. Rhizome transmission

Rhizomes obtained from quack grass grown for five weeks in soil infested with B. sorokiniana and in pasteurized soil were used in this experiment. After harvest, the rhizomes were washed in distilled water and cut into three bud pieces. Ten pieces of rhizome from infested soil were sown in each of eight 15 cm diameter pots containing pasteurized soil. Ten pieces of rhizome from pasteurized soil were similarly sown in eight pots. Ten barley seeds were then sown into four pots containing rhizomes from infested soil and four pots containing rhizomes from pasteurized soil. Four pots of each rhizome treatment were not sown to barley. Controls consisted of barley sown alone in pasteurized soil. Pots were placed on a greenhouse bench and maintained under a 14 hour photoperiod. Each of the five treatments were replicated four times and arranged in a completely randomized design. Plants were allowed to grow until growth stage 10.5.4 (Feekes scale) was attained. Results were recorded as percent emergence, percent diseased leaf area and percent root rot of barley. Isolations were made from the subcrown internodes of barley to determine the presence of B. sorokiniana. Powdery mildew was controlled by application of ethirmol (5-butyl-2-ethylamino-4-hydroxy-6-methyl pyrimidine) at a rate of 1.0 kg active ingredient ha^{-1}.

4.3. Results and Discussion

4.3.1. Field surveys

This research was conducted to determine if there was any relationship between disease levels on quack grass and disease levels on cereal crops. A positive relstionship would suggest a possible role

of quack grass as a secondary host for some of these pathogens which could result in increasing disease levels on crops in Prince Edward Island.

Brown spots or leaf necrosis were common leaf symptoms and could not be attributed to any one pathogen unless isolations were made from each leaf. As a result, disease ratings did not include ratings for individual diseases unless the disease was easily recognized, such as powdery mildew or rust. Also disease ratings were not done on quack grass occurring outside the field. Therefore, results presented here (Table 23) represent mean disease ratings of quack grass within the crop fields.

4.3.1.1. Barley

The overall mean disease rating for all quack grass leaves was 7.6% in 1979 and 11.29% in 1980. Disease ratings of quack grass were less variable than disease rating of the barley indicating a more stable disease level on quack grass. The only pathogens isolated from both barley and quack grass were Bipolaris sorokiniana (Cochliobolus sativus) causing spot blotch and Drechslera tuberosa (Atk.) Shoem (perfect stage: Pyrenophora japonica), causing a leaf spot. Rhynchosporium secalis (Scald), which was common on quack grass along the margins of the fields was never found on quack grass within the field. The most common symptoms were small brown streaks over the surface of the leaves. However, no pathogens were isolated from these lesions. Therefore, these symptoms could not be attributed to any pathogen.

There was a positive correlation between ratings of net blotch, incited by Pyrenophora teres, and quack grass disease ratings. There was no correlation between scald disease ratings of barley and quack grass disease ratings, nor was there any relationship between barley disease ratings and quack grass density. Correlation coefficients and their significance levels can be found in Appendix 2.

The positive correlation between quack grass disease ratings and net blotch disease ratings in both years, indicate that conditions

TABLE 23: Mean disease ratings for quack grass in various cereal crops in 1979 and 1980

Crop	Mean %		Standard error		Range	
	1979	1980	1979	1980	1979	1980
Barley	7.6	11.7	1.0	3.1	1.2 - 32.8	1.7 - 37.6
Oats	13.6	12.6	2.1	2.1	1.2 - 48.2	1.2 - 24.6
Mixed	13.8	6.8	1.9	1.7	1.8 - 30.0	1.3 - 28.0
Wheat	18.4	27.1	2.6	5.7	2.6 - 44.4	1.3 - 65.9

for symptom development were ideal for the agent responsible for the symptoms on both hosts. As previously mentioned, the agent responsible for the symptoms on quack grass could not be identified. However, disease ratings were always noted to rapidly increase as the crop matured and also at a time when disease levels were high on barley and conditions favorable for sporulation of P. teres. This is supported by a positive correlation between the growth stage of the crop and disease ratings of quack grass (r_s = 0.5895, significant at α = 0.035). Therefore, at the time when symptoms rapidly increased on quack grass, the plants would be subjected to a high inoculum level of P. teres. If conditions were particularly favorable for disease development, then there may have been opportunities for a large number of infections to occur on quack grass. This is supported by the fact that quack grass is susceptible to P. teres under experimental conditions even though quack grass is resistant to P. teres under field conditions (Shipton et al. 1973). Also, symptoms induced on quack grass by P. teres under experimental conditions resembled those found in the field. Therefore, it may be hypothesized that the spots noted in the field were a resistant reaction to P. teres, allowing only limited development of the pathogen. The limited development of the pathogen may have prevented its isolation.

There is additional evidence, however, to suggest that these lesions were not related to P. teres and the increase in disease levels in both species was coincidental. As the crop matured, the death and drying of the leaves would allow a greater air circulation through the canopy. Quack grass would, therefore, be less screened from airborne spores of other pathogens. At this time, tissue of quack grass was still green and susceptible to pathogen attack, whereas the crop would not be exposed because of its advanced growth stage.

Therefore, the role of quack grass in the epidemiology of leaf-spotting pathogens of barley is still unclear. B. sorokiniana and D. tuberosa were not isolated often enough to be considered important. If the symptoms noted on quack grass were caused by P. teres, then quack grass may play a role in the development of net blotch. Shipton et al. (1973) reported that P. teres can sporulate on leaves of resistant

hosts. If this is possible and the pathogen can sporulate on leaves of quack grass, then quack grass may influence disease development. However, one factor which may limit such a role is the fact that the pathogen, if it occurs on quack grass, occurs at a time when the crop is mature and the pathogen is no longer important to the crop. Also, the pathogen must be able to survive the winter on quack grass leaves and still be able to sporulate in the spring. Additional research is required to establish if the leasions noted on quack grass in barley were the result of a resistant reaction to P. teres.

4.3.1.2. Oats

Quack grass disease ratings in oats were slightly higher than those recorded on quack grass in barley in 1979 (13.6% as compared to 7.6%). In 1980, mean quack grass disease rating was 12.6%. There was a positive relationship between quack grass disease ratings and growth stage of the crop, indicating an increase in disease ratings as the crop matured. There was a much stronger positive correlation between quack grass disease ratings and septoria disease ratings on oats. Leaf spotting and leaf necorsis symptoms were observed near the tips of quack grass leaves but no pathogens were isolated from these lesions. The most common disease occurring on oats was septoria leaf blotch, incited by Septoria avenae. Since this organism was not isolated from quack grass and even though quack grass has been reported to be a host of this pathogen (Sprague 1956), there is no data to support the conclusion that a relationship exists between the two disease ratings. It appears that there was a coincidental increase of disease. Increase in disease levels later in the growing season is a common occurrence and therefore, it can be assumed that there was no relationship between the disease ratings on quack grass and oats.

4.3.1.3. Mixed grain - oats and barley

In 1979 disease ratings on quack grass in mixed grain were similar to those recorded on quack grass in oats. Mean disease rating was 13.8%. In 1980, the mean quack grass disease rating was 6.8% (Table 23).

In both years, symptoms on quack grass resembled those noted on quack grass in barley and correlations were similar between disease ratings of quack grass and crop disease ratings. As in barley, the symptoms on quack grass may have been due to a resistant reaction to P. teres. It is unlikely that there was a relationship between oat disease ratings and quack grass disease ratings.

4.3.1.4. Wheat

The highest quack grass disease ratings in any crop surveyed were recorded in wheat in both 1979 and 1980 (Table 23). The higher disease ratings were due to extensive leaf necrosis caused by infection of a Septoria species, probably S. nodorum since this pathogen was the most commonly isolated from leaves of wheat. Powdery mildew was also present at high levels on quack grass but on wheat, levels were not high enough to cause serious losses. Therefore, powdery mildew from quack grass was probably did not influence mildew levels on wheat.

There was a positive correlation between septoria leaf blotch ratings on wheat and quack grass disease ratings in 1979, indicating concurrent high disease levels on both wheat and quack grass. Symptoms on quack grass and wheat were similar in appearance. At early stages of infection, the symptoms appeared as diamond-shaped lesions which subsequently expanded to extensive leaf necrosis.

In 1980, unlike the 1979 results, there were no significant correlations between quack grass disease ratings and wheat disease ratings obtained. However, high disease ratings were still recorded on quack grass (27.1%) late in the season. Also there were no significant correlations obtained between the crop disease ratings and quack grass densities, indicating that densities were not high enough to result in increases in disease due to changes in the microclimate of the canopy.

Shearer and Zadocks (1972) and Harrower (1977) reported that S. nodorum may show some degree of specialization toward an alternate host following passage through that host. If such a situation exists in this case, then the organism on quack grass would be expected to be specialized toward quack grass and less virulent on wheat. It should be noted, also, that this organism did not develop to any extent on quack grass unless the pathogen was already established on wheat. It is likely that the wheat provided the source of inoculum for the quack grass. The importance of quack grass as a source of inoculum would, therefore, be reduced, although it could provide a secondary source of inoculum as the season progressed. Quack grass may, however, act as a means by which the organism can overwinter. S. nodorum can survive on wheat stubble and leaf debris (Harrower 1974), and, therefore, quack grass debris may act as a source of inoculum for the next season.

4.3.1.5. Conclusions

No specific conclusions can be drawn from the field surveys for leaf diseases concerning the role of quack grass as a source of inoculum for the specific diseases examined in the field. However, this type of survey does give an indication of what is occurring in the field and also gives an indication of what diseases may be affected by the presence of quack grass. Host specificity tests and cross inoculation studies may provide evidence which may support any theories suggested by field studies.

The only foliar pathogen observed in these studies for which quack grass may play an important role in its epidemiology is S. nodorum. However, other diseases, such as various rusts, were found only on quack grass in non-crop situations. Quack grass may be important in the epidemiology of these pathogens, by virtue of airborne spores which can be carried great distances, as a site for hybridization and as a source of variation of these pathogens.

4.3.2. Root rot

Two root rot diseases may cause serious losses in cereals in Prince Edward Island. Take-all, incited by Gaeumannomyces graminis, may cause serious losses in wheat, particularly where the crop has been grown continuously for two or more years (Clough and Johnston 1978a). Common root rot, incited by Bipolaris sorokiniana, Fusarium species, and possibly a number of other organisms, occurs on all cereals, but causes most serious losses on barley (Clough and Johnston 1978a). Measures commonly used to reduce the levels of this disease, including rotation, sanitation, and other management practices may not always be successful. High disease levels are often recorded in fields following long breaks between successive cereal crops. One of the possible reasons for this is the presence of quack grass in the fields. Quack grass has been reported to increase severity of these diseases on cereals (Padwick 1935; Blair 1936; Kirby 1922). This research was conducted to determine if there was a possible relationship between the presence of quack grass and cereal disease levels.

4.3.2.1. Take-all

Take-all was found in only three spring wheat fields in 1979. Highest percentage take-all recorded was 3%. Therefore there was insufficient information for correlation. Quack grass was found in all fields in which take-all occurred and showed symptoms of the disease. Although there was no apparent damage to the appearance or vigor of quack grass, infected rhizome sections were completely blackened. In contrast, diseased wheat plants exhibited a blackening of the stem base, rotting of the root system, stunting, bleached heads and sterile spikes or spikes containing shrivelled seed.

In 1980, take-all was recorded in 11 of 14 fields surveyed for this disease. The mean disease rating was 12.1%, with the percentages ranging between 1% and 24% in fields in which the disease occurred.

The take-all fungus, a soil-borne pathogen, may occur in patches in a particular field. Diseased wheat was always found in association

with diseased quack grass. Quack grass showing symptoms of take-all was also found in areas where diseased wheat was not present however. It should be noted that quack grass was the only grass species other than wheat found to be infected by G. graminis in the wheat fields surveyed. A positive correlation was found between percentage take-all on wheat and density of quack grass (r_s = 0.8074, significant at α = 0.003). Percent infection by G. graminis on quack grass was not determined because symptoms involved sections of rhizomes up to 25 cm in length and often more than one shoot originated from these sections. No correlation could be done, therefore, between percentage take-all on wheat and percentage take-all on quack grass. Observations from these field studies are supported however, by studies of Kirby (1922), Russell (1930), Padwick and Henry (1933), Ogilvie and Thorpe (1962), and others which indicate that quack grass may play an influential role in the development and persistence of this disease in Prince Edward Island fields.

4.3.2.2. Common root rot

Common root rot is one of the most widespread diseases of barley in Prince Edward Island with yield losses varying from 10-40% of potential yields (Clough and Johnston 1978a). Crop rotation, seed treatment, sanitation and other cultural measures are commonly recommended in attempts to reduce the severity of this disease but are not always successful.

The organisms commonly associated with this disease include Bipolaris sorokiniana and various Fusarium species. Pythium species, Rhizoctonia solani, and others, may also be involved however. Quack grass may serve as a host for all of the organisms. Padwick (1935) and Blair (1936) reported that the presence of quack grass in cereals will result in higher levels of this disease. Ledingham and Chinn (1964), although questioning the role of quack grass in disease problems, also acknowledged that perennial grasses such as quack grass may serve to maintain pathogens in the soil with the exception of Fusarium species. The relationship between Fusarium pathogens and

quack grass is still unknown. This study was, therefore, conducted to determine the possibility of quack grass contributing to the common root rot problem in Prince Edward Island barley fields.

Root rot was present in all fields surveyed. Quack grass was found in all but two fields in 1979 and in all fields surveyed in 1980. No positive relationships could be established between percent root rot and quack grass density. In 1979 the correlation was positive although not significant. In 1980, however, a negative correlation existed which was significant at $\alpha = 0.1$. It is possible that quack grass could be acting as a trap crop for this pathogen, especially if quack grass could not support sporulation of this pathogen as has been suggested by Ledingham and Chinn (1964).

All pathogenic fungi suspected of being involved in root rot that were isolated from both barley and quack grass are listed in Table 24. In some cases, two or more pathogens were isolated from the same piece of plant tissue. With the exception of Rhizoctonia solani and Pythium species, there was very little difference between the percentage isolation of these organisms from quack grass rhizomes or barley roots. Therefore, quack grass could conceivably contribute to inoculum levels of these organisms.

Isolations were made from rhizomes collected from potato fields to determine if quack grass could serve to maintain pathogen inoculum in the soil in the absence of a susceptible crop host. A number of fungi were isolated from rhizomes in both fields. Comparison of the percent isolation of Fusarium species and percent isolation of Bipolaris sorokiniana, the two most common pathogens associated with root rot, from rhizomes collected from barley and potato fields, indicate that there were no differences between percent isolation from both sources (Table 25). Fifty-nine percent of the species isolated from rhizomes collected from barley fields were Fusarium species compared to 56% isolation of Fusarium species from potato fields. The predominant Fusarium species isolated from both collections of rhizomes was F. avenaceum. F. oxysporum, F. sporotrichoides, F. equiseti and F. culmorum were also present however. Percent isolation of B. sorokiniana from quack grass rhizomes was 15% and 13%

TABLE 24: Pathogens isolated from barley roots and quack grass rhizomes collected from barley fields

Pathogen	Percent isolation	
	Quack grass	Barley
Bipolaris sorokiniana	15	13
Drechslera biseptata	12	9
Fusarium species	63	59
Microdochium bolleyi	32	28
Pythium species	15	3
Rhizoctonia solani	12	0

TABLE 25: Isolation of *Bipolaris sorokiniana* and *Fusarium* species from quack grass rhizomes in barley and potato fields

Crop	*Fusarium* species	*Bipolaris sorokiniana*
Barley	59	15
Potato	56	13

from barley and potato fields respectively. Since the potato fields surveyed had not been sown to grain for at least two years, quack grass may serve to maintain the pathogens in the field in the absence of a susceptible cereal host. Potato may serve as a host for Fusarium species (Booth 1971) but it is not known to act as a host for Bipolaris sorokiniana.

4.3.3. Cross inoculation of foliar pathogens isolated from quack grass, wheat, oats and barley

In order for quack grass to serve as a source of inoculum for cereal pathogens, the cereal crops must be susceptible to forms of the pathogens occurring on quack grass and, in turn, quack grass must be susceptible to isolates of those pathogens occurring on cereal crops. Therefore quack grass was inoculated with cereal isolates and wheat, oats, and barley were inoculated with quack grass isolates of those pathogens, if available.

4.3.3.1. Puccinia species

Results of inoculations of Puccinia species, indicate that, with the exception of P. coronata isolated from quack grass and barley, Puccinia species were restricted to their original host (Table 26). Therefore, quack grass probably does not serve as a source of inoculum for these rusts. Quack grass is not susceptible to the races of these pathogens occurring on the cereals nor is wheat susceptible to the races of P. graminis and P. recondita occurring on quack grass. Oats is not susceptible to the form of P. coronata (P. coronata f. sp. secalis) occurring on quack grass. A different situation exists between P. coronata from barley and quack grass. In cross inoculations, barley and quack grass were susceptible to P. coronata from both barley and quack grass. Therefore, quack grass may provide inoculum for development of P. coronata on barley, providing conditions are favorable.

TABLE 26: Cross inoculation of *Puccinia* species obtained from quack grass and cereal crops

Pathogen	Source	Host reaction[1] Wheat	Oats	Barley	Quack grass
P. coronata	quack grass	0;	0;	3	4
	oats	0	3	0	0
	barley	0	0	4	4
P. hordei	barley	0	0	4	0
P. graminis	quack grass	0	0	0	2
	wheat	3	0	0	0
P. recondita	quack grass	0;	0	0	4
	wheat	3	0	0;	0;

[1]
- 0 – no visible reaction
- 0; – hypersensitive reaction but no uredia
- 1 – minute uredia surrounded by a distinct necrotic area
- 2 – small to medium uredia usually surrounded by a necrotic or chlorotic area
- 3 – medium-sized uredia, no necrosis but chlorosis, coalescence of uredia infrequent
- 4 – large, often coalescing uredia, no necrosis but chlorosis may be present

0, 0;, 1, 2 – resistant reaction

3, 4 – susceptible reaction

4.3.3.2. Foliar pathogens other than Puccinia species

Results of cross inoculation with B. sorokiniana demonstrated that the isolates from barley and quack grass did not differ in pathogenicity and attacked all plants tested (Table 27). Barley and quack grass were most susceptible to both isolates while wheat was moderately susceptible and oats showed only limited development of the pathogen. Therefore, in a situation in which this fungus can sporulate on leaf surfaces, B. sorokiniana may be able to pass freely from barley to quack grass and vice versa. The same may apply to quack grass and wheat. This is also a root infecting pathogen and it is probable that inoculum originating from roots and rhizomes can also infect both quack grass and cereals.

A similar situation may exist with Drechslera biseptata. However, disease ratings were significantly less than those obtained with B. sorokiniana. D. biseptata was recorded only on the roots and rhizomes of barley and quack grass and never from leaves of either species and it may be that this is only a root or rhizome pathogen. However, in a situation where the pathogen can sporulate on plant surfaces, it is likely that spores originating from quack grass can infect barley, wheat or any other susceptible grass and vice versa. Very little is known about this organism, concerning its pathogenicity, saprophytic ability, method of infection, or ability to sporulate on plant surfaces. Also, since this is the first report of this pathogen in Canada, there is no information on its importance as a pathogen or its distribution. Until such information is known, no conclusions can be drawn on the role played by quack grass in the epidemiology of this pathogen.

Pyrenophora japonica was a pathogen recorded on quack grass and possibly barley but identity of the organism on barley has not been confirmed. However, in host specificity tests, this pathogen attacked a number of species of grasses, including wheat and barley. The same applied to P. teres except that this pathogen was only isolated from barley. These two organisms are morphologically similar and can be easily confused and atypical symptoms

TABLE 27: Cross inoculation of foliar pathogens (other than Puccinia species) isolated from quack grass, wheat and barley

Pathogen	Source	Disease reaction[1] Wheat	Oats	Barley	Quack grass
B. sorokiniana	quack grass	31.4	7.1	75.3	82.6
	barley	23.7	6.2	81.3	80.5
Drechslera biseptata	quack grass	3.2	1.2	11.8	18.8
	barley	4.8	1.2	16.2	16.3
Pyrenophora japonica	quack grass	2.3	1.2	67.9	79.3
P. teres	barley	3.2	2.5	59.7	50.7
Rhynchosporium secalis	quack grass	0	0	0	26.5
	barley	0	0	34.5	0
Septoria nodorum	wheat	62.4	0	0	28.8

[1] percent leaf area diseased

recorded on barley attributed to P. teres (Clough and Sanderson 1979) are similar to symptoms of P. japonica recorded on barley (Shoemaker 1962). This suggests that P. japonica may be present in barley on Prince Edward Island, but has been misidentified as P. teres. Therefore, cross inoculation studies were conducted with these two organisms. Results for the two pathogens were similar on all crops except that percent area diseased was higher with P. japonica. Disease ratings were low on wheat and oats with both organisms. Therefore, based on experimental evidence these two organisms can probably pass from barley to quack grass and vice versa provided conditions allow for it and these hosts are susceptible to these pathogens under field conditions. Also, the importance of P. japonica as a pathogen in Prince Edward Island is still unknown. Therefore further conclusions cannot be made until further information is obtained.

Results of inoculations with Rhynchosporium secalis indicate that this pathogen was restricted to its original hosts. The isolate from barley did not attack quack grass nor did the isolate from quack grass attack barley. Therefore, assuming that conditions for infection were the same for both pathogens on both hosts, it is unlikely that these pathogens can pass from one host to the other in the field. The absence of quack grass infected with R. secalis in barley fields heavily infested with R. secalis supports this conclusion. However, the assumption of similar conditions for infection by both isolates may be premature, as pointed out by Ali (1972) and Ali and Boyd (1974).

An isolate of Septoria nodorum from wheat was used for cross inoculation studies. Wheat and quack grass were both susceptible to this isolate, although, percent leaf area diseased was significantly less on quack grass. The susceptibility of quack grass to S. nodorum from wheat provides evidence that quack grass may play a role in the epidemiology of this pathogen. Field surveys indicated that this role may be as an overwintering host, providing an initial source of primary inoculum in addition to wheat debris.

4.3.4. Soil infestation with Bipolaris sorokiniana and Fusarium avenaceum

Barley and quack grass were grown in soil infested with B. sorokiniana and F. avenaceum to determine the pathogenicity of these two organisms. In addition, wheat, oats, rye, and alfalfa were also sown in infested soil to determine the pathogenicity of these organisms on these crops (Table 28). This was necessary to establish the susceptibility of the crops in question to those organisms isolated from quack grass.

Results indicated that F. avenaceum was very pathogenic on all hosts, reducing germination in all crops. Wheat appeared to be the most susceptible to this pathogen, with 27.6% germination. Alfalfa was also very susceptible with 38.7% germination. Alfalfa was the only crop plant which had post-emergence disease symptoms, expressed as damping-off. F. avenaceum was also isolated from all plants with damping-off. Rye appeared to be the most tolerant to this pathogen, showing 74.5% germination. Quack grass originating from rhizomes appeared to be more tolerant to F. avenaceum than quack grass originating from seed. Percent emergence of quack grass from rhizomes was 67.9% compared to 49.0% from seed. This is probably due to the fact that plants originating from rhizomes were larger and much more vigorous than plants grown from seed and better able to compensate for the effect of the pathogen.

All species were also susceptible to B. sorokiniana. Symptoms of infection by this pathogen were expressed as reduced emergence, deformed plants, and dark brown lesions on the leaves and stem base. Infected alfalfa plants also exhibited damping-off. This pathogen did not significantly reduce the percent emergence of barley when compared to controls. Percent germination of other crops was reduced however. Wheat was particularly susceptible, showing only 13.6% emergence. Rye and alfalfa appeared to be the most tolerant showing 81.7% and 87.8% emergence respectively. The number of emerged plants showing symptoms was 3.2% and 4.7% respectively. Although percent emergence of oats was significantly reduced (47.5%), none of the emerged plants showed disease symptoms. All of these species have been reported to be hosts

TABLE 28: Effect of *Fusarium avenaceum* and *Bipolaris sorokiniana* on quack grass, barley, oats, wheat, rye and alfalfa[1]

	F. avenaceum		*B. sorokiniana*	
	% emergence	% diseased	% emergence	% diseased
Barley	50.0	0	98.0	72
Quack grass (seed)	49.0	0	53.2	61.0
Quack grass (rhizome)	67.9	0	81.5	69.7
Oats	49.4	0	47.5	0
Wheat	27.6	0	13.6	46.5
Rye	74.5	0	81.7	3.2
Alfalfa	38.7	15	87.8	4.7

[1] Values expressed as percent of controls

of this pathogen (Connors 1967, Kommendahl and Ohman 1960).

Quack grass originating from seed was more susceptible to B. sorokiniana than plants originating from rhizomes. This was probably due to the more vigorous growth of the plants originating from rhizomes. B. sorokiniana was re-isolated from all diseased plants.

Results presented here indicate that the crops examined are susceptible to quack grass isolates of these pathogens. It is suggested, therefore, that quack grass can serve to maintain these pathogens in the soil and increase disease problems in cereal crops, provided that sufficient inoculum is produced. Results also indicate that these organisms and possibly other soil-borne pathogens may play a role in plant population regulation by reducing plant number, and preventing emergence of rhizomes, buds, and seeds. This effect is greater with seeds and may, therefore, reduce the importance of seed in the spread of quack grass, particularly in grain fields.

4.3.5. Rhizome transmission of Bipolaris sorokiniana

Quack grass produces an extensive underground rhizome system which would provide an ideal way for pathogens to be spread in the soil. The ability of a pathogen to pass from the rhizome system to a crop plant would increase the importance of quack grass in the epidemiology of the pathogen. Therefore, this study was undertaken to determine the possibility of B. sorokiniana passing from infected rhizomes to barley plants.

Fusarium species were not used in this study because they were always isolated from rhizomes of quack grass, whether inoculated or not. To prove that these were the original pathogens used for inoculation would be very difficult.

Common root rot was recorded on barley in all treatments including the control (Table 29). However, root rot ratings were higher in those treatments in which quack grass was included. The same applied to the development of leaf symptoms on barley. There was a significant difference between the barley control and the two treatments with quack grass in them. There was, however, no significant difference between the

TABLE 29: Transmission of Bipolaris sorokiniana from quack grass to barley

Treatment	% root rot[1]	Leaf symptoms[2] quack grass	barley	B. sorokiniana[3] isolated
barley control	10	-	1	+
quack grass control	-	0	-	-
barley+quack grass control	16	0	3	+
infected quack grass	-	39	0	+
barley+infected quack grass	22	47	12	+

1 % root rot refers only to disease on barley

2 expressed as percent of plants showing symptoms

3 + pathogen isolated
- no pathogen isolated

barley + quack grass control and barley + infected quack grass treatment. Therefore, based on this, it cannot be concluded that B. sorokiniana has moved from quack grass to infect the barley. There was, however, a significant difference between the barley + quack grass control and barley + infected quack grass treatments in reference to the development of leaf symptoms. Since there was no difference in these two treatments in reference to rot, it may be assumed that the increase in leaf symptoms was coincidence and not related to the presence of B. sorokiniana on quack grass rhizomes. This would agree with observations of Ledingham and Chinn (1964) who reported that this grass would not support sporulation of B. sorokiniana at sufficient levels to result in an increase in disease. However, their work did not take into account sporulation from decaying rhizomes and this experiment was not conducted over a long enough period for decaying of rhizomes to occur. Under these conditions, sporulation may be greater than from healthy tissue. Therefore, additional studies along these lines may be required.

The increase in disease ratings on barley in the pots containing quack grass raises another possibility of quack grass influencing disease levels. Kommedahl and Ohman (1960) reported that exudates from quack grass appeared to increase damage levels from B. sorokiniana and other root pathogens which may be the reason for the increase of disease in barley growing with quack grass in this experiment. The plants grew together in pots for approximately two months and it is conceivable that exudates could have accumulated to amounts to influence pathogen damage in the confines of the pot during this period. However, it would have to be proven that the exudates were positively responsible for the damage. Also, this cannot be applied to what is occurring in the field as the environment and conditions in the field are different from what is found in the confines of a pot, and factors to encourage accumulation of such exudates in the pots may be totally lacking in the field.

4.3.6. Conclusion

It appears that quack grass may play a role in the epidemiology of a number of cereal pathogens on Prince Edward Island. However, with the exception of Gaeumannomyces graminis, the cause of take-all, it appears that quack grass does not influence disease levels in a way that would increase disease problems of any of the pathogens. Quack grass may contribute sufficient inoculum to P. coronata f. sp. secalis to cause increased disease problems from crown rust, but, generally, crown rust only occurs late in the season and does not cause much damage. Therefore, the role played by quack grass to increase inoculum is of a lesser importance.

Quack grass may play a role in maintenance of a pathogen in a particular field, as is the case with Bipolaris sorokiniana or it may also provide a source of primary inoculum in the spring for pathogens such as Septoria nodorum and Claviceps purpurea. In such cases, the pathogen must be able to survive the winter on debris and still be able to sporulate when conditions become favorable. The specificity of organisms such as Rhynchosporium secalis, Puccinia graminis and P. recondita would discount the role of quack grass as a source of inoculum for these pathogens.

One area that has not been investigated in this study is the ability of quack grass to serve as a host where asexual recombination between different varieties or forms of a pathogen may occur. This would involve intensive studies to identify the different forms occurring on quack grass and then to determine if they may hybridize. Although the importance of such a phenomenon has been questioned by previous workers (Johnson 1949, Hiura 1962), such information would provide important data on the epidemiology of various pathogens and also provide some indication of the reasons for lack of specialization for forms of pathogens such as P. graminis or P. recondita.

V. GENERAL DISCUSSION

5.1. Quack Grass Pathogens Integrated into a Control Program

Plant pathogens have rarely been included as an important factor in an undisturbed ecosystem. In agroecosystems, the emphasis has been placed on the study of the etiology, epidemiology and control of pathogens attacking economic crops. Therefore, the study of pathogens of weeds within agroecosystems has been largely neglected. Plant pathogens, however, may exert strong selective pressures on plant populations (Harper 1977). The greatest forces are generally found in long-lived, relatively stable communities and the weakest pathogen pressures in transient weedy communities (Barrett 1982). Pathogens attack weedy species in both disturbed and undisturbed habitats. Understanding the mechanisms involved in selective pressures in stable plant communities could lead to possible adaptation of some of those mechanisms in agroecosystems.

Related studies in the past have brought about significant advances in the area of biological control (Cullen et al. 1973, Daniel et al. 1973). There have been no previous studies examining the pathogens associated with Agropyron repens (quack grass). This weed commonly occurs in both agricultural and undisturbed areas in eastern Canada and, as this study has shown, is attacked by over 30 pathogens. These pathogens may represent a regulatory mechanism influencing the size of the weed population and/or the competitive ability of the weed. Mortimer et al. (1980) maintain that successful control of this species may only be achieved by a thorough understanding of factors which may regulate plant populations, in addition to the whole life cycle of the species and its population.

In considering a biological control program, or any control program for that matter, the crop must be considered as a system in which the weed, crop and pathogen all interact to influence one another. In a cereal system, the close botanical relationship between the crops and quack grass could seriously limit the use of such a program involving pathogens, for control. This limitation may not be present in non-cereal crops.

The specificity exhibited by Puccinia recondita var. agropyri, Rhynchosporium secalis and Urocystis agropyri in these studies suggests that organisms may exist which could be utilized, to some extent, in a control program. Some pathogens which may be specific to quack grass may not have been tested or were overlooked.

P. recondita var. agropyri, R. secalis and U. agropyri require further study to determine their potential as biological control agents. There are some limitations in the use of these pathogens in a biological control program. Forms of these pathogens have been recorded on hosts other than quack grass. In addition, there is a close relationship between these and other forms of the same species attacking economic crops. Although P. recondita var. agropyri is considered to be specific (Samborski, personal communication), the reported specificity of R. secalis has been questioned on the basis of failure to recognize proper environmental conditions (Ali 1972, Ali and Boyd 1974). No host specific forms of U. agropyri from quack grass have been previously reported. An additional limitation to U. agropyri would be its dissemination. This organism is soil or seed borne (Griffiths 1924) and on quack grass, the disease occurs in patches which do not appear to spread much during the season. Percent infection of quack grass was also low (less than 25%) under experimental conditions. Additional information is required concerning conditions for optimum infection in a field environment.

Nevertheless, these pathogens have been shown to be host specific in this study. Manipulation of pathogen populations on quack grass may provide an additional control measure for quack grass. P. recondita appears to be more suited to an augmentation type program in which inoculum levels would be increased at an appropriate time in the quack grass life cycle. However, more intensive work on host specialization is required. U. agropyri appears to be suited to an approach in which the pathogen is introduced into a field where it did not occur before. This pathogen does not disseminate as easily as P. recondita and therefore, it would not be able to spread out as easily.

R. secalis appears to be well adapted to a mycoherbicide approach in which the fungus would be applied in a similar manner as would be a herbicide (Templeton and Smith 1977, Templeton et al. 1979, Templeton

1982). R. secalis is an indigenous pathogen occurring in undisturbed areas and can be grown and induced to sporulate in artificial culture. In addition, the pathogen is disseminated by rainsplash and not easily spread from one field to the next (Evans 1969). These are characteristics desired in a mycoherbicide (Templeton 1982). In addition to intensive host specificity tests, studies of the damage done by the pathogen, environmental conditions required for infection, longevity of the pathogen in the field, and artificial culturing of the pathogen are required to assess the use of R. secalis in such a program, particularly in a non-grass crop situation.

The concept of host range is essential to understanding the role pathogens may play in a weed control program. Therefore this is one area which requires intensive study. When dealing with wild plants, the hosts are a variable population in comparison to genetically uniform crops. Therefore, considering a species as a host does not necessarily mean that all individuals of these species will actually be suitable hosts for a particular pathogen (Dinoor 1974). Fischer (1935) noted marked intraspecific reactional differences for different collections of wild grass species when inoculated with various collections of Puccinia recondita. A similar situation may exist with other pathogens. Ali (1972), Ali and Boyd (1974) and (Kline 1960) have reported both inter- and intra-variability in both isolate pathogenicity and host reaction of Rhynchosporium secalis.

Some pathogens may be continually undergoing selection for maximum aggressiveness (Habgood 1973). This demonstrates the need for a population approach and it becomes necessary to consider host and pathogen populations as composed of variable individuals. Therefore, testing a number of different isolates of a pathogen on a number of different collections of the host should be considered. Within a pathogen population which does not exhibit strict specialization as a population, there may exist host forms worthy of further investigation. These studies may also provide a more accurate indication of the damage to the population which can be expected from the pathogen in the field.

Environmental conditions also have a strong influence upon specificity, particularly in artificial culture. Each pathogen has its range of temperature, humidity, light and nutrition required for the

disease cycle and life cycle. This may also apply to specific forms of a pathogen, as has been suggested for R. secalis (Schein 1958, Ali and Boyd 1974). Also, many fungi kept in culture may mutate with these mutants produced varying in virulence or required environmental conditions. Studies of specificity under experimental conditions, therefore, may not be representative of what happens in nature. It may be subsequently necessary to conduct host specificity tests in the field or under conditions more representative of those to which the pathogen would be exposed in the field. Quarantine procedures must be considered before such a study could begin when evaluating the possible use of exotic pathogens imported for use in a biological control program.

5.2. Quack grass as a source of inoculum for cereal pathogens

This study also considered the role that quack grass may play as a source of inoculum for cereal pathogens on Prince Edward Island. In considering a cereal field as a system, it becomes necessary to examine the interaction between pathogens, crops and weeds. Perennial grass weeds, such as quack grass, may be of particular interest as they may be host to a number of cereal pathogens.

Field surveys of both crops and quack grass provided preliminary data indicating which pathogens may be involved in this interaction. Field surveys of the crops indicated which diseases were most common on the crops concerned. The role of quack grass as a source of inoculum would be lessened for those diseases which are uncommon. Surveys indicated that, in barley, root rot was the most common and most likely, the most damaging disease of those found. Quack grass can serve as a host for the pathogens which may incite this disease, particularly Bipolaris sorokiniana and various Fusarium species. Cross inoculation of isolates of these pathogens from quack grass and barley demonstrated that the isolates from both species were pathogenic on these two hosts. The involvement of Drechslera biseptata in root rot is still unknown, but both barley and quack grass were susceptible and therefore, an interaction between the two hosts may occur, if conditions permit.

Sporulation by this pathogen from crowns and rhizomes, particularly decaying and dead plant tissue, is suspected, but not confirmed. While sporulation may occur from decaying and dead leaf tissue, the importance of this source of inoculum would be dependent upon the frequency of occurrence of the pathogen on quack grass leaves.

Therefore, the ability of quack grass to increase root rot levels in cereal fields is still unknown. Field surveys did not indicate that an increase actually occurred. Quack grass may be more important in the maintenance of inoculum levels in the soil. Isolations from rhizomes collected from potato fields indicated that the pathogens involved with root rot occur on quack grass in the absence of a cereal crop. Chinn (1976, 1977), has shown that spore numbers as low as 27 conidia of B. sorokiniana per gram of soil exceeded the threshold level for infection and can perpetuate the disease. No information is available concerning threshold levels of Fusarium. It is reasonable to assume however, that the population of the pathogen on the underground portions of quack grass can sporulate sufficiently to perpetuate the disease.

Quack grass may play a significant role in the epidemiology of four pathogens of wheat: B. sorokiniana, Gaeumannomyces graminis, Septoria nodorum and Fusarium species. The same situation applies to root rot in wheat, incited by B. sorokiniana and Fusarium, as that which occurs in barley. Field surveys indicated that there is a direct relationship between quack grass and take-all (incited by G. graminis), levels. Although this disease does occur in wheat fields which are quack grass free, higher levels of the disease were found in fields in which quack grass occurred. In all cases in infested fields, quack grass was also infected by G. graminis. This agrees with the findings of Kirby (1922), Russell (1930), Ogilvie and Thorpe (1962), and others. It is suggested, therefore, that control of quack grass in wheat fields in which this disease occurs should reduce disease levels and eliminate a host on which the pathogen can survive and be maintained in the absence of wheat.

Fusarium head blight is a disease which has been causing serious losses in wheat and other cereals in recent years on Prince Edward Island. A number of Fusarium species have been implicated in this

disease, all of which have been recorded on quack grass on Prince Edward Island during this survey. Fusarium species were the most common fungi isolated from seed, leaves, roots, and rhizomes of quack grass. As such, quack grass is most likely contributing high levels of inoculum to the soil and for rainsplash or wind dispersal. A control program aimed at reducing damage from fusarium head blight should include control of quack grass.

Septoria nodorum is the most serious leaf spotting pathogen of wheat on Prince Edward Island. Quack grass is also susceptible to this pathogen. In 1979, strong positive correlations between quack grass disease ratings and septoria ratings suggested a relationship between the disease on the two hosts. This correlation was not found in 1980 but the causal organism was present on quack grass. Cross inoculation studies with isolates from quack grass and wheat added support to the hypothesis that quack grass may serve as a source of inoculum for this pathogen on wheat. Under field conditions, however, the septoria on quack grass always appeared at the same time or later than it was found on wheat. This would suggest that wheat is probably serving as the source of inoculum for quack grass. Quack grass may, however, provide secondary inoculum or serve as a source of ascospores for the next season.

Quack grass did not appear to influence diseases occurring on oats. The disease which causes the most damage on oats on Prince Edward Island is septoria leaf blotch, incited by S. avenae. Although quack grass has been reported to be a host of this pathogen (Sprague 1956), it has not been recorded on quack grass on Prince Edward Island.

The only diseases on rye which may be influenced by the presence of quack grass are ergot, incited by Claviceps purpurea and take-all, incited by Gaeumannomyces graminis. The situation concerning take-all in rye is probably similar to that which occurs in wheat with quack grass playing a role in increasing disease levels and in overwintering of the pathogen. Although no studies were conducted with C. purpurea it would appear that the role of quack grass in the epidemiology of this pathogen on rye is minimal. This pathogen only infects the host when the florets are at anthesis or when the stigmas are exerted from

the florets (Mantle et al. 1977). On Prince Edward Island anthesis of rye occurs prior to flowering of quack grass and thus rye would already be infected, if at all, by the time quack grass would be receptive to the pathogen. Since rye is so commonly infected by this pathogen in this area, a large number of sclerotia are produced and returned to the soil. These sclerotia would probably be sufficient to provide high levels of inoculum the next season, even in the absence of sclerotia from quack grass.

Quack grass may play a role in the epidemiology of many plant pathogens on Prince Edward Island, but it would be premature to conclude that quack grass serves to maintain or increase inoculum levels of pathogens of economic crops simply because the pathogen attacks quack grass, however. This will depend upon factors concerning the pathogen, host, and the environment. The pathogenicity and method of dissemination of the pathogen are also important considerations. The pathogen involved must be able to develop and produce inoculum on quack grass that can be easily disseminated. In addition, pathogens in question must be able to infect the cereal host. Variation in the virulence of the pathogen on the two hosts in question would reduce the importance of quack grass as a source of inoculum. The ability of the pathogen to survive on debris from one season to the next or the ability of the pathogen to grow along the underground system of the weed in addition to its rate of spread are also important factors to be considered.

Characteristics associated with the host which must also be considered include crop vulnerability, sensitivity of the weed host, proper synchronization of the life cycle of the weed, crop and pathogen, density of the weed host and distance from the sensitive crop. Growing conditions also can seriously affect the development of disease on the crop and weed. Therefore, the subsequent passage of the pathogen from the weed to the crop and vice versa may be affected. Finally, the contribution of the crop to inoculum levels must also be considered. If the crop is contributing sufficient levels of inoculum to cause economically damaging disease levels on the crop then the importance of inoculum from quack grass may be minimal.

In this study, many of these factors were not considered. Only the pathogenicity of the pathogens on their hosts, sensitivity of the hosts, weed density, and, in the case of Bipolaris sorokiniana, the ability of the pathogen to spread from rhizomes were examined. Therefore, these results must be considered preliminary. More intensive studies are required which take the above mentioned factors into consideration, to prove conclusively the role of quack grass in the epidemiology of pathogens of cereals on Prince Edward Island and elsewhere.

VI. CONCLUSIONS

Results have shown that quack grass may be attacked by as many as of 30 pathogens in eastern Canada Pathogens such as Erysiphe graminis, Fusarium species, Puccinia coronata f sp secalis, P. graminis, P recondita and Rhynchosporium secalis appear to be causing serious damage to this species and therefore may be involved in population regulation of quack grass

Host specificity studies have indicated that Puccinia recondita, Rhynchosporium secalis and Urocystis agropyri are specific to quack grass and, therefore, require future research to determine if they may have potential use in a biological control program for quack grass. Puccinia graminis also showed some degree of specialization and could perhaps be included in any future studies for this purpose Concern is expressed about the effect of environmental conditions on such tests and also the existence of variability in the host and pathogen. Therefore, additional research is recommended to evaluate the influence which these factors may have on disease development. Studies should also evaluate the potential of integrating disease occurrence on quack grass with other control measures Such studies would assess the effectiveness of control measures applied at the time of greatest damage to quack grass from disease.

Quack grass may serve as a host for a number of pathogens occurring on cereals in Prince Edward Island and may, therefore, contribute inoculum for many of these pathogens. In particular, the presence of quack grass may influence incidence of common root rot and fusarium head blight of cereals, take-all of wheat and rye and septoria leaf blotch of wheat. These results are considered preliminary, however, and additional studies are required to evaluate, in more detail, the extent of the influence which the presence of quack grass may have on disease development in cereal crops.

It is also suggested that research be continued to assess the role of the pathogens in population regulation of quack grass. While knowledge of the pathogens on individual crops and on the biology of quack grass is well documented, little is known about the complex of

pathogens associated with quack grass or their interaction with other crops and crop-associated pathogens or with other organisms in an agroecosystem. A more complete understanding of quack grass-pathogen interactions and how they function in the natural populations of quack grass may provide knowledge that would be readily applicable to the agroecosystem and could subsequently be incorporated into an improved pest management system for both the weed and crop pathogens

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APPENDIX 1

QUESTIONNAIRE FOR CEREAL DISEASE SURVEY - 1979 - 1980

NAME: _____

ADDRESS: _____

PHONE: _____

FIELD LOCATION: _____

SEED VARIETY AND SOURCE 1979-1980: _____

PREVIOUS ROTATION:

1979: _____

1978: _____

1977: _____

1976: _____

1975: _____

SEEDING RATE: _____

SEEDING DATE: _____

FERTILIZER, LIME AND DATE: _____

MANURE APPLIED AND DATE: _____

CROP DENSITY: _____

WEEDS: _____

HERBICIDES: _____

FUNGICIDES: _____

NEMATICIDES: _____

APPROXIMATE AREA OF FIELD SURVEYED: _____

ANY SPECIAL CROP MANAGEMENT TECHNIQUES? _____

_____

COMMENTS? _____

_____

APPENDIX 2

Appendix 2. Spearman rank correlation coefficients for disease surveys of quack grass and cereal crops

A. Barley

1979

	Quack grass disease rating	Quack grass density
Net blotch	0.5264	0.0578
	sig. 0.0010	sig. 0.5760
Scald	0.1560	0.1541
	sig. 0.1310	sig. 0.1360
Root rot	-	0.1500
		sig. 0.5280
Quack grass disease rating	1.0000	0.2504
	sig. 0.0000	sig. 0.0970

1980

	Quack grass disease rating	Quack grass density
Net blotch	0.7243	0.2334
	sig. 0.0051	sig. 0.4220
Scald	0.2229	-0.1872
	sig. 0.4641	sig. 0.5216
Septoria leaf blotch	-0.1398	-0.2669
	sig. 0.6488	sig. 0.3564
Root rot	-	-0.3795
		sig. 0.0969
Quack grass disease rating	1.0000	0.2857
	sig. 0.0000	sig. 0.3440

B. Oats

1979

	Quack grass disease rating	Quack grass density
Septoria leaf blotch	0.8196	-0.1454
	sig. 0.0010	sig. 0.4790
Quack grass disease rating	1.0000	-0.0223
	sig. 0.0000	sig. 0.9140

1980

	Quack grass disease rating	Quack grass density
Septoria leaf blotch	0.6703	0.1364
	sig. 0.0087	sig. 0.6419
Quack grass disease rating	1.0000	-0.18042
	sig. 0.0000	sig. 0.5371

C. Wheat

1979

	Quack grass disease rating	Quack grass density
Septoria leaf blotch	0.6098	0.7214
	sig. 0.0050	sig. 0.0041
Powdery mildew	-0.1726	0.0653
	sig. 0.7756	sig. 0.9214

1980

	Quack grass disease rating	Quack grass density
Septoria leaf blotch	0.4134	0.5517
	sig. 0.2351	sig. 0.0785
Powdery mildew	0.0519	-0.0136
	sig. 0.8868	sig. 0.9623
Fusarium head blight	0.3209	0.0850l
	sig. 0.3659	sig. 0.8037
Take-all	0.6727	0.8074
	sig. 0.0330	sig. 0.0027
Quack grass disease rating	1.0000	0.6866
	sig. 0.0000	sig. 0.0382

D. Mixed grain

1979

	Quack grass disease rating	Quack grass density
Net blotch (barley)	0.8462	0.0070
	sig. 0.0005	sig. 0.9828
Septoria leaf blotch (oats)	0.8913	-0.2642
	sig. 0.0001	sig. .2605
Quack grass disease rating	1.0000	-0.15570
	sig. 0.0000	sig. 0.5122

1980

	Quack grass disease rating	Quack grass density
Net blotch (barley)	0.7709	0.3686
	sig. 0.0001	sig. 0.1098
Scald (barley)	0.6109	0.4260
	sig. 0.0004	sig. 0.0611
Septoria leaf blotch (barley)	0.3456	0.0026
	sig. 0.1356	sig. 0.9915
Septoria leaf blotch (oats)	0.7946	0.1060
	sig. 0.0001	sig. 0.6657
Quack grass disease rating	1.0000	0.4066
	sig. 0.0000	sig. 0.0752