Title	Management of Take-all Patch in High pH Soils
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Executive Summary

Take-all patch (TAP) is a fungal disease of creeping bentgrass (*Agrostis stolinifera* L.) most commonly caused by the fungus *Gaeumannomyces graminis* var. *avenae (Gga)* Saccardo. Between 2012 and 2014, 44% of diseased turf samples analyzed by the Guelph Turfgrass Institute (GTI) Diagnostic Laboratory at the University of Guelph were diagnosed with TAP, 47% of which were from Ontario. Developing management strategies to reduce TAP severity would be beneficial to the turf industry.

This disease is often not considered important on many golf courses as it is usually associated with new or newly renovated sites. In fact, in many agricultural and turfgrass systems, a natural decline in disease severity (called Take-all decline) is observed over time and is believed to be due to soil suppression. However, take-all decline has yet to be observed on southwestern Ontario golf courses. The disease is also often present on much older greens in the provinces of Alberta, Saskatchewan, and Manitoba. The absence of decline phenomena may be due to a variety of reasons, including: 1) Ontario turf TAP symptoms are not caused by *Gga* and 2) soil conditions (specifically high pH) are not conducive to the development of suppressive soils.

This study was conducted to confirm the causal agent of TAP in Ontario and to investigate alternative methods of disease management through cultural practices. Results to date suggest that we are dealing with a different pathogen in much of Canada than Gga, and we are in the process of confirmation that the most likely causal agent is actually *G. cylindrosporus*. Results also suggest that many of the previously researched cultural methods to reduce disease development (e.g. use of ammonium sulfate or manganese sulfate) had little to no effect on inoculum levels, but that acidifying the irrigation water may be a way to reduce disease over time. These results may be due to the fact that we appear to be managing a causal agent different from the one identified in areas where previous research was conducted, and this could be why we are not able to replicate those research results. We are currently working to confirm our identification results and focus on methods that appear to have the most promise for the pathogen present in Canada.

Background

Creeping bentgrass (*Agrostis stolinifera* L.) is considered to be an optimal turfgrass for golf course greens in cool-season environments (Lawson *et al*., 2012). Healthy stands of creeping bentgrass spread vigorously, tolerate low cutting heights, and recover quickly from damage (Lawson *et al.*, 2012). The uniformity of shoot and leaf density makes creeping bentgrass an ideal rolling surface for golf and lawn bowling (Croce *et al.,* 1995). The presence of creeping bentgrass prevents the growth of weeds, including weaker grass species that tend to require higher maintenance and nutrient inputs (Smiley *et al.,* 2005).

Creeping bentgrass is susceptible to the fungal pathogen *Gaeumannomyces graminis* var *avenae* (*Gga*), a known causal agent of take-all patch (TAP). *Gga* is a destructive pathogen capable of killing healthy stands of high quality turf (Clarke and Gould, 1993; Smiley *et al*., 2005). Although take-all patch is a serious disease of creeping bentgrass, the occurrence of this disease often wanes over time. Under acidic soil conditions, a phenomenon known as take-all decline has been observed (Cook, 1981; Bockus and Tisserat, 2000), whereby the presence of the pathogen will decline over five to seven years following its appearance (Smith, 1957; Clarke and Gould, 1993). Soil pH is typically variable in Ontario (Lauzon *et al.*, 2005). Severe *Gga* infections reportedly occur in soils with pH above 7.0 in the first 2.5 cm of the soil profile (Smith, 1957; Clarke and Gould, 1993), thus it is possible that this disease may be mediated through the application of acidifying soil amendments.

The purpose of this investigation is to study the management of take-all patch by confirming the causal agent in Ontario using both classic and molecular techniques, and determining the efficacy of various cultural and chemical practices on the disease as well as on the population level of the causal agent.

Objectives

The objectives of this study were as follows:

Objective 1: Confirm the causal agent of TAP in Ontario using classical and molecular identification techniques.

Objective 2: Conduct a greenhouse trial to investigate the combined effect of nitrogen formulation and acidified irrigation on disease development and severity.

Objective 3: Conduct a field trial to investigate the disease suppression of several cultural practices.

Materials and Methods

Isolation, Culture, Molecular Identification of TAP causal organism

Samples of *A. stolinifera* either displaying symptoms or with a history of take-all patch were obtained from various golf course sites across Canada. Roots from samples were observed

microscopically for evidence of runner hyphae on root tissues. Sections of the microscope slide where runner hyphae were located were designated with a marker for isolation. Approximately 1 cm of root tissue was obtained from each sample. Root tissues of *A. stolinifera* were surface sterilized with 1% silver nitrate. Sterilized root tissue was plated on potato dextrose agar amended with streptomycin, kanamycin, and gentamicin at 50ppm, 50 ppm, and 10ppm, respectively. Samples were incubated at 18^{o} C for 21 days. Samples were subcultured on to clean media to ensure that the culture was pure.

DNA was extracted from the homogenized fungal tissues using a kit purchased from Norgen. Polymerase chain reaction was used to amplify DNA sequences specific to fungal organisms. Molecular identification, through sequencing, of each sample was conducted using primers designed to amplify the ITS region of fungal organisms. The primers used for the identification of fungal isolates are: NSA3: AAACTCTGTCGTGCTGGGGATA and NLC2: GAGCTGCATTCCCAAACAACTC (Martin *and Rygiewicz,* 2005), synthesized by University of Guelph Lab Services. The visualization of amplified DNA was done using 0.1% agarose gel electrophoresis (with an expected band size of ~1100bp). Successfully amplified DNA fragments were submitted to University of Guelph Lab Services for DNA sequencing. Each fungal organism will have some variation within its ITS region and this information can be used to conduct a search for the identity of the fungal organism using the National Centre for Biotechnology Information (NCBI) database.

Greenhouse Trial: Managing TAP with Acidifying Fertilizer and Acidified Irrigation

To test the preventative effects of acidified soils on TAP development and severity, a greenhouse trial will be conducted by inoculating pots of creeping bentgrass with samples of what was believe to be *G. cylindrosporus*. The project began in January of 2017. The pots were 10 cm x 10 cm and were prepared with 90% calcareous sand and 10% peat moss. A4 creeping bentgrass seed was applied at a rate of 0.25 g/pot. The plants were left under misters during the winter break and full germination was seen early January 2017. Plants were transferred to a growth chamber after six weeks of growth and the preventative treatments were started. Treatments were administered weekly as outlined in Table 1. Inoculum consisted of 4 different Gc isolates. Plants were inoculated with 4.5 g of Gc per pot. Using a pencil to penetrate the soil surface, three holes approximately 4 cm deep were created and filled with inoculum, followed by 100 mL of deionized water. Three weeks after inoculation, curative treatments were applied using same rate as preventative treatments. Eight weeks after inoculation the samples will be harvested and analyzed for runner hyphae, both incidence and severity will be documented. It is possible that there will not be sufficient disease infiltration after eight weeks, if that is the case the experiment will be extended for a few more weeks.

Table 1: Treatments were administered on Tuesdays in 60 mL aliquots. Plants were watered with 60 mL of tap water on Fridays. Acidified irrigation & combination treatments received 60 mL of pH 4 water every watering.

Treatment	**Weekly nitrogen application[1]**	**Weekly potassium application[1]**	**Additional treatment, quantity and source**	**Watering**

Urea (Negative control)	3.553 mg urea per pot	0.77 mg mono-potassium phosphate fertilizer (MKP) per pot		tap water once per week
Heritage (Positive control)	3.55 mg urea per pot	0.77 mg MKP per pot	0.252 µL heritage per pot every 28 days in 20 mL of water	tap water once per week
Ammonium Sulfate (AS)	7.62 mg AS per pot	0.77 mg MKP per pot		tap water once per week
Manganese sulfate ($MnSO_4$)	3.55 mg urea per pot	0.77 mg MKP per pot	1.68 mg $MnSO_4$ in 20 mL of tap water every 12 weeks	tap water once per week
Acidified irrigation (AI) (pH 4)	3.55 mg urea per pot	0.77 mg MKP per pot	60 mL of acidified irrigation (pH 4)	All water applied was acidified, an additional 60 mL was applied once per week
Combination	7.62 mg AS per pot	0.77 mg MKP per pot	1.68 mg $MnSO_4$ in 20 mL of tap water every 12 weeks	All water applied was acidified, an additional 60 mL was applied once per week

[1] Nutrients were applied in 60 mL of water, if multiple fertilizers were applied the fertilizers were mixed in the solution

Field Trial: Comparing Cultural and Chemical Methods of Disease Control

The trial was conducted at the Guelph Research Station on an USGA sand green with each experimental plot measuring one meter by one meter. The treatments were randomly assigned due to the high uniformity of the green. The cultural treatments consisted of urea as the negative control, ammonium sulfate, manganese sulfate, acidified irrigation, and a

combination of treatments that consisted of acidified irrigation, manganese sulfate, and ammonium sulfate. Both the manganese sulfate and the acidified irrigation have urea applied as there nitrogen source. The acidified irrigation treatments had irrigation water applied that was altered to have a pH of 5 through the addition of 2 M sulfuric acid. Additionally, preventative and curative rates of the fungicide azoxystrobin were applied up to three times throughout the season in conjunction with some of the combination and urea treatments. For the specifics of each treatment including rate of application refer to Table 2. The nine treatments were replicated four times resulting in 36 experimental units (Figure 1).

Figure 1. Layout of the experimental plots at the Guelph Research station in the 2017 growing season.

Sterilized water-soaked Kentucky bluegrass seeds were used as the substrate for the growth of the pathogen. The seeds were sterilized by placing them in a beaker of water over night, draining them, and putting them in an autoclave safe beaker. The seeds were then autoclaved, for 45 minutes at 121 °C, three times with at least 24 hours between each cycle. Pieces of agar with actively growing Gc were added to the beakers and mixed with the sterilized seeds. The beakers were incubated at room temperature for three to four weeks, based on level of fungal growth on the seeds. The inoculum was disturbed once per week to optimize fungal growth. The inoculum was then dried over several days under sterile conditions. A sample of the inoculum was plated to ensure Gc was the only fungus present in the inoculum. Each plot had six 10-cm deep plugs removed and each hole was filled with 6 g of inoculum. All the plots were watered with non-acidified irrigation after inoculation. The temperature and moisture of the soil were monitored three times per week to ensure proper moisture during cool, wet periods to encourage hyphal growth. Turfgrass colour, quality, and disease presence were rated weekly for each plot.

In early October, three plugs were removed from each plot. The 15 cm deep plugs were removed along the diagonal of the plot and removed at 40 cm, 70 cm, 1m. The cores were washed and twenty roots from each plug were observed under the compound microscope (Olympus BX51) to look for runner hyphae, the characteristic sign of *Gauemannomyces* spp (Figure 2).

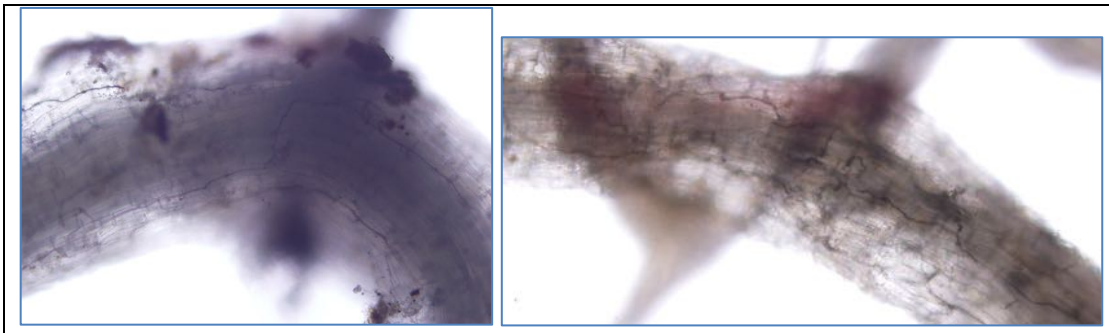

Figure 2. Photos of runner hyphae on and in root tissue from core samples taken from experimental plots in TAP field trial.

For replicates one and two, the number of runner hyphae present in the twenty roots were counted and this represented the severity of the infection. For replicates three and four, the number of the twenty roots that had runner hyphae were counted as incidence data, and the severity of infection was rated per root. The severity scale used to rate the pathogen is as follows: 0 - no runner hyphae, 1 - 1-10% of roots infected with runner hyphae, 2 - 11-25% of roots infected, 3 - 26-50% of roots infected, 4 - 51-75% of roots infected, and 5 - 76-100% of roots infected.

The data of the number of runner hyphae collected for replicates one and two was converted into the severity scale used for replicates three and four to allow the comparison of all four replicates. To ensure the data was converted correctly, a repeated measures ANOVA was performed on the collected data and the converted data. The results were the same, which allowed the use of the corrected data to compare all four replicates. Additionally, there was no significant difference based on the position of the plug in the plot. The visual ratings taken from June until October rated the plots on a scale of 1-9, based on ratings from the National Turfgrass Evaluation Program, with 1 being lowest quality or light in colour and 9 being highest quality or dark green in colour. The percentage of disease on the plots were also taken later in the season. The data collected, including severity, incidence, and visual ratings, were analyzed with SAS 9.4. The program *proc glimmix* was used to performed a repeated measures ANOVA on the data. The Tukey-Kramer method was used to perform pairwise comparisons of all the treatments to identify significant relationships between the treatment groups and assign letters to the data set.

Table 2: The nine treatments including the rates and frequency of application that were applied to the trial[1].

Cultural Practices	**Treatments - Rate**	**Frequency of Application**
Negative Control	Urea 1.5 kg N per season 2.72g per plot per applicaiton	Every 2 weeks
Positive Controls	Azoxystrobin curative – label rate Urea 1.5 kg N per season Azoxystrobin curative – label rate	Once Two applications

	Urea 1.5 kg N per season Azoxystrobin preventative + curative – label rate Urea 1.5 k N per season	 1 preventative application 2 curative applications
Manganese sulfate	Manganese sulfate - 0.125 g per plot per application Urea - 2.72 g per plot per application	Every 2 weeks Urea 2 weeks
Acidifying fertilizer	Ammonium sulfate 1.5 kg N per season 5.95 g per plot per application	Every 2 weeks
Acidified irrigation	4.5 ml of 2.5 M sulfuric acid added to 6 L of irrigation water Urea - 2.72 g per plot per application	Two times per week Urea 2 weeks
Combination	Ammonium sulfate - 5.95 g per plot per application Manganese sulfate - 0.125 g per plot per application Acidified irrigation - 4.5 ml of 2.5 M sulfuric acid added to 6 L of irrigation water	Ammonium sulfate every 2 weeks and manganese sulfate every 4 weeks Acidified irrigation twice a week
	Ammonium sulfate - 5.95 g per plot per application Manganese sulfate - 0.125 g per plot per application Acidified irrigation - 4.5 ml of 2.5 M sulfuric acid added to 6 L of irrigation water Azoxystrobin preventative and curative - 3 applications	Ammonium sulfate every 2 weeks and manganese sulfate every 4 weeks Acidified irrigation twice a week Azoxystrobin applications at the beginning of season and twice throughout the season

[1] In addition to the 9 experimental treatments, there was check plot per replicate that was not inoculated. This was to ensure that any presence of runner hyphae on the root tissue could be attributed to infection by Gc.

Results

Objective 1: Confirm the causal agent of TAP in Ontario using classical and molecular identification techniques.

Previous success (from interim reports): As mentioned in the previous reports, we had successfully identified the causal agent of TAP through molecular techniques using samples from Alberta, Saskatchewan, and Ontario. The next step was to confirm the results through classical techniques, specifically performing Koch's postulates. The study was initiated in November of 2016, but the pathogen is extremely slow-growing and less virulent (takes longer to infect the host than *G. graminis* var. *avenae*) so the study is not complete yet. Plants were infected with the pathogen on November 11, 2016 but signs of the pathogen did not become evident until the middle of January 2017. The plants were removed from the pots, slides of the roots were made and examined under the microscope. When a positive identification of runner hyphae was made the section was marked. The marked sections of roots were surface sterilized using a protocol involving silver nitrate. Roots were plated on selective media and incubated at 18°C. It was often necessary to subculture from these plates to reduce the incidence of contamination. Due to the slow-growing nature of the pathogen it will be several more weeks before we can confirm the identity of the isolated pathogen. We do have promising results from one of the four isolates tested but these need to be confirmed with molecular techniques.

Final report update: We have successfully inoculated plants with the Canadian isolates of TAP. We performed a Koch's postulates experiment where we successfully induced symptoms in a new, previously healthy plant. Several attempts were made to characterize the pathogen isolated from the plants with molecular techniques but we have been so far unsuccessful. We have had problems with the molecular identification of the organisms isolated from the infected plants which has caused quite a few delays. We believe that part of the problem is that there are naturally occurring fungi present in the seeds we are planting for our Koch's postulates. As such, we will repeat this experiment one more time in controlled growth chambers and with sterilized CBG seed. We are confident that we have identified the causal agent in our Canadian samples to a closely related species to Gga, *Gaeumannomyces cylindrosporus*, or Gc, but we need to isolate a pure culture from the infected plants to have the DNA of the organism sequenced before we can publish our results. It is our hope to publish the disease note in the summer of 2018.

Objective 2: Conduct a greenhouse trial to investigate the combined effect of nitrogen formulation and acidified irrigation on disease development and severity.

Update for April 2017: The growth chamber experiment is complete and the data analyzed. We noted a significant effect of the fungicide treatment and also noticed a reduction in the disease with the combined treatment that included ammonium sulfate, acidified irrigation and manganese sulfate. The cultural treatments did not significantly reduce the disease presence but there were some differences noted. This suggests that the cultural practices should be incorporated in the field trial that is beginning shortly so that their efficacy can be further evaluated. We may wish to repeat the growth chamber trial as well but need to let it run for longer since the newly identified pathogen takes longer to infect its host.

Final report update: There is no additional information on this objective as it was conducted as a preliminary study to help decide which treatments to include in the field trial. We were also trying to determine the best way to inoculate turfgrass with the pathogen as, to our knowledge, inoculation for the disease has not been conducted on turf to date.

Objective 3: Conduct a field trial to investigate the disease suppression of several cultural practices.

The results from the growth chamber experiment helped determine the treatments for the field trial. Due to the slow-growing nature of the pathogen the experiment needed to run for the duration of the summer and into the fall. This ensured that the pathogen had adequate time to infiltrate the root tissues of the plants.

Field Trial Outcome: After inoculation, symptoms of take-all patch and runner hyphae were present in all experimental plots, excluding control plots. The visual ratings of colour and quality suggested that manganese sulfate, acidified irrigation, and all urea treatments were able to matain a high standard of putting green surface (Figures 3 and 4). The plots that were given urea as a nitrogen source performed better than those given ammonium sulfate. This could suggest that the colour of creeping bentgrass is enhanced by the use of urea, and therefore leads to higher quality ratings compared to ammonium sulfate. We were unable to make a thorough assessment of disease ratings on the turf because we did not have the stress needed for the disease to cause symptoms on the turf. As such, the visual ratings did not differ by treatment and most of the treatments led to sufficient turfgrass quality. We did see a reduction in quality with combination treatment (ammonium sulfate, manganese sulfate and acifdified irrigation).

In order to more accurately compare the treatments, we collected samples from each of the treatment plots and observed the presence of runner hyphae (the fungal structures of the pathogen) on the root tissue. We then determined severity by the proportion of the roots that were covered in runner hyphae.

The severity of Gc was not altered by the different treatments, including the application of preventative and/or curative fungicides (Figure 5). This may suggest that the fungicide azoxystrobin, which is registered for use on *G. graminis var. avenae*, may not be as effective on Gc. However, these results are following just one year of field data, so we are not prepared to suggest that fungicides are ineffective for this disease at this point in time. What our data do suggest, however, is that inoculum level was reduced just as much with fertilizers as it was with fungicides. That said, our disease pressure was not very high, so we are not sure if this would be the case under higher pressure or more stressful conditions for the plant.

Finally, we noted that disease severity on the root tissue was significantly reduced in the plots with acidified irrigation when compared to ammonium sulfate. We also saw a numerical reduction in disease severity with the acidified irrigation plots when compared to all of the other treatments. Although the differences were not statistically significant, we believe this is a treatment to continue pursuing as it may have the greatest potential to reduce inoculum level and, ultimately, disease over time.

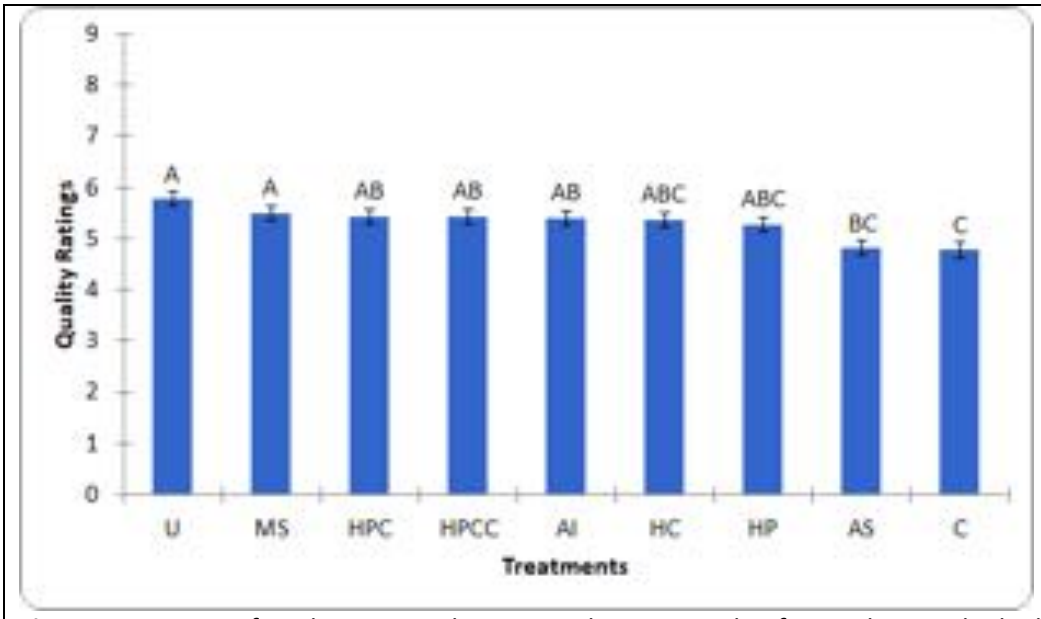

Figure 3: Average of quality ratings that were taken on a scale of 1-9, 9 having the highest quality, over the course of one summer. Treatments are ammonium sulfate (AS), combination (C), azoxystrobin curative (HC), azoxystrobin preventative (HP), azoxystrobin preventative, curative, and combination (HPCC), azoxystrobin preventative and curative (HPC), manganese sulfate (MS), urea (U), and acidified irrigation (AI). Error bars represent the standard error of the mean. Bars with different letters are statistically different from each other.

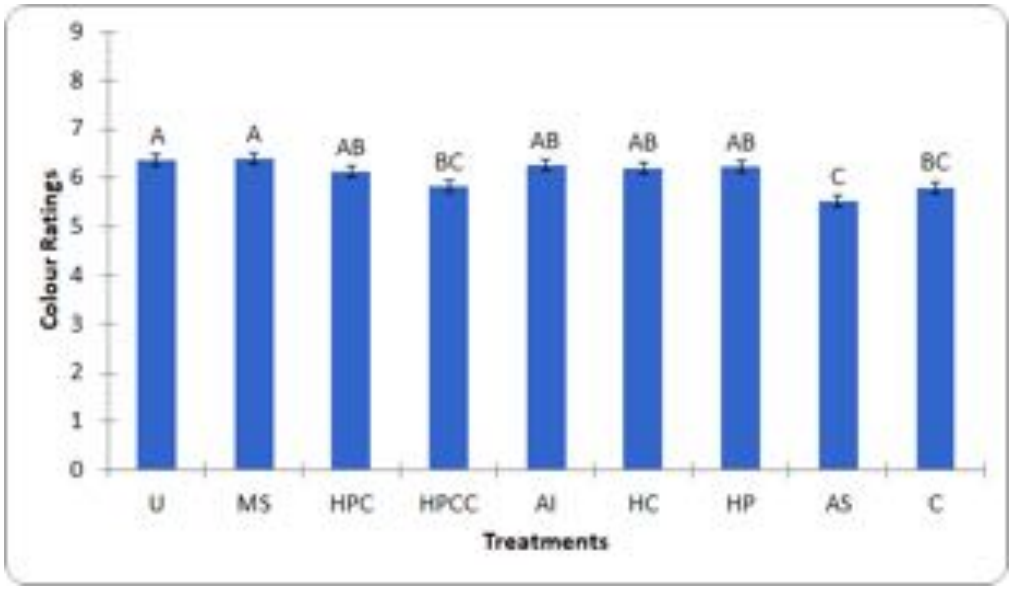

Figure 4: Average of colour ratings taken over the course of the summer. Scale of 1-9, 9 having the best colour a dark green. Treatments are ammonium sulfate (AS), combination (C), azoxystrobin curative (HC), azoxystrobin preventative (HP), azoxystrobin preventative, curative, and combination (HPCC), azoxystrobin preventative and curative (HPC), manganese sulfate (MS), urea (U), and acidified irrigation (AI). Error bars represent the standard error of the mean. A Tukey test was used to analysis the differences between treatments and assign letters to the treatments.

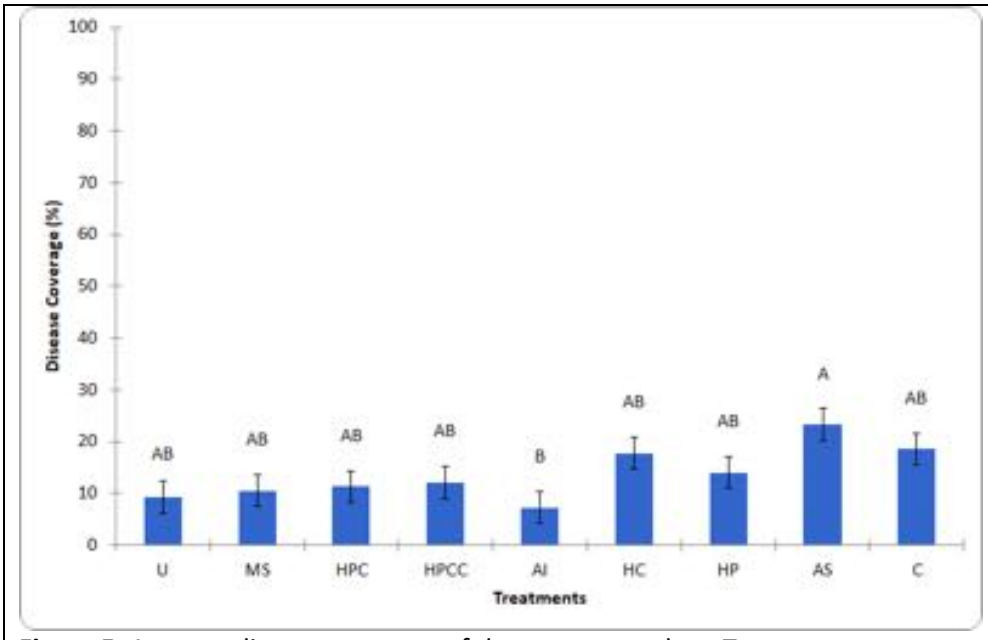

Figure 5: Average disease coverage of the treatment plots. Treatments are ammonium sulfate (AS), combination (C), azoxystrobin curative (HC), azoxystrobin preventative (HP), azoxystrobin preventative, curative, and combination (HPCC), azoxystrobin preventative and curative (HPC), manganese sulfate (MS), urea (U), and acidified irrigation (AI). A Tukey test was used to assign letters and the treatments with different letters are significantly different from each other. Error bars represent the standard error of the mean.

Accomplishments

During the course of this project we have accomplished the following:

1. We determined that the causal agent of TAP in AB, SK, MB and ON is not *Gaeumannomyces graminis* var. *avenae*, as was previously described. We are fairly certain that we are dealing with *G. cylindrosporus*, but are not comfortable with making this claim until we can isolate the DNA from our inoculated plants. This is a laborious process because we want to have the DNA confirmation, but are confident this will be published by the end of the summer of 2018. This is a significant finding as if we are able to confirm our results, it could suggest that many of the practices that have been recommended for this disease may not be as effective in Canada. It may also explain why we do not see the classic decline of the disease over time in most of the country.
2. We were able to determine that at low disease pressure, the use of adequate nitrogen can reduce disease development as effectively as the use of preventative and curative fungicides. This does not suggest that this will be adequate under heavy disease pressure (either due to excess inoculum or ideal conditions for development of the disease), however, but certainly warrants further research on increasing fertilizer to manage the disease.
3. We have determined that there may be the potential to reduce inoculum of *Gaeumannomyces* in the soil using acidified irrigation, without a significant reduction in turf quality. We are going to pursue this management practice in further trials to

determine if irrigating with acidified water can reduce disease development over time.

4. We were able to successfully inoculate *Gaeumannomyces* in the field, a feat that, to our knowledge, has not been done successfully in turf. There are no published reports of work on TAP that included artificial inoculation so this method had to be developed and optimized in our lab based on research with wheat and by trial and error. The presence of runner hyphae in our samples (Figure 2) indicate successful inoculation, evidenced by the lack of mycelia in the root tissue of the plots that were not inoculated.

HQP Training

To accomplish the project goals, MSc. Candidate Ernest Urquico was selected in September of 2014 to undertake this research project. Ernest had experience as a lab technician taking on responsibilities such as data acquisition, experimental design, and disease diagnostics in turfgrass studies. Ernest was accepted to the OAC graduate program under provisionary status, which encouraged him to focus on his academics. Ernest was able to achieve competitive grades of 87% and 85% in his first and second semesters, respectively. Through his courses, Ernest built a strong background in the subjects of plant disease, statistics, and molecular biology. Ernest also presented his preliminary results to industry professionals at the Ontario Turf Symposium (OTS), and assisted with the preparation of the OTS Plant Disease Diagnostic Workshop held February 2016.

Unfortunately, due to ongoing health issues, Ernest was not able to perform at his usual level during the winter semester of 2016 and as a result, he took a leave of absence for the summer semester and did not return after that point. As such, this project was turned over to Taylor Wallace, who recently earned her MSc with Dr. Jordan in turfgrass pathology and nematology. Taylor actively worked on this project as well as others within the lab.

Also trained on this project were undergraduate students Josh Callaghan and Karen Francisco. Josh conducted the greenhouse portion of the project as part of his undergraduate research project. He helped develop the interim report from Fall 2016. Karen Francisco conducted much of the field project as a summer student in 2017 and then analyzed the root data as fulfillment of her undergraduate research project in Fall 2017.

Karen graduated with her B.Sc. with honours in December of 2017 and was subsequently accepted to the Department of Plant Agriculture MSc program. She will be continuing her work on this project and is being funded by the Department of Plant Agriculture to do so.

Goals for Future Research

At this point, we are continuing with the project in order to definitively identify the causal agent of TAP in Canada. We were able to identify Gc as the fungus isolated from the samples collected from AB, MB and ON but have not been able to confirm this organism as present in the plants that we inoculated in-house. However, none of the isolates that were collected, submitted or isolated from the inoculated plants showed any presence of Gga. As such, we

are confident that the organism that is causing TAP in AB, SK, MB, and ON is **not** Gga and we should have a definitive causal agent by the summer. All morphological growth data and the DNA data to date suggest that the pathogen in these provinces is *G. cylindrosporus* but we would like to have DNA data from the inoculated plants to support our conclusions.

We are also continuing exploration into the potential for acidified irrigation to reduce the pathogen in the soil. We will be conducting a field trial following a short study to determine the most effective inoculation method. Although we were able to successfully inoculate established turf with the TAP pathogen, we would like to achieve a higher disease pressure and will be investigating various inoculation methods in order to do so.

It is our hope that by then end of Karen's graduate thesis, we will have a better understanding of the causal agent of TAP, the management methods to reduce pathogen survival in the soil, and the microbial community that could be useful for leading to take-all decline in turfgrass systems.