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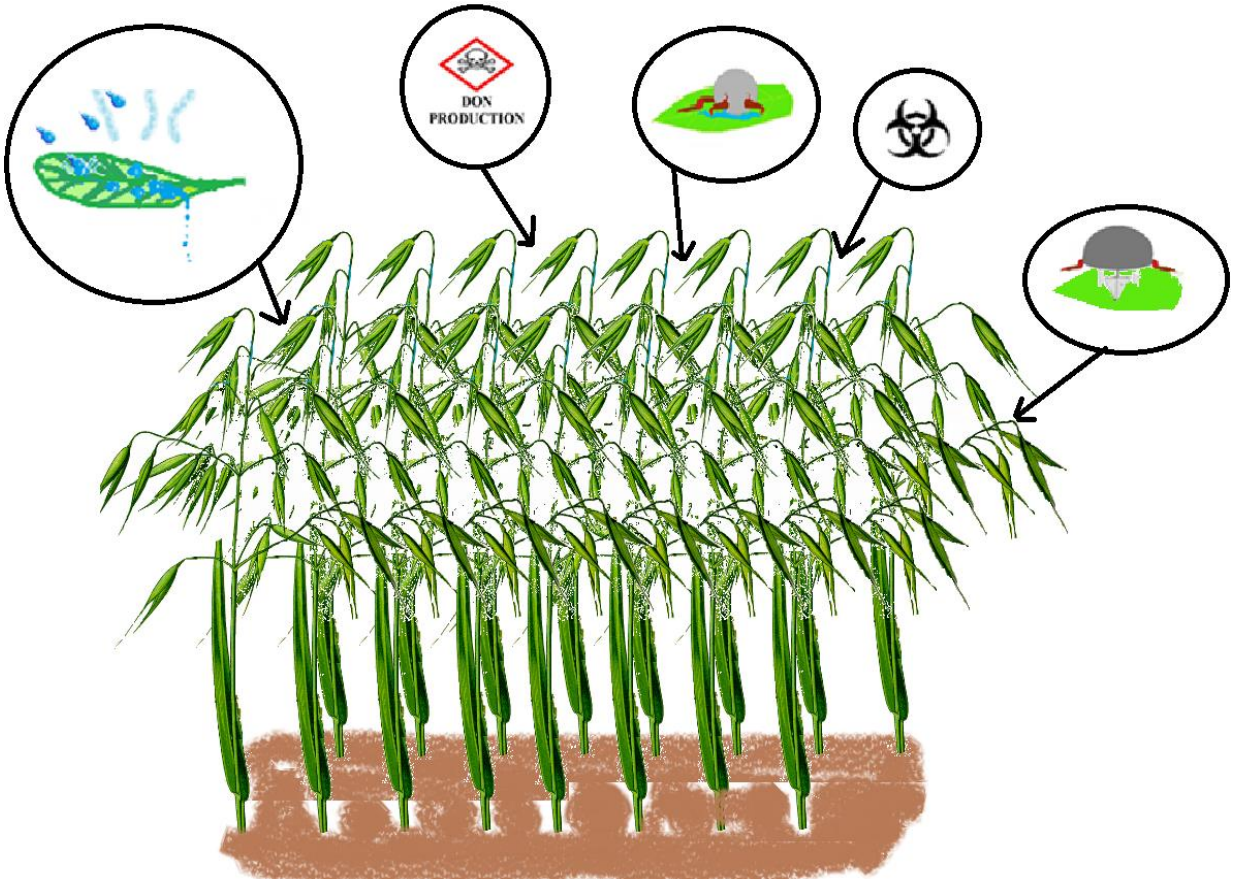
Deoxynivalenol prediction in oats

Deoxynivalenol prediktering i havre

Joel Markgren



Department of Crop Production Ecology
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Deoxynivalenol prediction in oats in English

Deoxynivalenol prediktering i havre

Joel Markgren

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"...Behold, seven ears of corn came up upon one stalk, rank and good. And, behold, seven thin ears and blasted with the east wind sprung up after them, and the seven thin ears devoured the seven rank and full ears. And Pharaoh awoke, and, behold, it was a dream."

"...the seven empty ears blasted with the east wind shall be seven years of famine."

"Behold, there come seven years of great plenty throughout all the land of Egypt: And there shall arise after them seven years of famine; and all the plenty shall be forgotten in the land of Egypt; and the famine shall consume the land; And the plenty shall not be known in the land by reason of that famine following; for it shall be very grievous."

Genesis 41:5-7, 27, 29-31

Popular scientific summary

Small cereal grains like wheat, barley and oats can be affected by a nasty disease called Fusarium Head Blight. This disease damages the plants by producing a certain compound in the grains called Deoxynivalenol (DON) which can still be on the grain even after the plants have been harvested. This compound is rather toxic and when humans consume it they can experience symptoms like vomiting, diarrhea and loss of appetite.

Every year large quantities of cereal grains are therefore thrown away because they are contaminated with this toxin. In recent years there have been extraordinary large quantities of the toxin in oats from Norway and Sweden. This in turn leads to problems for many people, the farmer will not be paid for his work and the price for oat grains increases which makes the breakfast porridge more expensive.

The problem can be relieved by the use of pesticides. Still it is not that easy, the pesticides must be applied at the right time, since the pesticides for Fusarium head blight can cause severe side effects on the environment. They are also rather expensive. However, despite the risk in some cases these pesticides must be applied if there is going to be any harvest at all.

In this work I have constructed a prediction model in form of a computer program that could aid the farmers to know when they are supposed to use their pesticides. The model simulates a virtual field of oat plants that are supposed to mimic real oat plants. These virtual plants are in the simulation exposed to weather conditions provided from a weather station nearby a real oat field. The virtual plants become susceptible for Fusarium head blight during flowering, which occurs at a certain number of days after sawing depending on the temperature. The Fusarium head blight disease is developed in the virtual plants when the conditions are suitable for the mould fungus Fusarium to germinate and attach on the plant surface with enough time to puncture it. The disease development is depending on temperature, sun light, humidity and leaf wetness. DON contamination occurs in the virtual plants after infection at higher temperatures and during leaf wetness. The leaf wetness data itself are simulated by calculating when there is water on the virtual oat plants´ leaves after rain, fog and dew events since leaf wetness is rarely provided by weather stations.

The model was tested in oat fields in Norway, to see if the virtual plants became contaminated with DON in the same extent as the real oat plants. The tests showed that the model so far is not capable to mimic DON contamination in real oat fields. Therefore it cannot be used with a weather forecast to make reliable predictions of DON contaminations. I tried to test other researchers´ models to see if their virtual plants became DON contaminated in the same extent as real oat plants. Unfortunately these models were not capable to mimic DON contamination either.

Some of the reasons why the models are thought to make these errors are that they are not analysed for miscalculations. They neither take in to account if the mould fungi responsible for the disease are present or not nor that the plant might recover from the disease.

Hopefully someone will enhance the models so the farmers will get proper support to answer the question “Should I use the pesticides or not?”.

Abstract

In recent years there have been problems with unacceptable high levels of the mycotoxin contaminant Deoxynivalenol (DON) in oats in Sweden and Norway. This is due to infections of the fungal pathogens *Fusamium graminearum* and *Fusarium culmorum*. The question which this thesis attempts to answer is: "Is it possible to predict these elevated levels of DON?" in order to time and conduct suitable countermeasures. A prediction model was created to answer this question. The model calculates a DON contamination risk index based on the likelihood of present germinated spores, plants susceptible for infection, infection event and production of DON. The model needs hour based weather data input for relative humidity and global radiation. Also, the model uses a leaf wetness model and a temperature driven phenology model to predict input data for leaf wetness, leaf surface temperature and plant growth stages. The model indices were compared towards DON measurements in oats in Norway and a regression analysis was conducted. The model did in a few cases show a strong correlation towards the measurements, but in most cases there was no correlation or a negative correlation. Therefore, it is considered that the model is not capable to predict DON contamination. Alternative model applications were conducted to predict DON in oats. Among the alternative applications, the prediction model ENV also known as GIBSIM for *Fusarium graminearum* infections in Brazil was included. However, only two instances with the ENV applications of all the alternative model applications showed strong positive correlation. Once again the models used failed to predict DON contamination. There is a risk that the models generated incorrect predictions due to calculation errors since no sensitivity analysis was conducted. The models might be capable to predict DON in oats if the study better compensates for environmental variance and if the models take into account factors like recovery and spore density.

Table of contents

Acronyms to remember

DON = Deoxynivalenol

FHB = Fusarium Head Blight

1. Introduction

There have been several major outbreaks of Fusarium head blight (FHB) disease in oats in Norway and Sweden the last ten years. This is quite unusual since the disease is seen as unproblematic in oat cultivation.

One of the associated problems with Fusarium head blight (FHB) is that the diseased parts of the plant start to produce a mycotoxin called Deoxynivalenol (DON). In both the Swedish and Norwegian yields there have been several reported cases with DON levels exceeding the Swedish national limits.

The Swedish University of Agriculture Sciences, The Norway agriculture research institute "Bioforsk", the Swedish farmers´ cooperative "Lantmännen" and the Swedish national food agency, are together with foundations from the Swedish Farmers' Foundation for Agricultural Research trying to find a way to once again have oat yields with low levels of DON.

One of the ways to combat the problem is to predict the presence of Fusarium head blight (FHB) in oat and rapidly counteract it with fungicides in early development stages. In order to do so a prediction model must be constructed. This master thesis will attempt to construct this kind of model.

1.1. Thesis disposition

This thesis aims on predicting Deoxynivalenol in oat with a modeling approach. In order to carry out the aims, the thesis is structured in the following way: First, a literature study that covers the field of study is conducted, which also provides a background. In the background questions like "what is FHB and DON?", " what kind of problems are involved with FHB and DON?" and "how are FHB and DON linked together?" are answered. In the background, there is also a presentation of previous attempts to predict FHB and DON in other cereals, and a short section about leaf wetness which is one of the main predictor variables in the models used in the method.

Second in this thesis structure the method part follows. The methods chosen for carry out the aim are to develop my own DON prediction model, use others prediction models and modify others prediction models. In the method section, there will be a detailed description of the models and the statistical methods used to evaluate them.

The rest of the report will consist of the sections results and discussion. In the results section the results from the methods are presented and in the discussion section, conclusions that can be drawn from the entire report are made.

2. Background

2.1. Agriculture problems related to Deoxynivalenol (DON)

Fusarium Head Blight (FHB) is a disease in small grain cereals and corn (Agrios 2005, p. 535). It causes yield and quality reductions due to damaged kernels and empty spikelets. The characteristic symptoms of the disease are brownish and pinkish colorations in the spikelets (Hörberg 2001, Tekle et al. 2013, Tekauz et al. 2004, Jansen et al. 2005). In oats however, the disease can be difficult to detect in field due to the oat morphology (Tekauz et al. 2004). The disease is also very problematic on a subclinical level, in which the symptoms are too weak to be detected. The problem is then, that mycotoxins are produced in the infected tissue- in this case the spikelets and kernels- without any visible symptoms. This thesis will focus especially on the FHB produced toxin Deoxynivalenol (DON) (Bottalico & Perrone 2002).

Recently there have been major problems in oat cultivations in Norway and Sweden due to elevated levels of DON. Two Swedish agriculture news magazines mention that the Swedish farmers´ cooperative, *Lantmännen*, have had major problems with their deliveries from their contracting farmers due to elevated levels of DON. They also mention several reported cases of deliveries in which the DON level has been above the acceptable limit. (ATL.nu 2012, Jordbruksaktuellt 2010). On a global scale DON contaminated grains is considered a serious problem which can lead to severe economic damages striking at all levels of society (Farmers Guardian 2008). Contaminated grains can lead to elevated food prices caused by detoxification costs, increased veterinary costs in animal production, increased human medical costs and non-harvested crops. Sometimes it can even be economically motivated to destroy the crop rather than harvest it when contaminated (Charmley et al. 1995).

2.2. Toxicological effects of DON

DON, which is also called vomitoxin, is a potential lethal toxin. Petska (2010) has reviewed animal studies in pigs and rats on DON poisoning and mentions the following clinical symptoms: diarrhea, vomiting, weakened immune system, induced anorexia, cell death and instant death. DON can cause a paradoxal immune suppressing and enhancing effect by inducing a ribotoxic stress response in the leucocytes, leading to either positive defense gene up regulations or apoptosis depending on dose (Petska et al. 2004). How DON induces anorexia is still not completely clear but it is suggested by laboratory experiments with rats by Flannery et al. (2012) that DON somehow creates a hormonal imbalance in the gut. The diarrhea and vomiting symptoms might be referred to injuries in the intestinal part by oxidative stress and decreased intestinal barrier permeability, leading to higher risk of infection and toxification (Pinton et al. 2009, Kouadio et al. 2005). In order to limit the problems associated with DON for humans and animals, some countries´ national authorities have put up limits for its maximum occurrence in small cereal grains. The Swedish National Food Agency has decided to limit DON occurrence in non-processed oat to 1250 µg/kg for human consumption and 8000 µg/kg for animal consumption (NFA 2012).

2.3. DON producing infections

For an infection process to be successful, many steps need to be succeeded many times in certain sequences. For FHB, the disease cycle can be summarized by **figure 1**. In order to have DON contamination in the harvest, there has to be an FHB agent that infects the kernels. The infection is only possible after the start of anthesis. The infection itself is only possible in certain conditions. Although an infection has occurred there still are possibilities for the plant to recover. The infection might lead to DON contamination or mycelia growth, which leads to DON contamination. The different parts necessary for infection and DON production are described more thoroughly in the sections below.

2.3.1. Pathogens

In order for infectious diseases to occur, a pathogen is necessary. The pathogen in fungal cases is responsible for the disease by parasitizing or consuming the host. In some cases it might also be the other way around, it is the host who recognizes the pathogen but launches the wrong type of countermeasures ending in the pathogen´s favour, or the countermeasures are affecting the plant in undesirable ways. There is not only one pathogen responsible for FHB, most of them belong to the fungi genus *Fusarium* (Xu 2008). Those species considered more often responsible for FHB are *F. avenacum, F. culmorum, F. graminearum, F.Poae,* since they are most frequently found when screening field samples, based on two field studies together with over 100 fields by Waalwijk et al. (2004) and Yli-Mattila et al. (2004). Of these four pathogens only *F. culmorum* and *F. graminearum* are of interest in this thesis, because those two are the only two FHB pathogens that manage to produce DON when they infect a host plant. There is a third FHB pathogen that is known to have DON producing strains: "*F.crookwellense".* This pathogen is omitted from this work since those strains are considered rare and the species is not known to be a problematic pathogen in Scandinavia (Bakan et al. 2002, Miller & Greenhalgh 1991, Bottalico & Perrone 2002).

2.3.2. Spore germination

In **figure 1**, it is possible to see the presence of inactive FHB pathogens in the spore cloud. In order to become infectious the spores need to germinate, which is the successive step to the spore cloud in figure 1. The *Fusarium* pathogens start their life cycle and become infectious on the plant or neighbouring plants by spore germination. The germination procedure is controlled by type of spore, quality of the spore, temperature, relative humidity (RH) and light. There are two types of spores (Agrios 2005, p. 388-389):

- Asexual spores also called conidospores.
- Sexual spores also called ascospores.

F.graminearum can germinate by both types of spores but *F.culmorum* has not yet any known sexual reproduction stage and is therefore only known to germinate through conidospores (Leslie & Summerell 2008, p158-159).

In order for a spore to germinate the following criteria need to be met:

- Temperature between 4 °C to somewhere over 20 °C.
- Relative humidity between 80% to 100%.
- That the spore is alive.

Also the germination is faster under darkness. The criteria are based on a laboratory experiment on *F.graminearum* conidospores by Beyer et al. (2004). According to the same experiment the germination procedure is completed after 2 to 6 hours at optimal conditions (20 °C, 100% RH, in darkness). The ascospores require similar criteria for germination as the conidospores, the only thing differing is that they need a relative humidity above 90%. The average time for ascospore germination at optimal conditions is slightly faster. These ascospore data are based on a laboratory experiment on *F.graminearum* ascospores by Beyer et al. (2005).

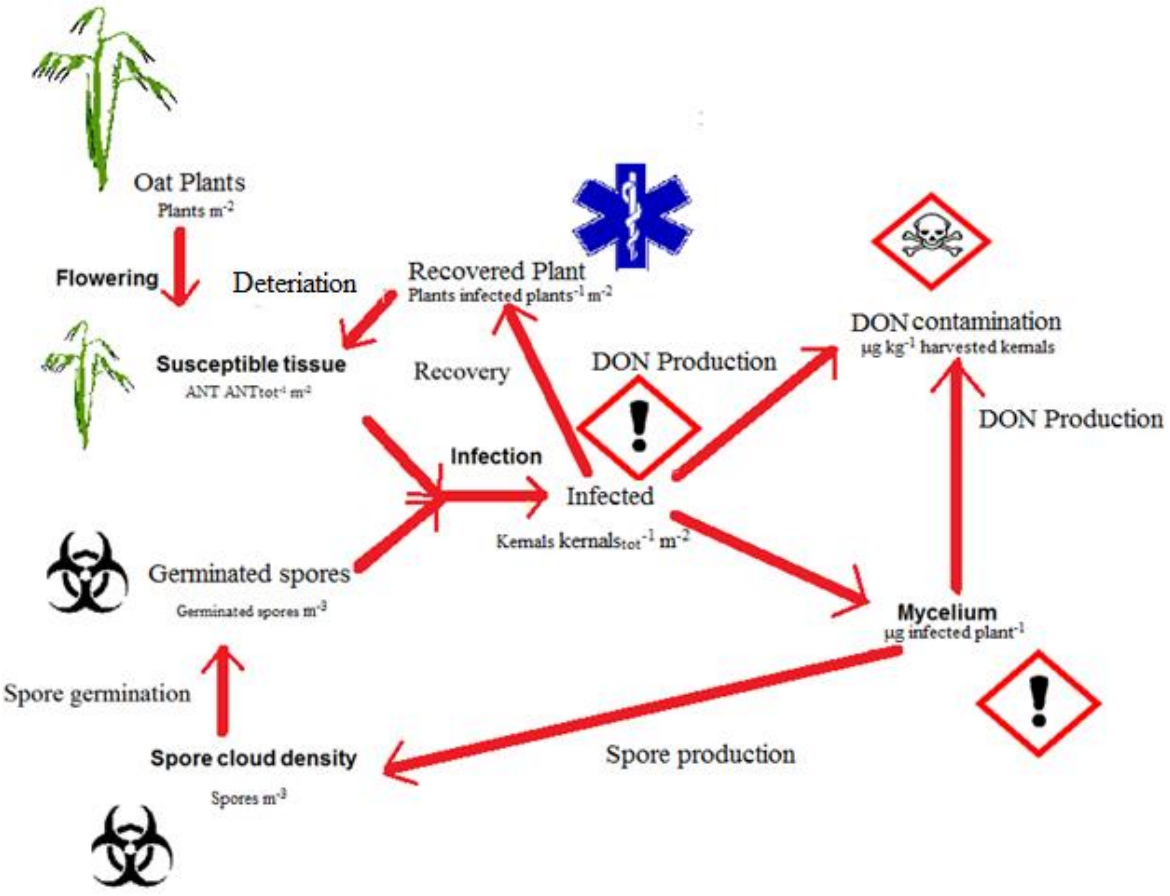


Figure 1. Basic overview of essential needs for an FHB infection and the possible outcomes of an infection. The red arrows indicates process, the sections accompanied with biological hazards logo are infectious matter related states, the sections accompanied with oat plants are plant phenology related states, the sections accompanied with an caution logo are infection related states, sections accompanied with the star of life are recovery related states and sections accompanied with a death skull logo are related Deoxynivalenol production states.

2.3.3. Infection

When studying **figure 1**, it shows that both germinated spores and susceptible tissue are needed for an infection to occur. This complicated process will therefore be more thoroughly described in the mechanisms necessary for infection both from a pathogen´s and a plant´s view in the subsections, "infection mechanisms" and the "susceptible host". There will also be a short description of the necessary environmental conditions of an infection.

2.3.3.1. *Infection mechanisms*

After the pathogen, *F.graminearum* or *F.culmorum*, has germinated, it needs time to recognize the host, attach and colonize the host and form an apresoria. An apresoria is an infection structure, often composed of a needle like structure called an infection peg (Agrios 2005, p. 82-86,). The apresoria penetrates the epidermis and cuticula by puncturing it with its peg. When the epidermis is penetrated, the fungus starts to invade the plant. The time to succeed with an infection after spore germination takes about 18 hours, according to cytological studies on *F.graminearum* infecting wheat plants by Wanjiru et al. (2002). For those who are interested in fungal infection procedures the following review for the model fungal pathogen organism *Magnaporthe oryzae* by Wilson & Talbot (2009) is recommended.

2.3.3.2. *The susceptible host*

Oat is susceptible to *F.graminearum* and *F.graminearum* infections related to FHB with the start of anthesis. That is because the pathway of infection is found to be through the floret mouth. This was discovered by Tekle et al. (2012) who inoculated oat plants with *F.graminearum* and then stained the pathogen with lactophenol blue and observed it in a microscope. In the same experiment it was also found that there was a particular high risk of infection when the anthers where extruded and present. Although the anthers are not essential for infection they seem to promote increased fungal hyphae development. They also seem to enhance the infection process according to an experiment by Kang & Buchenauer (2000) where the infection process in a wheat spikelet by *F.culmorum* was monitored in an electron microscope. Wheat and barley are also particularly susceptible during anthesis due to extruded and present anthers. In the latter two species, it seems like betaine, choline and another unknown substance present in the anthers are contributing to the infection, and it might be the same for oat (Strange et al. 1974, Yoshida el al. 2007). It is rather unclear, but it seems like some oat cultivars never drop their anthers, therefore it is speculated that these cultivars always suffer from high risk of infection after start of anthesis.

2.3.3.3. *Environmental conditions for infection*

In order to infect the plant, the environmental conditions need to be right, this might be for activating infection mechanisms and/or making the pathogens enough dominant towards competing microbes enabling them to infect. The environmental conditions necessary for infection for the two pathogens *F. graminearum* and *F. culmorum* are stated in **table 1**.

Table 1. Necessary environmental conditions for FHB pathogens to infect.

Species	Temperature, °C	Relative humidity, %
F. graminearum	10-35	91-100
F.culmorum	20-35	65-100

The infectious conditions for *F.graminearum* in **table 1**, are based on laboratory experiments by Rossi et al. (2001), where wheat spikelets were inoculated at different temperatures and humidity. But according to those results is it possible for *F.graminearum* to infect at 85% relative humidity. The patoghen is despite Rossi et al. (2001) ´s experiments considered to infect only at 91-100% relative humidity. That is because those experiments were conducted at sterile conditions, whereas in field, there are no sterile conditions. The pathogen is rather sensitive towards microbial competition and manages to be dominant only at relative humidities above 91%. That is according to a revive article by Doohan et al. (2003) who refer to three different fungal competition experiments on different growth media, temperatures and moisture by Magan & Lacey (1984), Marín et al. (1998) and Marín et al. (1998).

The temperature requirements for *F.culmorum* in **table 1**, are based on laboratory experiments by Rossi et al. (2001). The minimum humidity conditions are based on the same experiment by Rossi et al. (2001). The results were confirmed by Chandelier (2011) where 698 samples were collected during 6 years, where it seemed like *F. culmorum* is capable to infect in field at low relative humidity even if it is challenged by other microbes. The maximum humidity conditions are based on a field experiment by Lacey et al. (1999) with inoculated wheat plants in a mist irrigation system.

2.3.4. DON production

F.graminearum and *F.culmorum* are both necrotropic pathogens, meaning that they consume their host by killing its organic tissue. To do so, the pathogens trick the plant´s defensive system by making it think it is a biotroph trying to parasitize the plant. The plant is then activating a kind of suicide defense containing among many mechanisms the sacrifice of its own cells by reactive oxygen bursts. One of the compounds the pathogens use to trick the plant to conduct this suicide behaviour, is the DON (Jansen et al. 2005, Desmond et al. 2008). Therefore, it is possible to see correlations between the pathogen´s capability of producing DON and injuries on the plant (Mesterházy 2002).

The environmental requirements for both *F.graminearum* and *F.culmorum* are stated in **table 2**, based on laboratory experiments by Ramirez et al. (2006), Schmidt-Heydt et al. (2011) and Hope et al. (2005). In **table 2**, it is possible to see that both pathogens require a relative humidity in the leaf wetness regime (above 93% RH) in order to produce DON. But the fungi differ in one point

apart from their temperature requirements, the *F.culmorum* fungus is capable to produce 7 times more DON under optimal conditions (20 °C 99.5% RH) (Schmidt-Heydt et al. 2011).

Table 2. Necessary environmental conditions for FHB pathogens to produce DON.

Species	Temperature, °C	Relative humidity, %
F.graminearum	15-30	99
F.culmorum	20-30	93-99

2.4 Modeling

In order to predict DON contamination in oat yield all knowledge about how the contamination arise needs to be gathered in a model. **Figure 1** is a form of a model where all necessary states for DON contamination are gathered. The stages are driven by processes, for example between the infected and DON contamination stages there is a DON production process. The processes are in turn driven by different driving variables. For example, the DON production process in **figure 1** is according to section "2.3.4. DON production" driven by temperature and humidity variables. One of those variables that will be used a lot in the modeling attempts in this study is leaf wetness which is described further down in the section "2.4.2. Leaf wetness".

2.4.1. Who benefits from modeling?

Since it is difficult to observe both *Fusarium* infection and DON production in field, it is in the farmers´, buyers´/insurances´ and the researchers´ interest to create a model for the disease in order to monitor real time outbreaks, forecast outbreaks, countering the disease and learn more about it.

- It is in the farmers' interest to have a reliable prediction model for both estimating the status of real time infections and to predict future problems with DON. This provides information needed to combat the infections with an Integrated Pest Management (IPM) approach, where it is possible for the farmer to forecast upcoming problems and have time to develop sustainable fungicide strategies. Cooley & Autio (1997) conducted this kind of IPM strategy and applied fungicides in an apple orchard when it was the most economically and ecologically sustainable, resulting in a drop of fungicide usage with more than 30%. It is important to time the fungicides applications for FHB since there are few fungicides that are effective, and unjustified fungicide applications increase problems with resistance (Brent 2011). Those fungicides that are registered globally for FHB are often composed of triazole or benzimidazole compounds, but the results after treatments are often disappointingly bad (Mesterházy et al. 2011, Bradley & McMullen 2008). Besides, in Sweden only a triazolinthion compound "Proline EC 250" is registered for FHB in oats (Swedish Board of Agriculture 2013, Swedish chemical agency 2013).

- It is in the buyers´ and insurance companies´ interest to know risk areas of disease outbreaks when contracting the farmer in order to negotiate countermeasures and prices. It is also in the buyers´ and insurance companies´ interest to have a high quality of harvest prognosis with the aid of a model before laboratory results from test samples are available. If the model is good the need of laboratory test might decrease (Myers et al. 2010). The model might also be in the buyers´ interest in the way that it is possible to estimate and forecast the quality of competing buyers, and therefore it might be easier to conduct strategic business actions (Zajac & Bazerman 1991).

- It is in the researchers' interest to gain knowledge about the disease through modeling. Through the model, mathematical relations and mechanistic functions between pathogens, hosts and environmental factors provide a gathered knowledge (Van der Maanen & Xu 2003).

2.4.2. Leaf wetness

The term leaf wetness means that there is free water available on the leaf surface, this occurs when the relative humidity is above 91% or when water somehow intercepts the surface. Many agronomical and horticultural pathogens need leaf wetness to fulfill one or all infection stages to successfully infect their host (Huber & Gillespie 1992). For example, apple scab, mango malformation disease by *F. mangiferae* and *Pseudomonas syringae* infections on tomato require a certain amount of leaf wetness to complete the infection procedure (Hartman et al. 1999, Gamliel-Atinsky et al. 2009, and Preston 2000).

As seen in **table 3** both fungi *F. graminearum* and *F. culmorum* need leaf wetness at least in one of the stages that are required for DON production. *F.culmorum* is not that sensitive towards leaf wetness during infection phase. Both fungi can germinate from a conidospore which does not require direct leaf wetness but close to it. *F. graminearum* requires leaf wetness on all stages of infection stages; spore germination (when germinating as an ascospore), infection and DON production.

Table 3. Which stages toward DON production requires leaf wetness for the fungi F.graminearum and F.culmorum. Leaf wetness=leaf wetness is required, No leaf wetness= leaf wetness is not an essential requirement.

	F.graminearum	*F.culmorum*
Spore germination	No leaf wetness, leaf wetness	No leaf wetness
Infection	Leaf wetness	No leaf wetness
DON production	Leaf wetness	Leaf wetness

2.4.2.1. Leaf wetness prediction

Leaf wetness can be measured on field level with leaf wetness sensors but it is difficult for the sensor to mimic the leaf canopy perfectly and therefore it is difficult to obtain true leaf wetness values, which can be seen in a leaf wetness comparison trial by Gillespie & Kidd (1970). There is seldom meteorological service providing forecasted leaf wetness data because the leaf wetness varies from crop to crop due to size of canopy etc. Therefore often a leaf wetness model for the specific crop and area is needed.

2.4.3. Previous model approaches

Since leaf wetness is such an important environmental requirement for DON production and FHB infections, it can be found as an important predicting variable in several FHB and DON predictions models. The models are either in the form of regression models or mechanistic models. The regression models are based on statistical correlations between the factors involved in the model. In the mechanistic models the functions of the pathogen and the host are considered, and not only the statistical relations between environmental factors and disease occurrence. Some of the models used for DON or FHB are presented below:

De Wolf et al. (2003) and Bondalpati et al. (2012) used the duration in hours at temperatures 15-30C and a humidity above 90% RH after anthesis in wheat in a regression model that predicted 84% of all observed FHB infestations correctly.

Moschini & Fortugo (1996) got a correlation of R^2=0.886 at a 2 year old validation trial in Argentina. They used a regression model for FHB prediction in wheat using the combination of a rainy day with high relative humidity (≥81%) followed by a day with high relative humidity (≥71%) after anthesis, as a prediction variable.

Rossi et al. (2003) constructed a mechanistic model for DON prediction in wheat and barley. The model requires leaf wetness in order to register DON occurrence or FHB infection. The model was validated in the Emilia-Romagna region of northern Italy and included 981 plots of wheat and 102 plots of barley providing a correlation of r=0.64 on a significance of P<0.001 (Rossi et al. 2007).

Del Ponte et al. (2005) constructed a mechanistic model for FHB prediction in wheat. The model registers FHB infections when there are two rainy days or one rainy day followed by a day with high humidity. The model was validated in Passo Fundo in Brazil with parameter data from three years, providing a correlation of r=0. 84 on a significance level of P<0.01.

Hooker et al. (2002) constructed a regression model for DON prediction in wheat based on temperature, relative humidity and precipitation. The model is commercialized on an international scale named as “DON cast” (WIN 2013) and has an 80-85% accuracy (Schaafsma & Hooker 2007).

Detrixhe et al. (2003) constructed a model specified on the relation temperature and leaf wetness duration. The model considers the combination of a temperature 12°C or above during leaf wetness 6 days before to 8 days after flowering and it registers increased risk of FHB when time of leaf wetness and temperature exceeds 50 hours. The model was validated on 43 wheat fields in Libramont in Belgium and managed to classify 32 of them correctly.

There also exist regression models for DON prediction in oat by Elen et al. (2010) and Lindblad et al. (2012), but these models unfortunately only find correlations towards DON occurrence in certain geographical locations.

3. Aims

The objective of this thesis is to aid Swedish and Norwegian farmers to time fungicide applications, which in turn leads to less DON contamination and an agriculture based on IPM principles. To do so this thesis is focused on the following goal:

- Predict DON contamination in oat with a modeling approach.

4. Methods

4.1. Field samples

The Norwegian Institute for Agricultural and Environmental Research "Bioforsk" has provided this thesis with field data from the work of Hofgaard et al. (2010). The data contain DON values from 64 field samples in two oat cultivars Belinda and Bessin from Norway between the years 2004 to 2008, presented in **table 4**.

Table 4. Number of DON measurement samples from each location and year.

	2004	2005	2006	2007	2008
Roverud	2		6		
Tjølling					2
Øsaker			2	2	1
Gvarv	1	1	1		
Hokksund		1	1		
Ramnes			2	2	3
Rakkestad	2	4	4	3	4
Ås	2	5	6	7	

4.2. Weather data

Hourly weather data for mean global radiation per hour, precipitation per hour, mean relative humidity per hour, mean temperature per hour, leaf wetness at 2 meters´ height and mean wind speed per hour at 2 meters´ height were used for corresponding location and growing season for the field samples. The weather files were downloaded from Landbruksmeteorologisk tjeneste (LMT) operated by Bioforsk (2013).

In the field sample data from the Norwegian Institute of Agricultural and Environmental Research it was noted that some samples were taken far away from the weather station. The longest noted distance between sampling site and corresponding weather station was 50km.

4.3. Modeling strategy

All models were constructed and run in the modeling program Powersim constructor Version 2.51. Those who wish to repeat or inspect the constructed models can also use other software similar to Powersim, since modeling is based on conceptual thinking.

The main modeling strategy involved the use of the **DON model** together with the **phenology model v.1** and a **leaf wetness model**.

The **DON model** is a model for predicting DON contamination. The **DON model** requires relative humidity data and net radiation data from weather files and also leaf wetness and canopy temperature data from a **leaf wetness model**. For simulating infection under certain growth stages for the crop a **phenology model for the host plant** is required.

Phenology model v.1 describes the oat development during the anthesis period and is taken from Del Ponte et al (2005) but is slightly modified by Persson et al (2013). To operate the model only ambient air temperature from weather files and sowing date for the crop are needed.

The **leaf wetness model** predicts hour based canopy temperatures and leaf wetness occurrence from rain, fog and dew. The model requires net radiation, wind speed, ambient air temperature and relative humidity inputs from weather files to operate.

4.4. DON model

The DON model is developed on my own and based on the literature reviewed in section "**2.3 DON producing infections**". The model is designed for identifying spore germination, infection and DON production, which is illustrated in **figure 2**. The model calculates infection and DON production events for *F. culmorum* and *F.graminearum* separately. The final DON risk index is based on the accumulated sum of the DON production for the two pathogens.

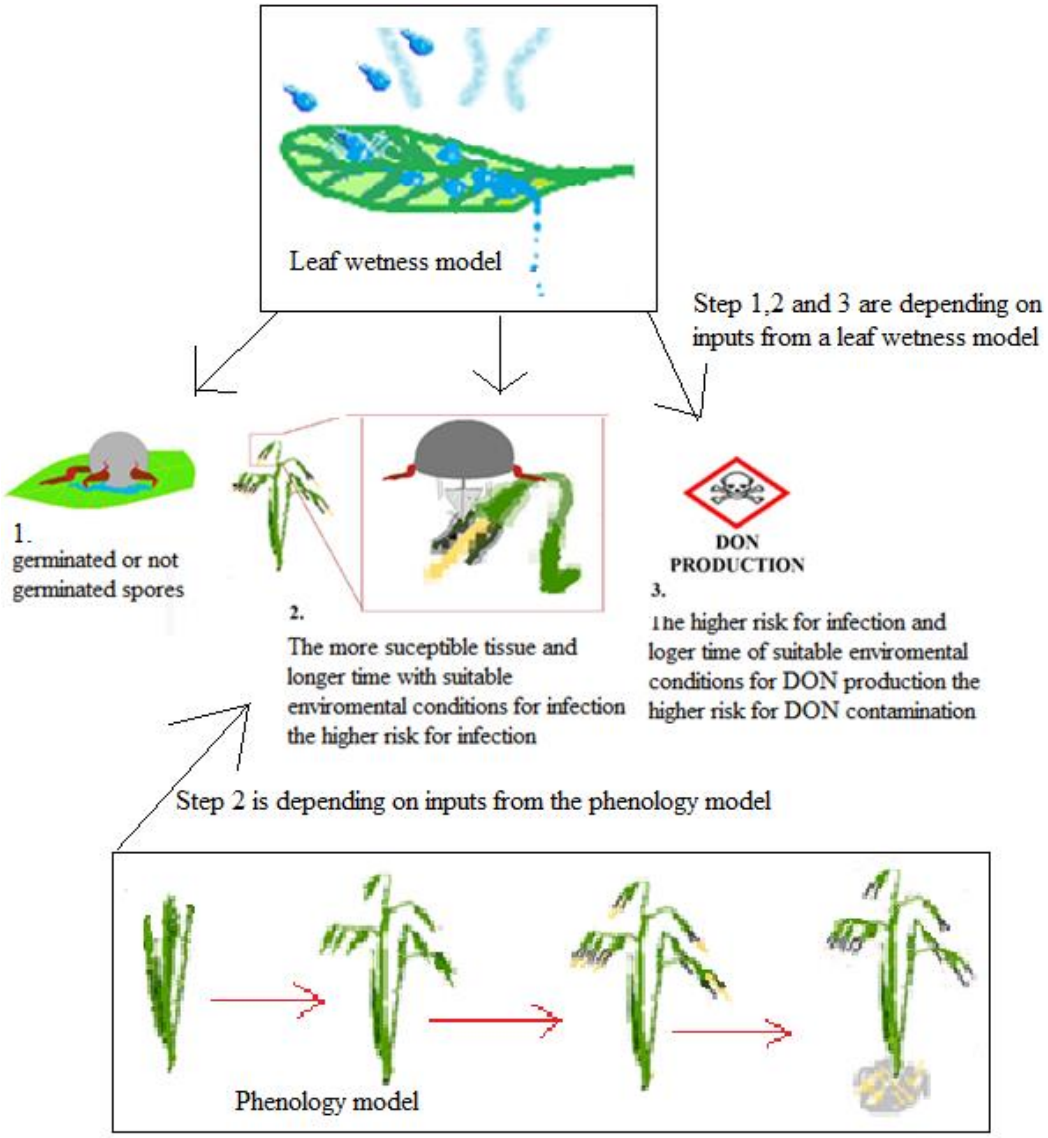


Figure 2. Flow chart of DON model. First the spores germinate and becomes infectious matter. Second the infectious matter infects present susceptible tissue, this step requires input data from a phenology model. Third the infected oat plants starts to produce DON. All steps require data from a leaf wetness model.

4.4.1. Spore germination

The DON model starts with the first step in **figure 2** "germination of spores" reviewed in the section"**2.3.2 Spore germination**", which it assumes is always present. Spore germination in the model occurs either by ascospore (spore $germ_a$) or conidospore germination (spore $germ_c$) for *F.graminearum*, while germination for *F.culmorum* only occurs by conidospore germination (spore $germ_c$). The "spore germination process" used in the model is based on spore germination biology reviewed in section

The ascospore germination (spore $germ_a$) is the most rapid and takes four hours at required conditions while conidospore germination (spore $germ_c$) needs five hours. The germination is reduced with one hour if it occurs during darkness ($R_s<0$), for both conido- and ascospores. The

environmental requirements are a canopy temperature (T_c) of 4-21˚C and leaf wetness (L_w) during the germination. Conidospores can also germinate if the relative humidity (RH) is above 80% if there is no leaf wetness (L_w). The conditions necessary for conidospore (spore $germ_c$) and ascospore germination (spore $germ_a$) are formulated in **equation 1** and **2**.

spore $germ_c$= 1 IF $R_s \geq 0 \rightarrow \int_{t=0 \rightarrow t \geq 4}$ $4 \leq T_c \leq 21$ AND (L_w=1 OR RH>80), OR IF $\rightarrow R_s < 0 \int_{t=0 \rightarrow t \geq 5}$ $4 \leq T_c \leq 21$ AND (L_w=1 OR RH>80) ELSE =0	Spore germ=1,indicates succesful germination, Spore germ=0 indicates failed germination	1
spore $germ_a$= 1 IF $R_s \geq 0 \rightarrow \int_{t=0 \rightarrow t \geq 3}$ $4 \leq T_c \leq 21$ AND (L_w=1), OR IF $\rightarrow R_s < 0 \int_{t=0 \rightarrow t \geq 4}$ $4 \leq T_c \leq 21$ AND (L_w=1) ELSE =0		2

where spore $germ_c$ = indication of conidospore germination, spore $germ_a$ = indication of ascospore germination, t= time (h), T_c =canopy temperature (℃), R_s = global radiation(W m^{-2}), Lw = leaf wetness

4.4.2. Infectious matter

After spore germination (spore germ) the pathogen is according to **figure 2** and the sections "**2.3.3.2. The susceptible host**" and "**2.3.3.3. Environmental conditions for infection**" supposed to infect the plant. If the conditions for infection are not suitable, there is a possibility in the model for the pathogens to survive as infectious matter (Inm) and infect at a later opportunity. This model process is based on infection biology reviewed in the sections ".

The indication of present infectious matter (Inm) requires that there has been an indication of spore germination for respective pathogen. In order for the infectious matter (Inm) to survive, leaf wetness (L_w) is the only necessity. The infectious matter (Inm) of *F.culmorum* can instead of leaf wetness (L_w) also survive if there is a relative humidity (RH) above 65%. The requirements for pathogen survival as infectious matter (Inoc) are formulated in **equation 3** and **4**.

Inm_c= 1 IF (L_w=1 OR RH>65) AND $\int_{t=0}$ spore $germ_c$=1ELSE = 0	$Inoc_c$= 1, indicates present surviving infectious matter, $Inoc_c$= 0. indicates non present or perished infectious matter.	3
Inm_g =1IF L_w=1 AND $\int_{t=0}$ (spore $germ_c$=1OR spore $germ_a$= 1) ELSE=0		4

where Inm_c = indication of F.culmorum infectious matter presence, Inm_g = indication of graminearum infectious matter presence, t = time (h), RH = relative humidity (%)

4.4.3. Infection

As illustrated in **figure 2** and explained in section "**2.3.3. Infection**" an infection requires both that the host has susceptible tissue and that there are pathogens ready to infect when the environmental conditions are suitable.

If there is an indication of present infectious matter (Inm) at environmental conditions suitable for infections, an infection score is produced (Inf) scoring one point for each hour after 18 hours. This score indicates a risk for infection, the higher score the higher risk for infection. The reason why the scores are not produced until after 18 hours is that in order an pathogen to fulfill an infection process it needs 18 hours.

F. graminearum requires a canopy temperature (T_c) of 10 °C to 35 °C and leaf wetness (L_w) to succeed with the infection. *F. culmorum* requires a canopy temperature (T_c) of 20 °C to 35 °C, leaf wetness (L_w) or a relative humidity (RH) above 65% during the infection. The infection requirements are described in **equation 5** and **6**.

$Inf_g = n + t_{18}$ IF $\int_{t=0 \rightarrow t=18+n} Inoc_g = 1$ AND $10 < T_c < 35$ AND $L_w = 1$ ELSE $= 0$	$Inf \leq 1$, indicates an elevated risk of succesful infections, Inf = 0. indicates no risk of succesful infection.	5
$Inf_c = n + t_{18}$ IF $\int_{t=0 \rightarrow t=18+n} Inoc_c = 1$ AND $20 < T_c < 35$ AND ($L_w = 1$ OR RH>65) ELSE = 0		6

where Inf_g and Inf_c = infection score, t=time h, t_{18}= time 18 hours (h), n = number of hours after 18 hours (h)

If the infection scoring starts, but later one or more criteria are missing, the scoring is halted and reset.

The infection score is multiplied with the relative total amount susceptible tissue (ST) and produces an accumulated disease score, according to **equation 7** and **8**.

Risk $inf_g = \sum(Inf_g * ST)$	7
Risk $inf_c = \sum(Inf_c * ST)$	8

where Risk inf = disease score

4.4.4. DON production

The final step in the model is, as illustrated in **figure 2** and explained in section "**2.3.4. DON production**", the DON production. When the circumstances are favourable for DON production the model creates a DON risk index (Risk DON), which is based on the disease score produced during indications of possible infections.

F.graminearum infected plants require leaf wetness (L_w) and a canopy temperature (T_c) of 15°C to 20°C for DON production. *F.culmorum* infected plants also need leaf wetness (L_w) but a canopy temperature (T_c) of 20°C to 35°C. *F.culmorum* can when the canopy temperature (T_c) is between 20°C and 21°C produce a sevenfold index value. The criteria's for producing DON risk indices are summarized in **equation 9** and **10**.

$\text{Risk DON}_g = \text{Risk Inf}_g$ IF $15<T_c<20$ AND $L_w=1$ ELSE =0	Risk DON≤ 1 indicates risk of DON contamination, Risk DON=0 indicates minor risk of DON contamination	9
$\text{Risk DON}_c = \text{Risk inf}_c$ (IF $20< T_c<35$ AND $L_w=1$) OR Risk inf_c * 7 IF $20< T_c <21$ AND $L_w=1$ ELSE = 0		10

where Risk DON_g is the DON risk index for F.graminearum, Risk DON_c is the DON risk index for F.culmorum.

The hour based Risk DON production indices are added together and accumulated, seen in **equation 11**, making a summarized DON index based on both pathogens.

$$\text{Risk DON}_{tot} = \sum (\text{Risk DON}_g + \text{Risk DON}_c) \quad (11)$$

where DON_{tot} = summarized DON index based on both pathogens

4.5. DON sub models

4.5.1. Phenology model v.1

The phenology module predicts the time for heading and anthesis in spring wheat, and is made by Del Ponte et al. (2005). A couple of adaptions were made to make simulations resemble oat phenology better. The adaptations are based on observations in oats (cultivar Belinda) in a field experiment at Bjertorp in western Sweden in 2012. The module describes the crop development from the first panicle emergence (growth stage 55) (Zadoks et al. 1974) and is illustrated in **figure 3**. The phenology module is constructed in the following way:

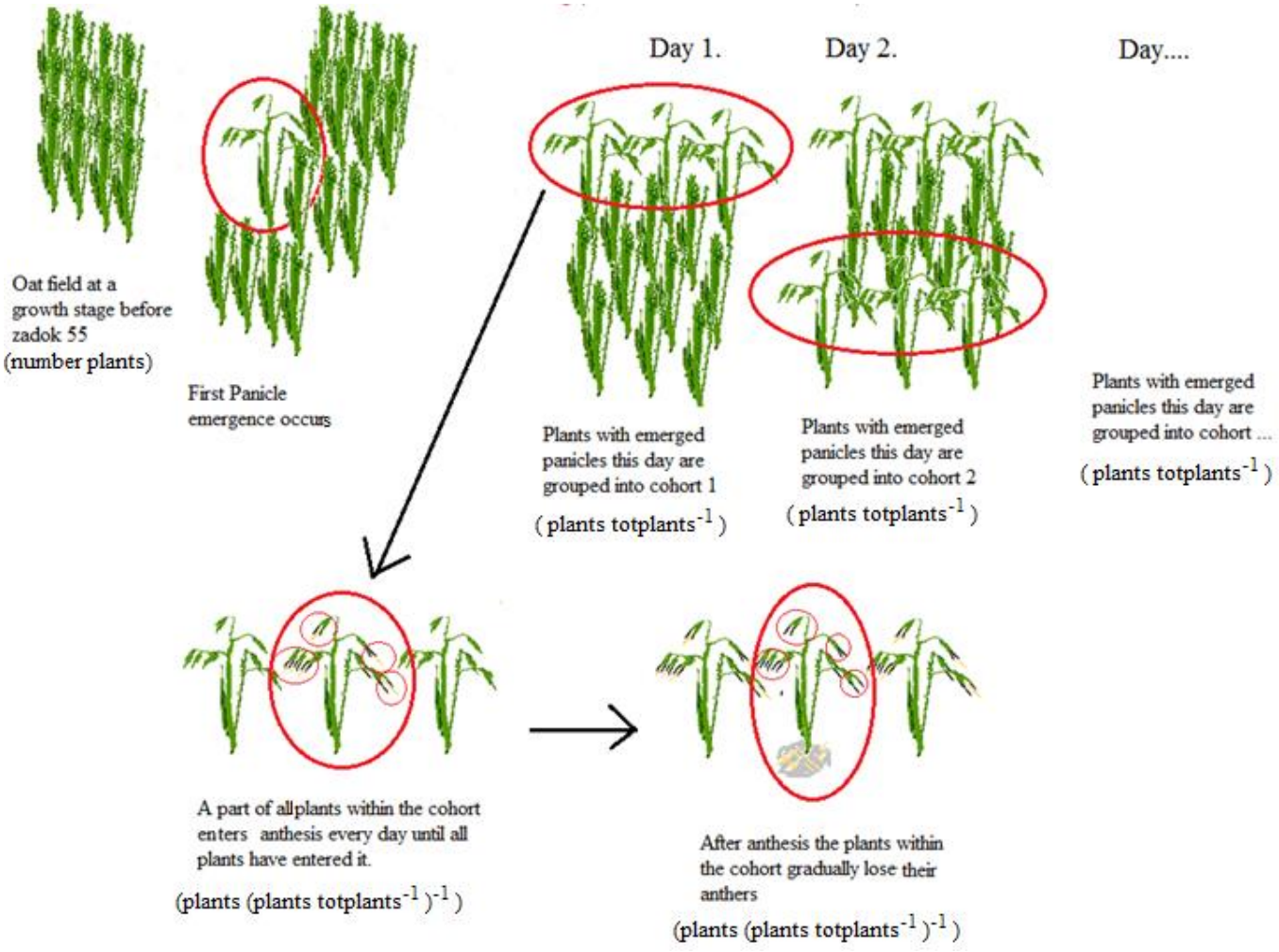


Figure 3, Basic overview of the phenology model`s functions. It starts with a certain number of plants. When the first panicle emergence occurs the plants are divided into cohorts after which day their panicle emerges. Within the cohorts are groups of the plants entering and exiting anthesis every day until all plants have exited anthesis.

- As seen in **figure 3**, the anthesis progression starts when the point of the first panicle is fully emerged (FPE). This occurs 445 day-degrees above 5 °C after sowing.
- Not all plants´ panicles are emerged simultaneously. Therefore they are grouped into cohorts after their panicles` emerging day, as illustrated in **figure 3**, which means that each day after the start of the first panicle emergence (FPE) a new cohort will be formed. The proportion of plants which panicles are emerging under a day (PNG) is regulated by the elapsed time from first panicle emergence (FPE). This is formulated in **equation 12** originated by Del Ponte et al. (2005) who used a similar equation describing the total proportion of plants with emerged panicles per day.

$$PNG=(1^{-(-0.0127(t-1)^{\wedge}2.4352)})-(1^{-(-0.0127t^{\wedge}2.4352)}) \quad 12$$

where PNG = the proportion of plants which panicle is emerged under a day, t = time in days after first panicle emergence FPE.

- The extrusion of anthers (ANText) is the start of anthesis. This starts simultaneously with the first panicle emergence (PNG).
- The proportion of extruding anthers (ANText) in a certain cohort of panicles follows a Weibull function formulated in **equation 13**, illustrated in **figure 3**. The function is regulated by a scaling and shape parameter that responds positively respective negatively to temperature, seen in **equation 14** and **15**.

The following equations (2-4) are taken from Del Ponte et al. (2005).

$$ANText=1^{-(at\hat{}b)} \qquad \sum(ANText-ANTdrop)=ANT \qquad 13$$

$$a=0.255-0.029T_a+0.0009T_a^2 \qquad 14$$

$$b=5.773+0.966T_a-0.0278T_a^2 \qquad 15$$

where ANT = the daily proportion of present anthers, ANText = proportion of extruding anthers, ANTdrop = the proportion of dropping anthers, T_a = ambient temperature in °C

- The anthers are attached to the plant for three days after extrusion. Thereafter they drop (ANTdrop) as seen in **figure 3**, which means that the amount of present daily proportions of anthers per cohort (ANT) is the accumulated sum of the subtraction of extruded and dropped anthers (ANText-ANTdrop) seen in **equation 13**.
- The proportion of susceptible tissue per cohort (ST) during the flowering is set equal to the amount of daily proportions present anthers per cohort (ANT) from the start and through the peak of the anthesis, and until ANT decreases to 0.25, according to **equation 16**.
- After peak flowering, when the amount of daily proportions of present anthers per cohort (ANT) has dropped below 0.25, the amount of proportions susceptible tissue per cohort (ST) is considered as 0.25, described in **equation 17**.
- When the amount of daily proportions of present anthers (ANT) drops below 0.01 and a time period of seven days passes, the amount of susceptible tissue (ST) is considered as 0.1, shown in **equation 18**.
- If the daily proportions of anthers (ANT) values are lower than 0.01 for more than 14 days, the amount of proportion susceptible tissue per cohort is considered to be non-existing, shown in **equation 19**.

$$ST=ANT \qquad \text{IF } ANT>0.25 \qquad 16$$

$$ST=0.25 \qquad \text{IF } ANT<0.25 \text{ AND IF } ANT<0.01 \text{ AND } t<7 \qquad 17$$

$$ST=0.1 \qquad \text{IF } ANT<0.01 \text{ AND } 7<t<14 \qquad 18$$

$$ST=0 \qquad \text{IF } ANT<0.01 \text{ AND } 14<t \qquad 19$$

where ST = susceptible tissue and t = time in days when ANT value is constantly below 0.01.

- The relative total amount of anthers and susceptible tissue (ANTtot & STtot) is calculated by summarizing all cohorts´ daily proportions of present anthers (ANT) and proportions of susceptible tissue (ST), shown in **equation 20**.

$$(ANT)_{1+}\,(ANT)_2+(ANT)_3+\ldots(ANT)_n=ANT_{tot} \qquad (ST)_{1+}\,(ST)_2+(ST)_3+\ldots(ST)_n=ST_{tot} \qquad 20$$

where ANT_{tot} = the relative total amount of anthers, ST_{tot} = the relative total amount of susceptible tissue

4.5.2. Leaf wetness model v.1

The leaf wetness model predicts the presence of leaf wetness per hour in the way of 0 or 1 which means that if there is only one second of leaf wetness the model registers it as there was leaf wetness the entire hour. The model recognizes leaf wetness from rain, fog and dew formation and is formulated in **equation 21** and illustrated in **figure 4** and more thoroughly explained in **Appendix 1**.

The fog component predicts leaf wetness due to fog and depends on relative humidity as seen in **figure 4** and is more described in the section "fog component" in **Appendix 1**.

The rain component predicts the amount of leaf wetness due to rain. This component is, as seen in **figure 4,** depending on the amount of intercepted water after rain storms and how rapidly this is dried up. The amount of interception is in turn controlled by throughfall and canopy capacity factors and amount of precipitation. The drying up process is regulated by evaporation from the dew and evaporation component. The rain component is more detailed described under the "Rain component" section in **Appendix 1**.

The dew component predicts evaporation and leaf wetness due to dew. This component is depending on input data inform of cloud coverage (cloudy), ambient temperature (T_a), global radiation (R_s), relative humidity (RH) and wind speed (u) to operate. This input data is as seen in **figure 4** used in different ways and culminates in the so called energy balance that decides the formation of dew or evaporation. This is much more thoroughly described under the section Dew formation- and evaporation- component in **Appendix 1**.

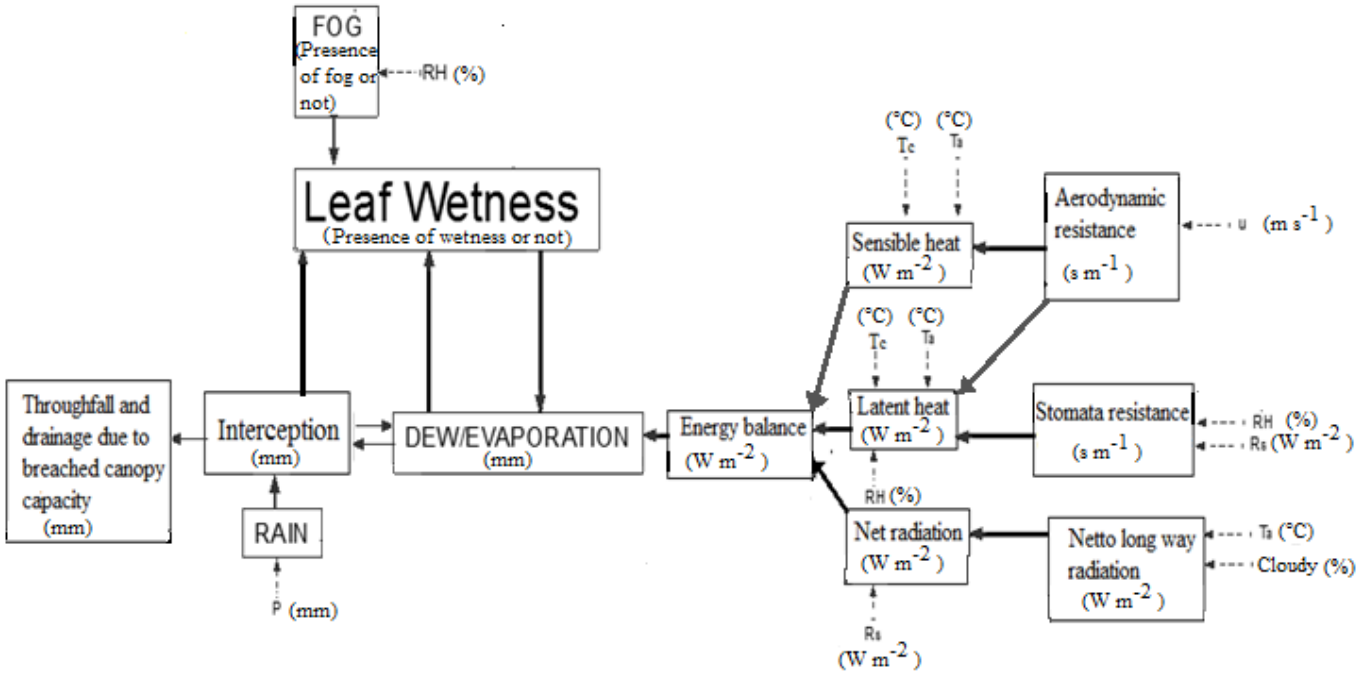


Figure 4. Flow chart of the leaf wetness model. The boxes are process that lead to leaf wetness. The arrows describe how the processes are linked with each other. Doted arrows are input data inform of weather files.

L_w=1 IF (E<0 OR I_{Acc}>0 OR P>0 OR RH≥91) ELSE=0	L_w=1, indicates occurence of leaf wetness. L_w=0 indicates no leaf wetness	21

where L_w = leaf wetness occurrence, E = evaporation (mm h^{-1}), I_{Acc} = accumulated interception (mm) and RH = relative humidity (%)

4.5.2.1. Validation of leaf wetness model

To ensure that the leaf wetness model is simulating leaf wetness values correctly, the model is validated against the instruments measuring the leaf wetness observations. These instruments are leaf wetness probes of the type Model 237 Leaf wetness sensor (Campbell Scientific. Inc 2010). Following modifications in the leaf wetness model have been done to better mimic the leaf wetness sensor.

- The canopy capacity value is set to 0.1mm after calibrating it for the weather data at Ås 2004 to 2008.
- The through fall coefficient is set to 0.5 which is calculated by the deployment of the sensor. The sensor is deployed at a 60° angle leading to the surface is halved when viewed from above compared to when it is deployed flatly.
- The sensor measures 2m above soil surface therefore the crop height is also altered to 2m.
- The latent heat term seen in **equation 11** in **Apendix 1** includes a term for stomata resistance which the sensor does not have. The latent heat term is therefore reformulated according to **equation 22** where there is no term for stomata resistance.

$$LE=((Cp*\rho)(e_{cs}-e_a))/(\gamma*r_a) \quad 22$$

The validation was conducted with two different methods:

- All simulations after the leaf wetness model and the modifications stated above. This method is called **RA1.**
- All simulations after the leaf wetness model, the modifications stated above and the way of calculating the aerodynamic resistance (r_a) is altered. This method is called **RA2.**

In the first validation the RA1 method was applied, and RA2 was applied in the second validation.

The aerodynamic resistance in **RA2** is altered because the one based on **equation 17** in **Appendix 1** is according to Monteith (1965) most suited for a uniform crop surface. The Leaf wetness sensor is not part of a uniform crop surface, it is rather more like a single leaf. There is a more accurate way of calculating the aerodynamic resistance for a single leaf. This is based on the convection around the leaf and differs depending on the size of it. Based on heat transferee calculations from wind tunnel and field experiments on plant leaves by Landsberg & Powell (1973), Monteith (1965), Thom (1968) and Thorpe & Butler (1977) is **equation 23** therefore supposed to provide a more accurate aerodynamic resistance.

$$r_{a2}=2.25\ (d/u)^{2/3} \quad 23$$

where d = leaf length(longest side) (cm).

The simulated values for all locations and years, for respective method are then compared with a matching method against actual leaf wetness instrument readings.

4.6. Alternative modeling applications

Other DON prediction attempts were conducted with following models:

DON prediction models

- DON model
- ENVsim v.1
- ENVsim v.2

Phenology models

- Phenology model v.1
- Phenology model v.2

Leaf wetness models

- Leaf wetness model

Simulations were conducted for all three DON prediction models without phenology input in the following way:

- DON model + Leaf wetness model
- ENV v.1
- ENV v.2+ Leaf wetness model

Simulations were carried out for the two alternative models with phenology input from phenology model v.1, in the following way:

- ENV v.1+ Phenology model v.1
- ENV v.2+ Leaf wetness model + Phenology model v.1

Last of all, all three DON prediction models were used together with phenology inputs from an alternative phenology model, in the following way:

- DON model v.1+ Leaf wetness model + Phenology model v.2
- ENV v.1+ Phenology model v.2
- ENV v.2+ Leaf wetness model + Phenology model v.2

The alternative models are stated and summarized below and are explained more in detail under its corresponding heading further down in this report:

ENV v.1 is a *Fusarium* infection model for wheat by Del Ponte et al. (2005). The model is designed for predicting FHB related infections by *F.graminearum*. However in this thesis the indices are calculated from the model used for predicting DON contamination, which is tightly linked with *Fusarium* infections. The model needs relative humidity, precipitation and ambient air temperature data to operate. To operate under certain growth stages for the crop also input from a phenology model is needed.

ENV v.2 is a modified version of ENV v.1. The model needs leaf wetness and canopy temperature data from a leaf wetness model to operate. To operate under certain growth stages for the crop also input from a phenology model is needed.

Phenology model v.2 is a slightly modified version of Phenology module v.1 with a modification related to an extended flowering period. To operate the model needs input data about ambient air temperature from weather files and sowing date for the crop.

4.7. Alternative DON model "ENV v.1"

The alternative model ENV v.1 illustrated in **figure 5** is developed for spring wheat by Del Ponte et al (2005).

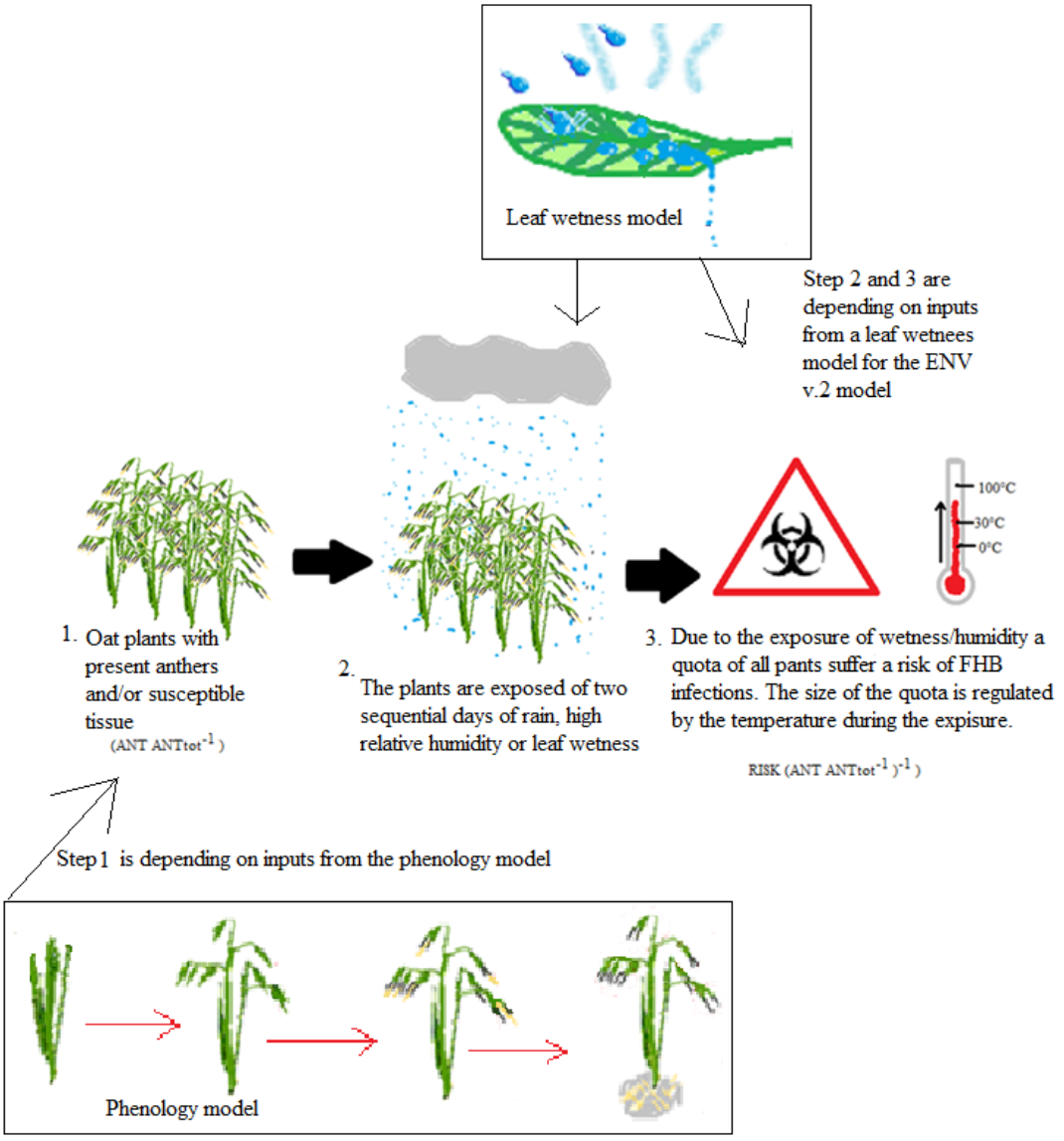


Figure 5. A basic overview of the alternative model applications. When there are simultaneously present susceptible tissue, wet conditions in form rain or high relative humidity and high temperatures. There are an increased risk of FHB infections.

In order for a plant to be infected by *F.graminearum* it needs to carry anthers or to have susceptible tissue, illustrated as the first step in **figure 5**. The knowledge about the plants´ phenology stages is provided by a phenology model that predicts the relative total amount of anthers and susceptible tissue which is used in this model.

The model is working in a two day moving window. If one of the environmental conditions stated below is met, the model registers that a proportion of plants suffer a risk of *F.graminearum* infection:

- Precipitation> 0.3mm and mean daily RH ≥ 80% two days in a row
- Precipitation> 0.3mm and mean daily RH ≥ 80% one day, non-rainy day and mean daily RH ≥85% one day.

The size of the proportion plants risking to be infected is regulated by an infection frequency formula based on the work of Rossi et al. (2001) viewed in **equation 24** and depends on the temperature during the potential infection event. Since only plants that carry anthers or have susceptible tissue can be infected, the relative total amount of anthers and susceptible tissue is multiplied with the infection frequency shown in **equation 25** and **26**. This part of the model is illustrated as the last step in **figure 5**.

The following equations (8-11) are taken from Del Ponte et al. (2005).

$$INF=0.001029^{(0.1957\ T_a)} \quad 24$$

$$ANT*INF=GIB2 \quad 25$$

$$ST*INF=GIB3 \quad 26$$

where INF = the size of the proportion of plants that risk being infected under an infection event, GIB2 and GIB3 = the proportion of anther carrying respective plants with susceptible tissue that risk being infected under an infection event.

Finally an infection risk index over the risk for FHB is calculated. The calculation is done by multiplying the sum of the proportions plants with anthers and susceptible tissue that risk being infected under every infection event (GIB2 & GIB3) with one hundred and then accumulate it, as in **equation 27**.

$$\Sigma((GIB3+GIB2)*100)=GIB \quad 27$$

GIB = FHB risk index

4.8. Alternative DON model "ENV v.2"

This alternative DON model works in the same way as the ENV v.1 model but has the following modifications:

- The precipitations and humidity requirements, seen as step two in **figure** 5, were replaced with minimum 12 hours of leaf wetness during a 24 hours´ period, which means that in order for an infection to be registered, 12 hours of leaf wetness per day for minimum two sequential days are necessary.

The temperature requirements are originally given in ambient air temperature (T_a), (step 3 in **figure 5)**. In this modified version the canopy temperature (T_c) is used instead, since the infection

occurs on the canopy surface. The canopy temperatures (T_c) are imported from the leaf wetness model described further down.

4.9. Alternative sub model Phenology model v.2

This phenology model functions in the same way as the phenology model v.1 apart from one point. This model simulates an oat cultivar, which anthers do not drop after flowering. Therefore the anthers are not dropped 3 days after extrusion in this model.

4.10. Statistical methodology

4.10.1. Comparison between simulated and measured leaf wetness values

The simulated leaf wetness values are compared towards their corresponding measured leaf wetness value in the following way:

- The measured leaf wetness values are processed so that the presence of leaf wetness per hour is registered in the 0 and 1 method like the simulated values. Meaning that although a specific hour only has one minute of measured leaf wetness, it is registered as if the leaf wetness occurred the entire hour.
- The simulated and their corresponding measured values are compared and the number of matching and non-matching results are registered.
- The comparison is redone for the model´s individual components, rain, fog and dew, in order to estimate their contribution to the model.

4.10.2. Alternative aerodynamic resistance calculations

The results for the two leaf wetness modeling methods using alternative aerodynamic resistance (explained in section "**4.5.2.1. Validation of leaf wetness model**", involving **equation 23** and in **Apendix 1 equation 11**) are compared by a two tailed paired T-test to find a significant difference.

- The ratio for matching leaf wetness is calculated for both methods between simulated and measured values for each simulation.
- The difference for the ratios between the two methods (d) is calculated according to **equation 28**.
- The variation between the methods (S_d^2) is calculated. This is done by:

Step 1. Summarize the differences as squared values (Σd^2).

Step 2. Subtract the squared difference per simulation ($(\Sigma d)^2/n$) from the calculated sum ($\Sigma d^2 - ((\Sigma d)^2/n)$).

Step3. Divide the calculated sum with the number of simulations minus one (n-1). All steps are given by **equation 29**.

- A score (z) for the comparison is calculated according to **equation 30**. In the denominator the standard error is calculated by calculating the root of the variation and the number of simulations ($(S_d^2/n)^{0.5}$). In the numerator the difference between the mean values for each method is subtracted ($\mu_1-\mu_2$) from the median difference ($\bar{d}$).

To summarize the t-test method you can say it scores (z) the amount of variation between two samples, where the score (z) of one points stands for the absolute smallest variation. In this comparison, it is thought that one of the methods is better than the other (H_1: $\mu_1 \neq \mu_2$) leading to the null hypothesis that they are both equally good (H_0: $\mu_1 = \mu_2$), formulated in **equation 32**. The null hypothesis (H_0: $\mu_1 = \mu_2$) is then tested since it is easier, making the difference between the mean values for the methods ($\mu_1-\mu_2$) becoming 0.

To determine if the hypothesis is correct **equation 31** is used. The equation describes the probability that the differences of the comparison are a random accident. If the score turns out to be higher than the equation, the comparison is called to show a significant difference meaning differences are less probable to be due to randomness. The necessary scores for defining the comparison significant depends on how certain the probability is supposed to be and can be found in statistical tables for determining significance. In this thesis a table in Olsson et al. (2005) p281 has been used, with the probability set to 99,5%.

The t-test is rather tricky to explain shortly in a master thesis and those wishing to understand more are recommended to read Olsson et al. (2005).

$$d = x1 - x2 \quad (28)$$

$$S_d^2 = (\Sigma d^2 - ((\Sigma d)^2/n))/(n-1) \quad (29)$$

$$z = (\bar{d}-(\mu_1-\mu_2)/(S_d^2/n)^{0.5} \quad (30)$$

$$z>z_{(1-a/2|n-1)} \quad (31)$$

$$H_1: \mu_1 \neq \mu_2 \quad \rightarrow \quad H_0: \mu_1 = \mu_2 \quad (32)$$

Where x = leaf wetness matching ratio for respective method and simulation, d = difference for leaf wetness matching ratio for each simulation between the methods, n = number of simulations, S_d^2 = variation between the two methods leaf wetness matching ratio, μ = mean leaf wetness matching ratio for respective method, $\bar{d}$ = the median difference for leaf wetness matching ratio between the two methods, z = amount of t-test scores, a = probability, H = hypothesis

4.10.3. Regression and correlation analysis

The indices from the models are plotted with their corresponding measured DON values by regression and correlation analyses. The regression analysis calculates a pattern among the plotted values. The kind of pattern it calculates follows a straight line function also called y = kx+m pattern. This function is calculated in the following way:

- Calculate the difference from the summed squared indices values ($\sum x_i^2$) and the squared sum of indices divided with the number of simulations ($(\sum x_i)^2/n$), (SS_x), formulated in **equation 33**.
- This difference (SS_x) is then divided with the difference of the summed product of both measured and index values ($\sum x_i y_i$) and the product between summed measured vales and index values divided with amount of simulations($((\sum x_i)*(\sum y_i))/n$), ($SP_x$), as formulated in equation and **equation 34**.
- This quota (β_1) is the inclination factor of the regression equation seen in **equation 35** and **equation 37**.
- The difference between the mean measured value (y_i) and the product of inclination factor (β_1) and the mean index value (x_i) is how much of measured values there are when the index is zero (β_0), formulated in **equation 36**.

$$SS_x = \sum x_i^2 - (\sum x_i)^2/n \quad (33)$$

$$SP_{xy} = \sum x_i y_i - ((\sum x_i)*(\sum y_i))/n \quad (34)$$

$$\beta_1 = SP_{xy} / SS_x \quad (35)$$

$$\beta_0 = \bar{y} - \beta_1 \bar{x} \quad (36)$$

$$y_i = \beta_0 + \beta_1 * x_i \quad (37)$$

where β_0 = primary expected value, β_1 = the slope of the function, the x_i values = indices values, the y_i values = measured DON values, $\bar{x}$ = mean index value, $\bar{y}$ = mean measured value, SP_{xy}, SS_x = different kinds of sums related to the x_i and y_i values, n = the number of simulations

The data points will not always be fully aligned with the calculated regression line. That is because the regression analysis is not perfectly consistent with the plotted values. In other words there is not a perfect correlation. To find out the correlation a so called R^2 value is calculated.

This is done by calculating the squared quota of the SP_{xy} from **equation 34** and the root of the product of the SS_x factor from **equation 33** and the SS_y factor which is principally equal the SS_x factor but is based on the measured values y_i, all formulated in **equation 38** and **equation 39**.

More detailed explanation about the regression and correlation methods are found in Olsson et al. (2005) p 235-252.

$$SS_y = \sum y_i^2 - (\sum y_i)^2/n \quad (38)$$

$$R^2=(SP_{xy} / \sqrt{(SS_x SS_y)})^2 \quad (39)$$

where R = the correlation coefficient, SP_{xy}, SS_x and SS_y = different kinds of sums related to the x_i and y_i values, n = the number of simulations.

5. Results

5.1. The DON model predictions

The results from the DON modeling are presented in the following subsections.

5.1.1. Visualization of simulations

The DON model provides output in the form of the following information:

- When infectious matter is present
- When there is infectious matter present and for a sufficiently long period with suitable environmental conditions for an infection to occur
- When environmental conditions for DON production are suitable
- When susceptible tissue and extruded anthers are present

The bullets above are visualized in the two **figures 7** and **8**. Both pictures are from the same region, same cultivar (cv Belinda) but different dates and sowing times. One can see in the figures that there are many occasions of present infectious matter and sometimes there are even occasions suitable for infections but the infection is not registered if there are not any anthers and or susceptible tissue. In the red encircled areas all requirements necessary for the model to register a potential infection are fulfilled. When a potential infection has been registered the model enables the possibility of DON production to occur, and those events can be seen after the red encirclements.

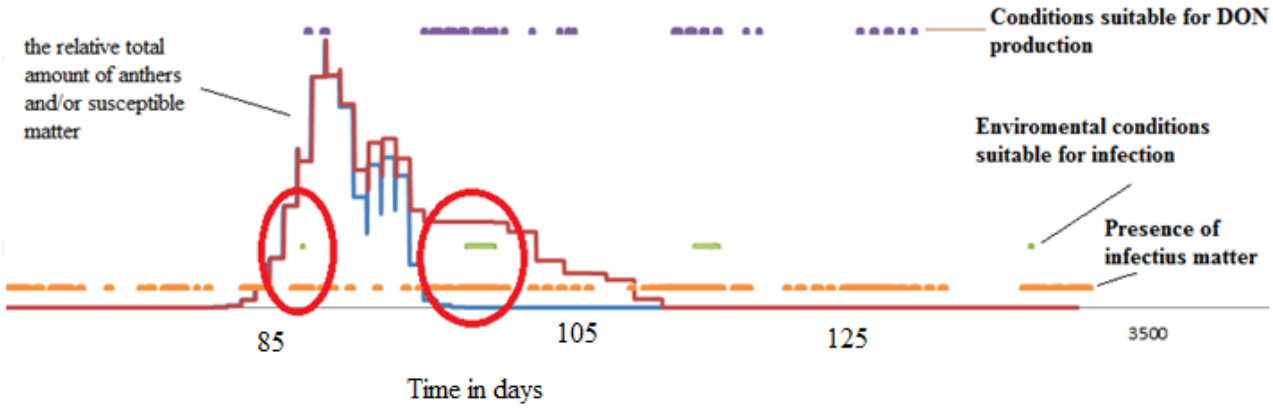


Figure 7, Visualized simulation of the Belinda cultivar at a sample site in Roverud in 2004 with the sowing date14th of June and the harvest date 10th of October. The index value produced for the simulation is 502.29.. The yellow lines indicate presence of infectious matter, the green lines indicate presence of infectious matter and for a sufficient long period suitable environmental conditions for infection, the purple lines indicate suitable conditions for DON production, the red line indicate the relative presence of anthers, the blue line indicate relative presence of susceptible mater and the red encircled areas are point of time when the model registers a potential infection (Note that the time line does not start from 0).

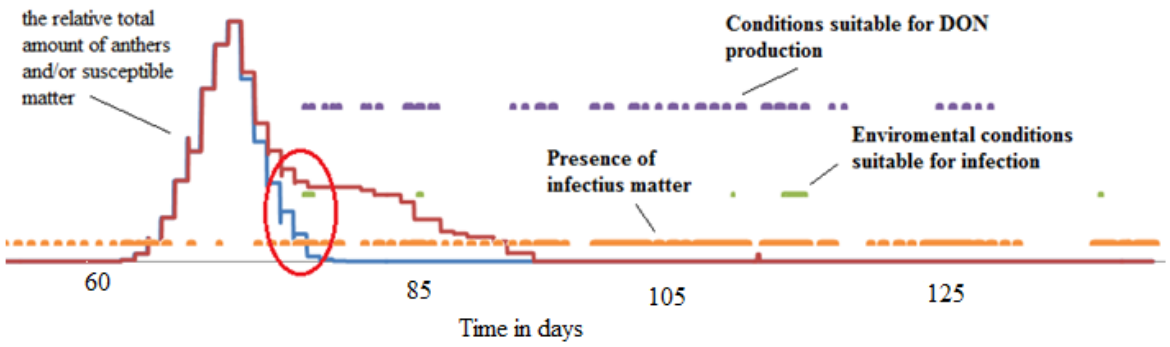


Figure 9, Visualized simulation of the Belinda cultivar at a sample site in Roverud 2006 with the sowing date 14th of June and the harvest date 19th of August. The index value produced for the simulation is 349.26. The yellow lines indicate presence of infectious matter, the green lines indicate presence of infectious matter and for a sufficient long period suitable environmental conditions for infection, the purple lines indicate suitable conditions for DON production, the red line indicate the relative presence of anthers, the blue line indicate relative presence of susceptible mater and the red encircled areas are point of time when the model registers a potential infection. (Note that the time line does not start from 0.)

5.1.2. Regression and correlation analysis

The regression and correlation results from the DON modeling turned out to show a small but negative correlation of R^2=0.044 (where R^2 close to 1 means perfect correlation meanwhile R^2 close to 0 means randomness) for the Belinda cultivar at all locations and years in Norway, seen in **figure 9**.

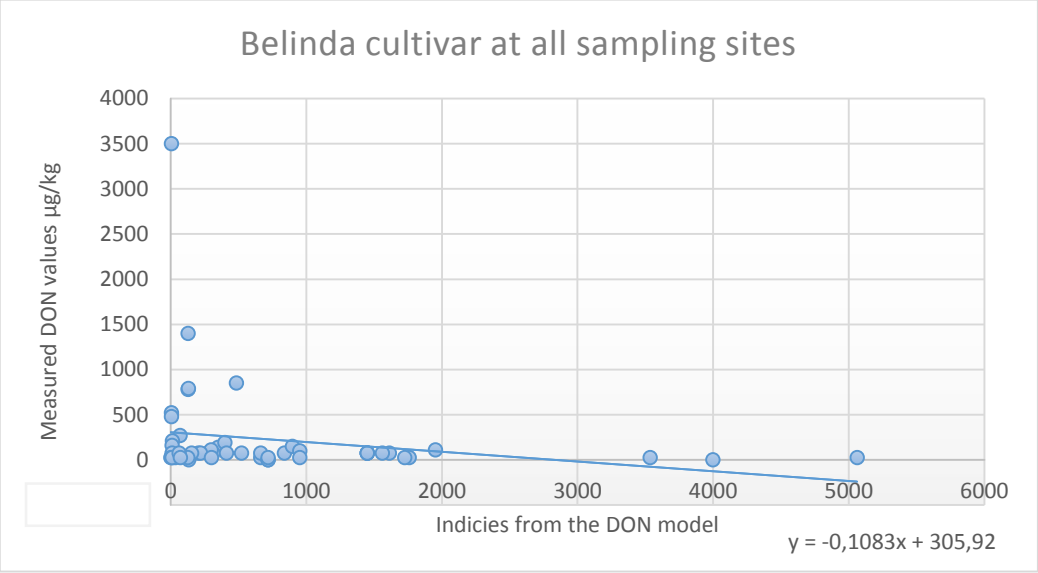


Figure 9. Showing the measured DON values plotted against their corresponding model indices for all Belinda cultivar at all sampling sites for all years. The regression analysis is shown as a trend line and its corresponding regression equation is shown in the lower right corner.

When scaling down to regional scale results from Tjølling and Hokksund, are lacking because those locations contained too few (less than three) measured DON samples for making any regression and correlation analysis. Strong correlations were found for the Belinda cultivar for all years in Gvarv and Øsaker (R^2=0.925 respective R^2=0.675), but there are only three samples from each location and the correlation found in Øsaker was negative.

For all years in Rakkestad a negative correlation of R^2=0.407 was found. It can be seen in **figure 10** that although the measurements reveal low DON levels the model registers them as more potentially contaminated then the measured samples that actually contain elevated levels of DON.

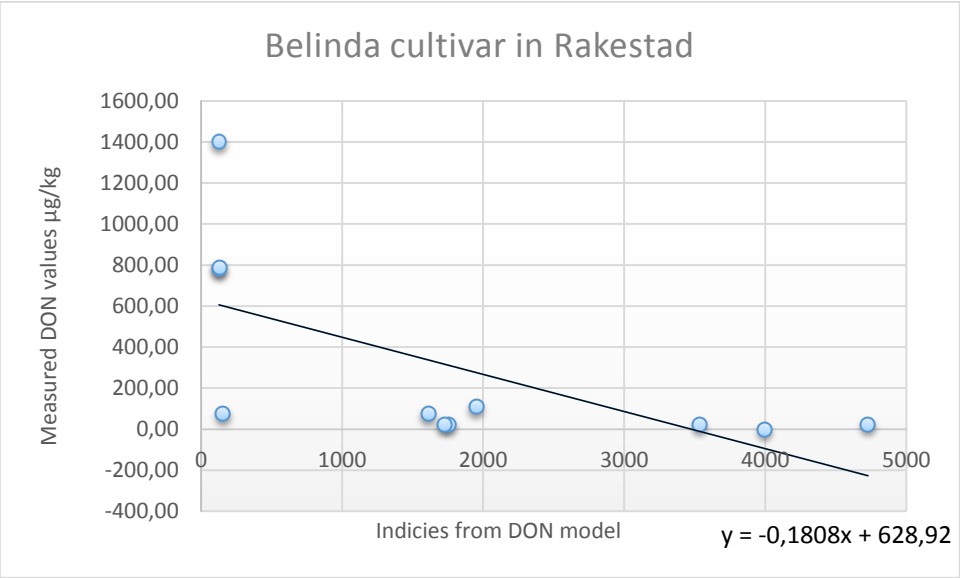


Figure 10. Showing the measured DON values plotted against their corresponding model indices for all Belinda cultivar samples in Rakkestad for all years. The regression analysis is shown as a trend line and its corresponding regression equation is shown in the lower right corner.

When all years in Rakkestad were included no correlation R^2=0.011 was found for the Bessin cultivar.

When including all years in Roverud a strong positive correlation of R^2=0.86 was found for the Belinda cultivar, seen in **figure 11**.

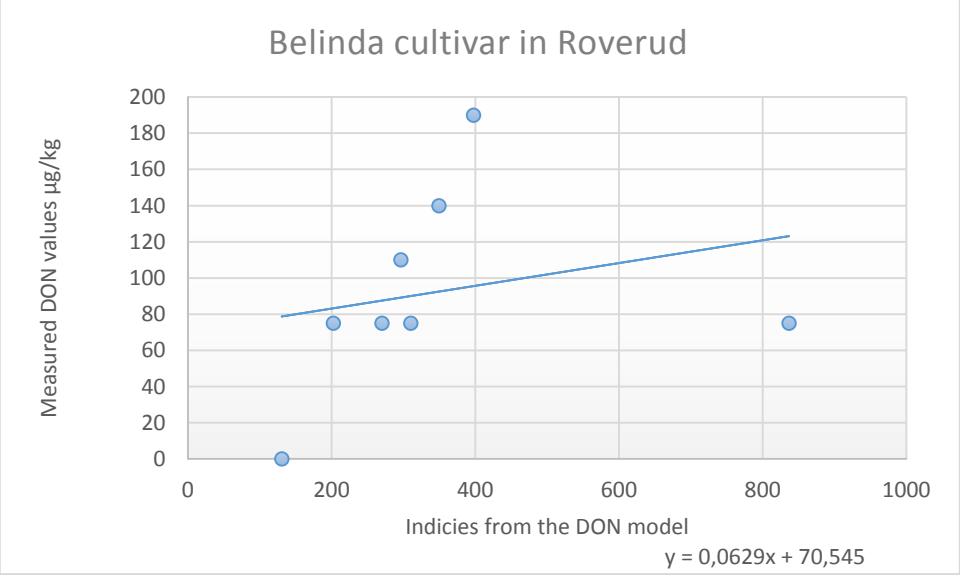


Figure 11. Showing the measured DON values plotted against their corresponding model indices for all Belinda cultivar samples in Roverud for all years. The regression analysis is shown as a trend line and its corresponding regression equation is shown in the lower right corner.

Including all years in Ramnes resulted in a weak positive correlation of R^2=0.308 for the Belinda cultivar. When examining **figure 12**, one outlier encircled with red seems to lower the correlation. When removing the outlined measurement in the red circle the correlation increases to R^2=0.979.

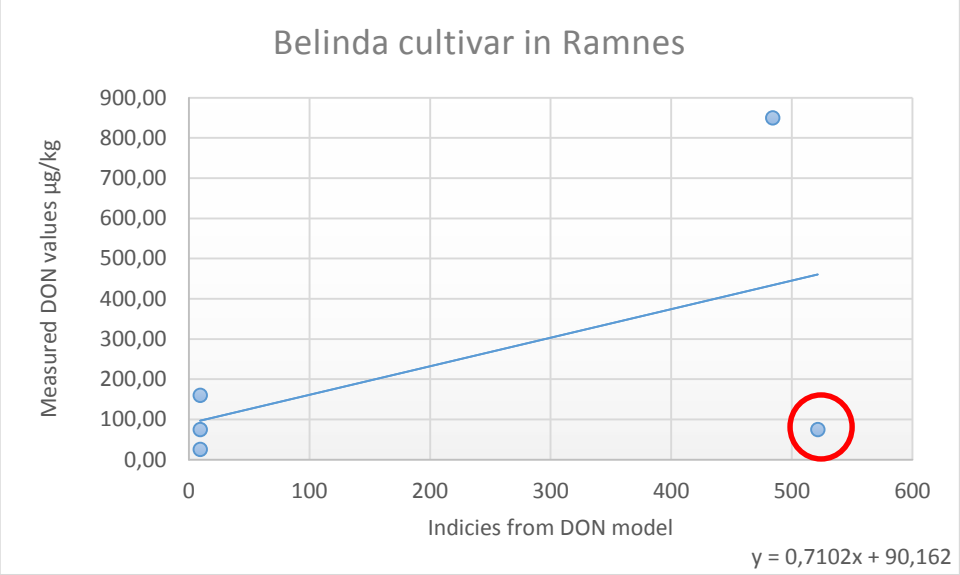


Figure 12. Showing the measured DON values plotted against their corresponding model indices for all Belinda cultivar samples in Ramnes for all years. The regression analysis is shown as a trend line and its corresponding regression equation is shown in the lower right corner. The sample encircled with red is counted as an outlier.

When all years in Ås were included a weak correlation of R^2=0.302 was found for the Belinda cultivar. The correlation increases to 0.58 when removing three outliers seen in **figure 13**.

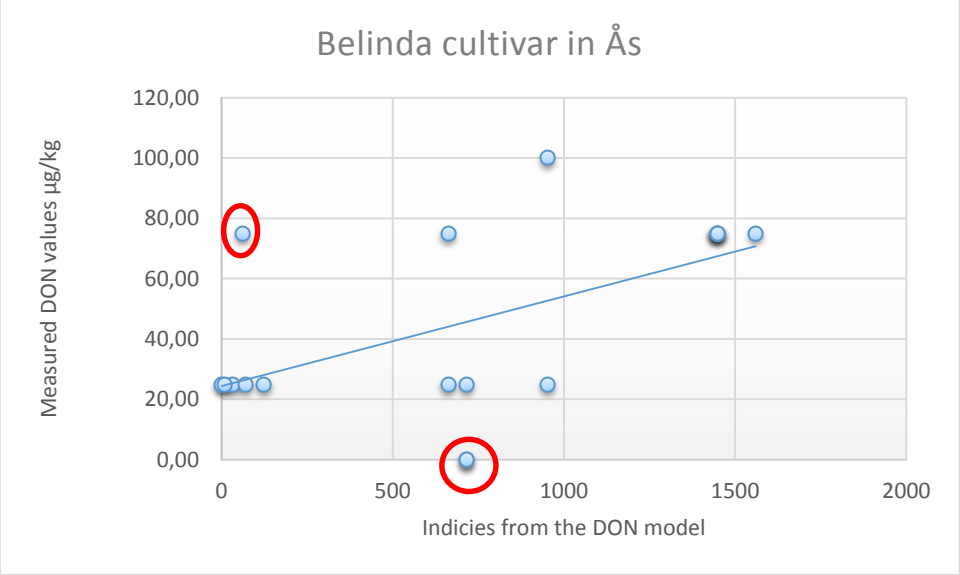


Figure 13. Showing the measured DON values plotted against their corresponding model indices for all Belinda cultivar samples in Ås for all years. The regression analysis is shown as a trend line and its corresponding regression equation is shown in the lower right corner. The samples encircled with red are counted as outliers.

5.1.3. Leaf wetness validation

The leaf wetness validation resulted in a high matching frequency between the estimated leaf wetness hours from the models and actual leaf wetness hours from the sensor. The results are summarized in **table 5**. The RA2 method scored highest frequency between estimated and actual leaf wetness hours.

The RA2 method estimates dew with a significant higher matching ratio between registered and matched leaf wetness with a z value on 3.234 than the RA1 method. Still the final difference between the results for the two methods is small and the T-test shows an insignificant z value on -0.96311. The rain module does differ when comparing the two methods, but the difference is not significant with a z value on 0.18, meaning that the RA2 method matches better than the RA1 method, but it cannot be proven on a statistical level.

Method	Simulation time (h)	Number of matches	Matches in percent of simulation time (%)	Ratio between registered and matched leaf wetness due to **dew**	Ratio between registered and matched leaf wetness due to **rain**	Ratio between registered and matched leaf wetness due to **fog**
RA1	81880	70925	86.62	8855/11288	13628/16093	15141/18832
RA2	81880	71198	86.95	7610/9142	13596/15960	15141/18832

Table 5. The results from the leaf wetness model validation towards the leaf wetness sensor. The results are both for method RA1 and RA2 and the individual components in the model.

5.2. Alternative modeling applications

For the alternative modeling strategies the strongest correlation between all measured DON values and their corresponding indices was R^2= 0.0255 for the ENV V.2 model coupled with the Phenology model V.1.

When scaling down to regional scale results from Tjølling and Hokksund, are lacking because those locations contained too few (less than three) measured DON samples for making any regression and correlation analysis.

Strong negative correlations ranging R^2=0.682 to R^2=0.925 were found for the Belinda cultivar Øsaker for all years and all alternative model applications except for ENV models coupled with the Phenology V.2 model. It is important to note that there are only three samples from Øsaker.

For all years in Gvarv a strong correlation was found R^2=0.725 and R^2=0.945 for the ENV V.1 respective ENV V.2 models coupled with the Phenology V.1 model. The other alternative model applications yielded inferior correlations ranging between R^2=0.61 and R^2=0.023. It is important to note that there are only three samples from Gvarv.

In Roverud a weak correlation was found R^2=0.534 for the ENV V.2 model coupled with the Phenology model V.2. The other alternative model applications yielded inferior correlations ranging between R^2=0.001 and R^2=0.4254. When studying **figure 14**, it seems like the outlier encircled in red lowers the correlation. When the outlier is removed the correlation increases to R^2=0.676.

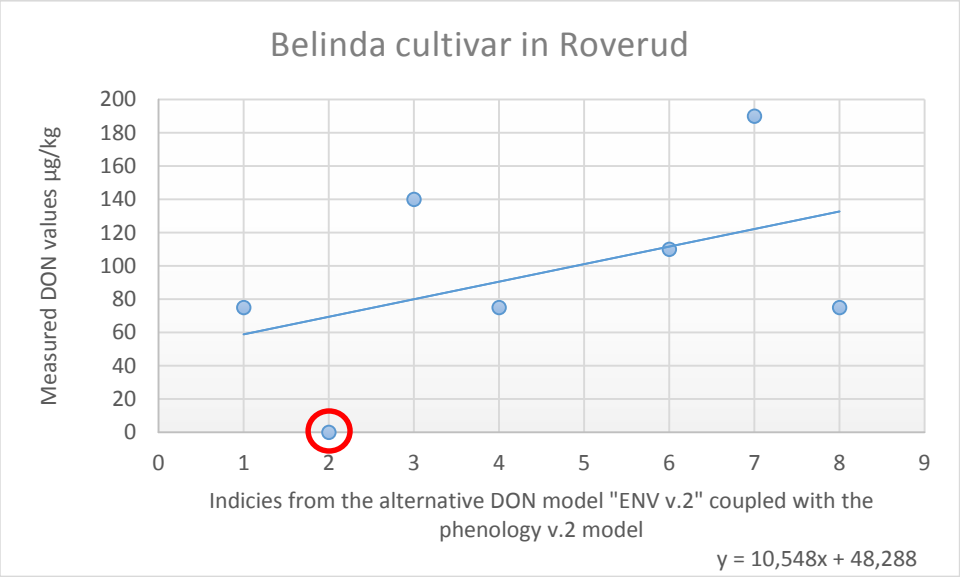


Figure 14. Showing the measured DON values plotted against their corresponding model indices for all Belinda cultivar samples in Roverud for all years. The regression analysis is shown as a trend line and its corresponding regression equation is shown in the lower right corner. The sample encircled with red is counted as an outlier.

For all years in Ramnes weak correlations were found R^2=0.394 and R^2=0.371 for the ENV V.1 respective ENV V.2 model coupled with the Phenology V.1 model. When studying **figure 15** it seems like the outliers encircled in red lowers the correlation. When the outliers are removed the correlation increases to R^2=0.979 for both model couplings. The other alternative model applications yielded inferior correlations range between R^2= 0.024 and R^2= 0.333.

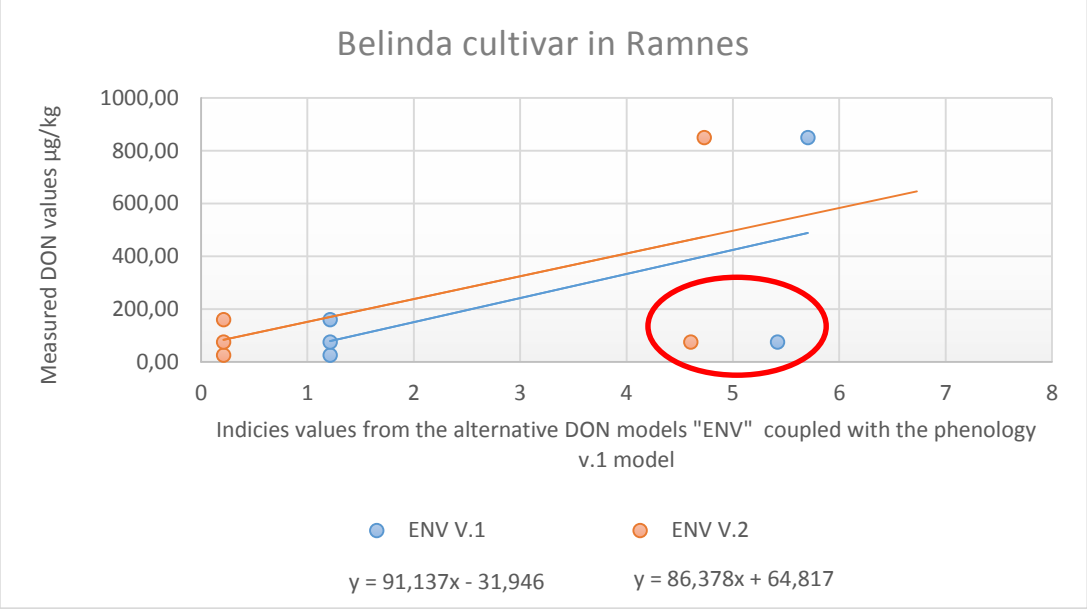


Figure 15. Showing the measured DON values plotted against their corresponding model indices for all Belinda cultivar samples in Ramnes for all years. The regression analysis is shown as a trend line and its corresponding regression equation is shown below the corresponding name of the model. The samples encircled with red are counted as outliers.

For the Belinda cultivar for all years in Rakkestad and Ås and also the Bessin cultivar for all years in Rakkestad no correlation of level worth mentioning were found for any model application.

The results from the alternative modeling applications are summarized in **table 6**. In the table the correlations in form of R^2 values between measured DON values and their corresponding model indices values for all cultivars, regions and years are mentioned.

Region and cultivar	DON model + Phenology V.2	DON model	ENV V.2 +Phenology V.2	ENV V.1 + Phenology V.2	ENV V.1	ENV V.2	ENV V.1 + Phenology V.1	ENV V.2 Phenology V.2
Belinda cultivar in Rakkestad	0.409	0.523	0.211	0.319	0.125	0.360	0.187	0.195

Belinda cultivar in Ramnes	0.211	0.333	0.024	0.027	0.295	0.332	0.394	0.371
Belinda cultivar in Øsaker	0.833	0.742	0.001	0.143	0.718	0.925	0.682	0.699
Belinda cultivar in Gvarv	0.61	0.023	0.050	0.201	0.102	0.591	0.725	0.945
Belinda cultivar in Roverud	0.021	0.091	0.534	0.001	0.003	0.34	0.425	0.081
Bessin cultivar I Rakkestad	0.028	0.004	0.260	0.188	0.119	0.111	0.169	0.119

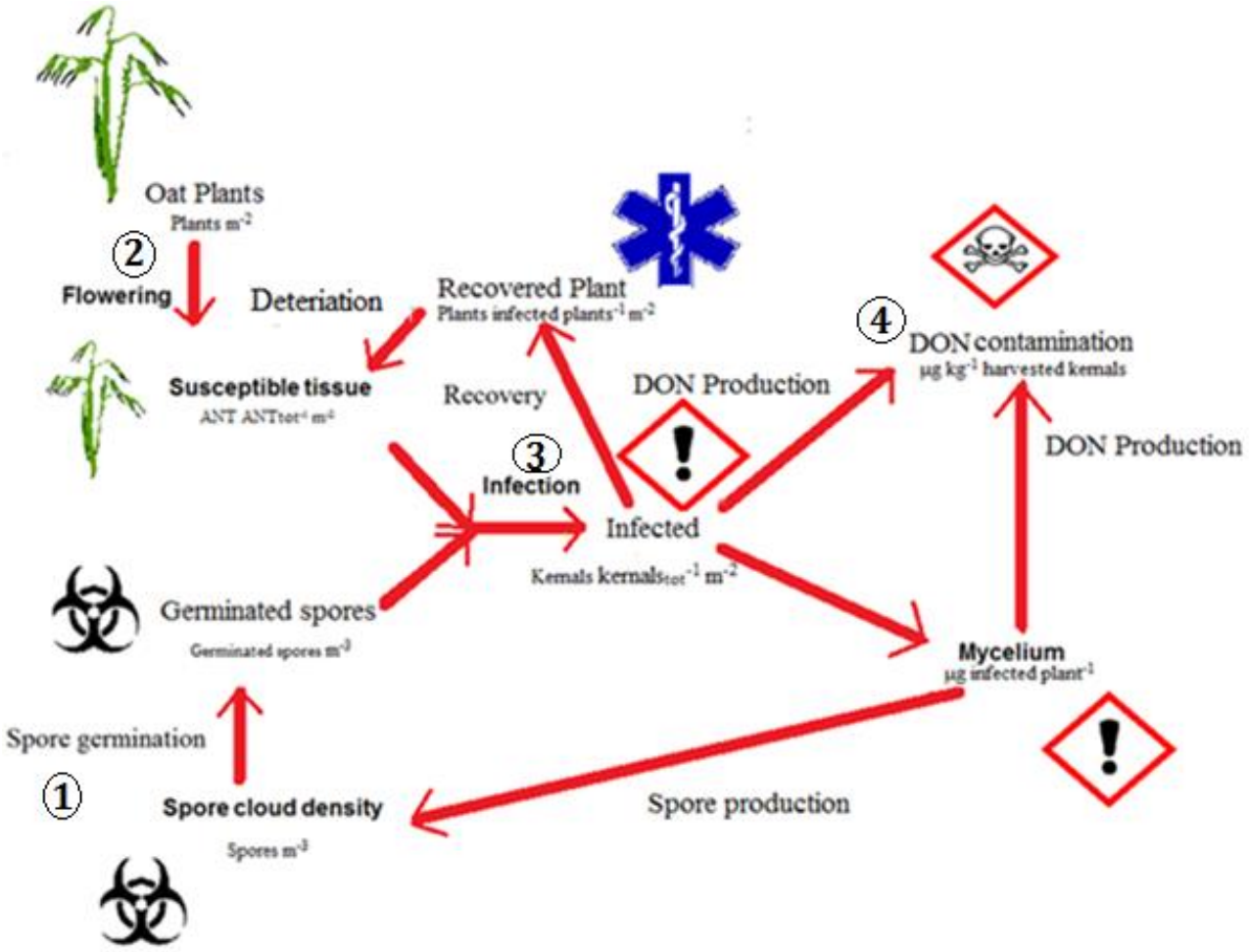
Table 1. R^2 values for the all model couplings in all regions and cultivars.

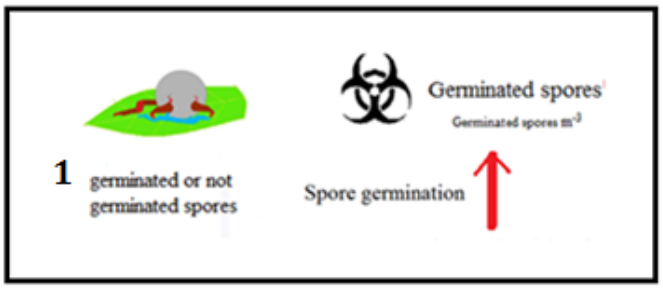
6. Discussion

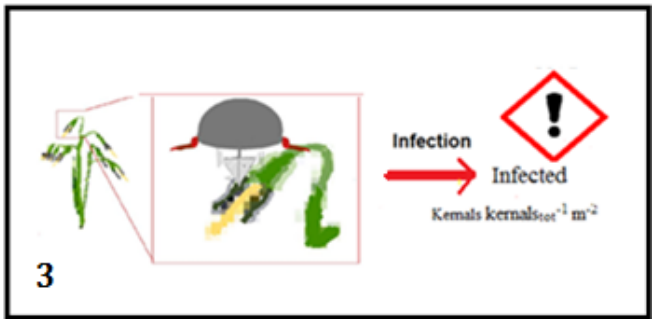
6.1 The DON model, potential errors

The results from the DON model indicate that it was not possible to predict the presence of DON in oat with the models and observations included in this thesis. Although there were promising results in Gvarv, Roverud and Ramnes, the model clearly failed to estimate DON presence in Øsaker, Rakkestad and Ås. Also the positive results from Gvarv, Roverud and Ramnes contain too few samples in order to draw any statistically firm conclusions. The reasons why the main modeling strategy failed to estimate DON are many and it is not known what error sources contributed the most but some of them are presented below and discussed in the following subsections:

- **Errors with the DON model.** The current DON model does not take into account: the plant`s capability to recover from the disease, incubation periods during infection, the influence of Mycelia growth on DON production and the lack of infectious spores. Also, the model assumes that oat responses in the same manner as wheat when exposed to *F.graminearum* and *F.culmorum* which might be incorrect.
- **Errors with the leaf wetness model**. The model has a high accuracy when validated towards the leaf wetness sensors, but the model does not necessary need to be highly accurate towards the oat crops.
- **Errors with the phenology model**. The phenology model is still based on previous studies on wheat and might not be compatible for oat.


Oat Plants
Plants m-2
2
Flowering
Deteriation
Recovered Plant
Plants infected plants-1 m-2
Susceptible tissue
ANT ANTtot-1 m-2
Recovery
DON Production
4
DON contamination
µg kg-1 harvested kernals
3
Infection
Infected
Kernals kernalstot-1 m-2
DON Production
Germinated spores
Germinated spores m-3
Mycelium
µg infected plant-1
Spore germination
1
Spore cloud density
Spores m-3
Spore production


1
germinated or not
germinated spores
Germinated spores
Germinated spores m-3
Spore germination


Infection
Infected
Kernals kernalstot-1 m-2
3

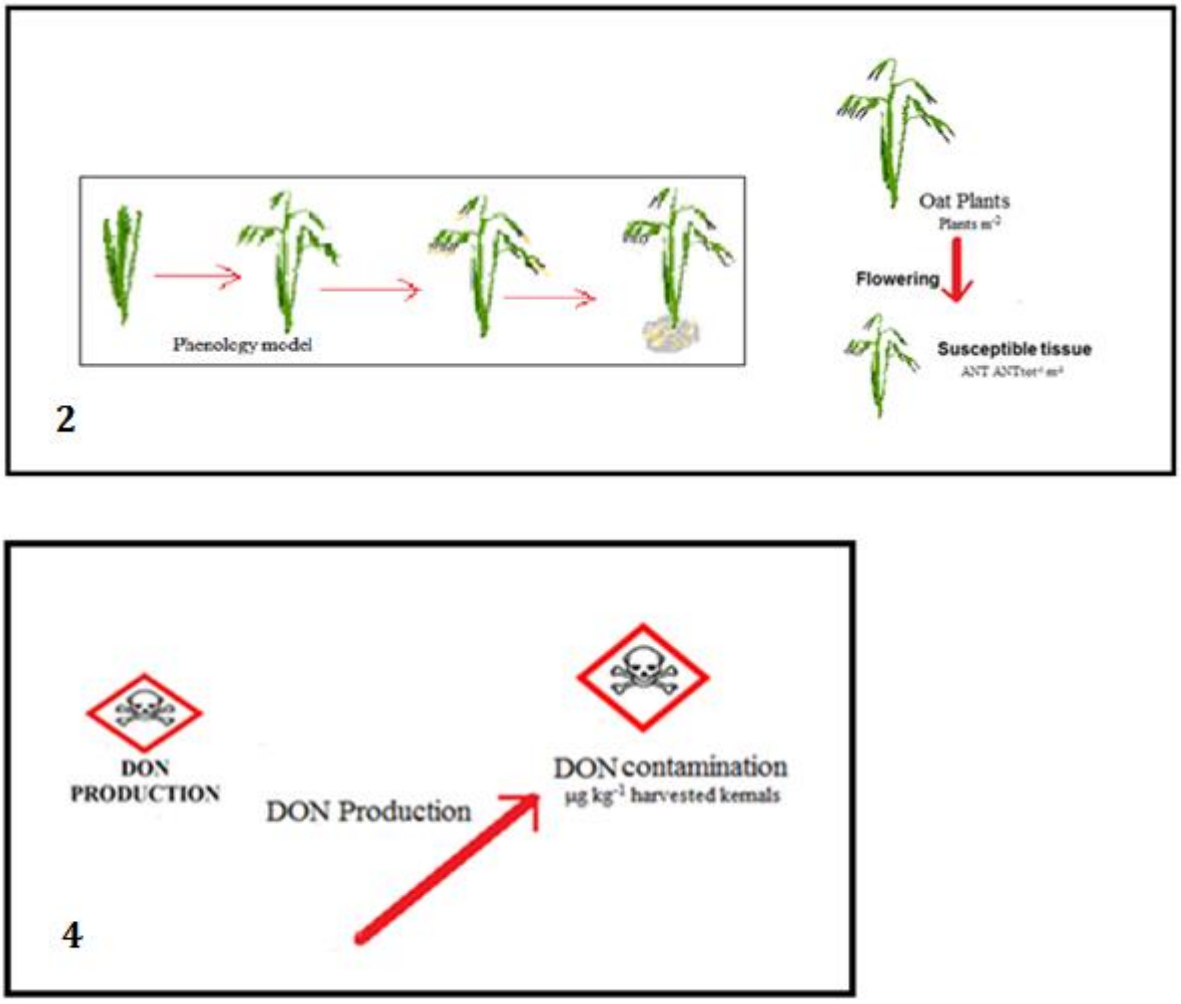


Figure 16 ***On the top basic overview of essential needs for an FHB infection and the possible outcomes of an infection, explanations to that part of the figure are found in the description for figure 1. The Boxes are similarities between the top of the figure with the DON model. Explanations for the DON model figures are found in the description for figure 2.***

6.1.2. DON model uncertainties

When studying **figure 16** which illustrates a comparison between the essential needs for an DON producing FHB infection from **figure 1** with the steps from the DON model in **figure 2** one can see that the DON model has omitted some parts for an DON producing infection. Those parts omitted are the spore cloud density, infection process, recovery process, the recovered plants, the deterioration process after recovery and the mycelium influence on DON production.

6.1.2.1. Are there any spores present that are capable to infect?

When examining the figures in the result section for the main modeling strategy it looks like there actually were high risk regions, but there were still no infections. As it was for the Belinda cultivar in Rakkestad where some of the highest indices are corresponding to some of the lowest

measured DON values. This makes me wonder if there were any spores present and capable to infect in this region?

Plant pathogens like human pathogens often need more than just one piece of infectious matter to start an infection (Cohen 1977, Komiya et al. 2008). It is hypothetically possible that one piece of infectious matter can infect the plant, but most probably it will fail since it is "not so simple" for the pathogen to infect its host. There are many factors influencing an infection procedure, like environmental conditions, host susceptibility, competition from other microbes, activation of pathogenicity, activation of host target susceptibility, density of pathogens (quorum sensing (strategy discussion among pathogens)), entry point of infection etc.

In **figure 16** there is a factor for spore densities. This factor is not included in the simulations with the DON model. The simulations assume that there are enough spores to infect the plant and this might be incorrect in many cases. Therefore it is suggested that the strategy is enhanced with a spore density model which provides values of the spore density.

Both Rossi et al. (2003) and Del Ponte et al. (2005) have created spore models but they differ a bit. Rossi et al. ´s (2003) spore model is designed for *F. graminearum, F. culmorum, G. avenacea* and *M. nivalis*. Del Ponte et al. ´s (2005) spore model is only designed for *F.graminearum*. Also Rossi et al. ´s (2003) model is based on mathematical relations regarding environmental factors that are favorable for sporulation, meanwhile Del Ponte et al. (2005) module is based on infected crop residues in surrounding environment from previous crop season. For developing a spore model a combination of these two previous models might be the best alternative, since there is a need of both crop residues and favourable environmental conditions for sporulation.

To know at which spore densities infection is possible and the actual importance of a spore model it is important to know the minimum required infectious dose. This minimum infection dose can be determined by testing how oat plants responds toward different densities of *Fusarium* spores and see how many plants get sick at different densities and at which density only one plant gets sick. This kind of test would also answer the question "can oat plants get infected by *Fusarium* if only the spore density is enough high regardless of the environmental conditions?".

If the minimum required infectious dose turns out to be very low it might be possible to presume that enough infectious matter is always present and a sporulation model is not necessary. Alternatively, a very high density of infectious mater is needed and then perhaps the sporulation model is even more important than the other models. In other words the DON model might need to compensate for spore density.

6.1.2.2 Disease recovery

The DON model does not consider that a plant can recover from a FHB infection and therefore it misses the recovery process, the amount recovered plants and the deterioration process found in **figure 16**.

The model assumes that if a plant is infected, it will keep being infected until harvest, but this is not completely true. Depending on the defense genes in the plant, the pathogenic genes in the pathogen and the surrounding environment, there always exists a possibility of recovery. Some of the recovery mechanisms might be an activation of systemic defense genes, aborting infected tillers or negative competition between the pathogen and other microbes.

The *F.graminearum* and *F.culmorum* pathogens are of the type necrotrophic meaning that they live on dead host tissue. The plant often responds to an infection by ordering infected cells and their closest neighbouring cells to activate apoptosis mechanisms, meaning that they conduct suicide. This defensive strategy is controlled by the so called "SA defensive system". However this kind of defense does not work on necrotrophic pathogens it only makes the situation worse for the plant, since it supplies the pathogen with dead cells. The plant must activate the so called "JA- ET defensive systems" to hinder the necrotrophic pathogens. This system makes attacked cells produce antibiotic products like phytolaxins which are toxic to the pathogens, it makes the cells produce a denser cell wall hindering the pathogen to kill it and it makes the plant stop conducting cell apoptosis (Glazebrook 2005). In the specific case with FHB it has been found in wheat that the JA-ET system enhances resistance towards *F.graminearum* (Li & Yen 2008, Gottwald et al. 2012, Makandar 2012). To activate the "JA- ET defensive systems" the plant either needs to recognize an ongoing necrotrophic pathogen attack (which it does not always do) or get damaged or attacked by an insect (Dodds & Rathjen 2010, Pieterse et al. 2009). The plant might activate the "JA- ET defensive systems" both quicker and with more strength if it gets stimulated by beneficial microbes (Pieterse et al. 2009).

I would suggest that a recovery process would be added to the simulated DON prediction model which activates when there are dry, cold (below 20 °C) and gently windy conditions. The dry conditions will make the pathogens susceptible towards antagonistic microbes, and it will also, according to a plant hormone signaling review by Pieterse et al. (2009), promote the "JA- ET defensive systems" and simultaneously affect the "SA defensive system" negatively. However it is important that the plant does not sense any shortage of water since that can according to another plant hormone signaling review by Fujita et al. (2006) lead to a shutdown of the "JA- ET defensive system". The cold temperatures below 20 °C will slow down the pathogen´s progress. The gently windy conditions will affect the pathogen negatively by increasing the evaporation.

This suggested recovery process could be tested in a laboratory experiment. The oat plants would be infected with photo-fluorescent *Fusarium* strains, and thereafter exposed to the suggested environmental conditions and monitored for eventual recoveries.

6.1.2.3. Incubation period

When comparing the DON model with all models from the "**2.4.3. Previous model approaches**" section, the ENV v.1 infection model by Del Ponte et al. (2005), the model by Detrixhe et al. (2003) the infection model by Rossi et al. (2003) and the model by Moschini and Fortugo (1996), there is one thing differing; That is the necessary time for an infection to be registered.

The DON model only needs about 21 hours at optimal conditions for a potential infection to be registered, whereas the other models require 48-50 hours. The models by Detrixhe et al. (2003) and Moshini & Fortugo (1996) base the necessary time for infection on statistic knowledge, which means that the models are based on the knowledge that after 48 hours there is often an infection. The models by Del Ponte et al. (2005) and Rossi et al. (2003) are based on an experiment by Rossi et al. (2001). In that experiment seeds are exposed to different moisture and temperature regimes and later screened for infections. However, the screening only detects clinical symptoms, which are visible after a short incubation period. Based on the presence of infections alone, even subclinical infections the DON model on the other hand does not taken any incubation period in account.

When a potential infection can occur there is actually an infection risk and it should be accounted for. However, the incubation period should be considered since an infection is rather weak in early stages. To improve the DON model the event of a potential infection followed by an incubation period with favourable environmental conditions would register a more elevated risk of infection than the current version.

6.1.2.4. Mycelia growth

The DON model does not explicitly simulate the effects of mycelia growth and presence of mycelia inside the kernels as seen in **figure 16**. It is tempting to think that if the fungus becomes larger by producing more mycelia, perhaps it would be capable to produce more DON. In laboratory experiment Lori et al. (1990) found that infected kernels with no visible mycelia growth had higher DON concentrations than infected kernels with a lot of mycelium growth. This suggests that mycelium growth has no or a negative linkage to DON production. However, this is a bit contradicted by a laboratory experiment by Wang et al. (2012) who found that parts of the amt gene group are linked to both mycelia growth and DON production. Some connection between mycelia growth and DON concentrations might therefore exist, especially as, according to Hope et al. (2005), mycelia growth and DON production occur during the same temperature and humidity regimes. More research in this field would be necessary to understand its importance in a DON prediction model.

6.1.2.5. Pathogenic response

Many of the data for the DON model are based on wheat and barley data. In principle all studies in the two sections "**2.3.3.3. Infection requirements**" and "**2.3.4. DON production**" are done on wheat, and these two sections define the infection- and DON production- processes in the model. Therefore it might be a risk that the model is not compatible with oat since wheat and barley have different pathogenic response towards *Fusarium*, than oat.

Walter et al. (2010) mention in a review that wheat plants sometimes detect the *Fusarium* pathogen and primes their defenses inducing a so called type 1 resistance, meaning that initial infection is hindered. Certain cultivars of barley also have a natural type 1 resistance against *Fusarium* infection, due to physiological barriers to infection because they flower with a closed

flower (Yoshida 2007). Oat has no known type 1 resistance against *Fusarium* infection, and chemical analyses show that it does not have any passive effective antibiotic or quorum quenching effect against *F.graminearum* (Bahriminejad et al. 2008). These differences between oat, wheat and barley might mean that the infection and DON production processes for oat are slightly different but enough for making the model unsuitable for oat.

6.1.2.6. Type two resistance

Although evidence are lacking it is speculated that oat has a strong Type II resistance towards infections, meaning that infections cannot spread systemically within the panicle. Systemic spread in oat only occurs when the wind speed is high, since the infected kernels can spread the disease by direct contact between mycelia in infected kernels with non-infected kernels (Bjørnstad & Skinnes 2008, Yan et al. 2010. Teckle et al. 2012). In wheat systemic spread occurs after 5 days of infection when the fungal growth reaches the vascular system in the rachis nodes (Kang & Buchenauer 2000). Barley has a prolonged systemic spread at early dough stages (Skadsen & Hohn 2004).

The systemic spread of Fusarium in oats can be tested by a laboratory experiments. By dividing plants into different groups that are exposed to different wind speeds. The plants are supposed to be planted in normal field density. A couple of plants in each group are by purpose infected with photo-fluorescent *Fusarium* strains in some of their spikelets. Then it is possible to observe how and if the infection spreads within the infected plants and towards adjacent not primary infected plants. If it is true that oat has a Type II resistance, the current model might overestimate the infection, giving indications that the entire plant is infected when only one kernel is infected. With the enhancement of a systemic spread function more correct indices might be produced.

6.1.3. Phenology model uncertainties

The output in form of time periods with available anthers and susceptible tissue from the phenology model is a key component for the DON prediction to be correct. However, the phenology model has not yet been fully validated and calibrated towards field data. There might therefore be some inaccuracy in its outputs regarding amount and start and stop period of present anthers that are potentially susceptible to infections.

The model only recognizes the extruded anthers as susceptible tissue, there might be other tissue parts that can be susceptible. Skadsen and Hohn (2004) found in a laboratory experiment with gfp *F. graminearum* strains in barley that the infection process is enhanced due to the hair on the top of the seeds. Wheat and oat also have hair on the top of their seeds, but it is unknown what role it has in the infection process. This might explain occasions of late infections. A similar experiment similar to what Skadsen and Hohn (2004) performed in wheat is also suggested in oats .

6.1.4. Leaf wetness model uncertainties

Both methods for the leaf wetness model provided good results when validated towards the leaf wetness sensors. The two methods “RA1 and RA2”, described in the section “**4.5.2.1. Validation of leaf wetness model**”, did not show any significant difference between their results. Although

the way of calculating the aerodynamic is the same, still there are many differences between the leaf wetness sensor and oat crops. For example the leaf wetness sensor does neither have any stomata nor any real canopy rain interception capacity. Therefore it is suggested the model validation is redone towards leaf wetness sensors that are located in a field of oat crops at the highest canopy layer. The type of sensors should be changed for a type that is placed on a Gore-Tex film with the other side sticky. The sensor could then be placed on a leaf where Gore-Tex which enables the leaf´s stomata to operate. The sensor itself is suggested to be an open circuit made by thin gold wires with a small current which registers leaf wetness when the circuit closes.

It was not expected that the matching frequency would be so high in the model´s validation towards the leaf wetness sensor since the model is quite simple. The sensors were according to Campell scientific. Inc (2010) not suitable for plant pathology purposes, since they are not efficient enough to register dew. This might explain some of the models overestimations regarding leaf wetness due to dew and fog, still it seemed like the sensors were capable to detect some of the model´s estimated dew and fog. However, the model would need to be enhanced in order to be fully suitable for use in oat crops.

6.1.4.1. Soil surface evaporation

The leaf wetness sensors were placed on a two meters´ height meanwhile the oat crops are up to one meter high. At the height of the oat cultivars there is another factor that contributes a great deal to leaf wetness "Soil surface evaporation" Monteith (1957). Soil surface evaporation is when water fumes from the soil. There is a formula by Monteith (1957) confirmed by Burrage (1971) seen in **equation 40**, that describes the soil surface evaporation process. One problem with including this component in the model is that soil surface evaporation is strongly depending on the temperature 1cm above the soil, which is difficult to measure.

$$D=K_v(T_c-T_{soil})(\Delta\chi/\Delta Tc) \qquad 40$$

Where K_v is a diffusion coefficient ($cm^2\ s^{-1}$), Tc is canopy temperature (°C), T_{soil} is temperature 1cm above soil surface (°C), $(\Delta\chi/\Delta T)$ is the change of absolute humidity due to canopy temperature($g\ m^{-3}\ °C^{-1}\ cm^{-1}$).

6.1.4.2. Cloud coverage

The cloud coverage variables in **equation 7** in **Appendix 1** which are important for dew formation and evaporation are in the model forced to be a constant value even if they in reality vary a lot. In some cases, this can have influenced the transpiration and dew formation; how much is unknown. There are digital produced cloud charts from radar and satellite observations from the Swedish Meteorological and Hydrological Institute or the Norwegian Meteorological Institute that could be used as input data for clear sky and cloud coverage.

6.1.5. Summarizing main modeling strategy discussion

To summarize the main modeling strategy, one can say that the strategy is not capable to estimate and predict DON contaminations. Neither the individual models nor the entire modeling strategy

has been sensitivity analyzed, meaning that it is unknown how big the consequences are for miscalculations.

The models involved in the strategy might also need to be improved to better estimate and predict DON contaminations.

- The **DON model** might need data regarding spore densities from a spore model. It might also need functions that estimate recovery, incubation, mycelia growth and systemic spread processes.
- The **Phenology model** needs to be fully validated and calibrated to oat plants. The enhancement of available susceptible tissue in the end of the growth season due to seed hair needs to be evaluated.
- The **Leaf wetness model** needs to be validated against observations on actual oat plants. The model also might need to include a component estimating leaf wetness due to soil evaporation. The model would also need to use cloud coverage as an input to better estimate evaporation and dew formation.

6.2 Alternative modeling applications

The alternative modeling applications did not manage to estimate DON contamination either. Many of the reasons are similar to the main modeling strategy. The strategies have not had any sensitivity analysis and none of the models has taken any of the following factors into account:

- Disease recovery
- Spore densities
- Mycelia growth influences on DON production
- Systemic spread of infection

The ENV models are also suspected to be incompatible with oat since they only have been developed and validated towards wheat and not oat.

The problems with the phenology model v.2 are the same as for phenology model v.1. The model has not been fully validated and calibrated towards oat plants. Therefore it is difficult to specify any exact problem with the model.

6.3. Alternative perception of the results for both main and alternative modeling strategies

There is actually a risk that results are perceived negative on a false basis. The DON model and/ or at least one of the alternative modeling applications might actually at current state estimate and predict DON contamination correctly. This is because there are errors in the DON measurement sampling strategy. These errors are environmental variation and measurement errors and are described in the subsections below.

6.3.1. Environmental variation

When examining the figures in the result section for the DON model, it is possible to see that the index value on the y axis for a certain measured DON value differs a lot depending on the measurements´ corresponding location. For example in **figure 11** for the Belinda cultivar in Roverud the indices values vary between 150 to 300 for DON measurements at 75µg/kg meanwhile in **figure 10** and **13** for the Belinda cultivar in Rakkestad and Ås the indices values vary between 0 to 5000 for the same DON measurement value. This indicates that the Belinda cultivar plants for some reason often are more sensitive in Roverud than in Rakkestad and Ås. This might be because the site specific environmental conditions in Rakkestad have a positive impact on FHB infection and DON production and therefore the weather conditions are less important. The DON measurement sites might be on locations with poor drainage and/or a topography where the soil is wet most time of the growth season. In total this suggests that index values produced for the specific DON measuring site only can be compared with locations with similar environments. In this thesis this has not been taken in to account and no correlation might have been found because it has been assumed that all fields have the same risk to get infected as long as only the weather conditions are equal.

Also, some of the DON measurements sites were noted to be far away from the regional weather station. This increases the risk that there actually is different weather at the sampling site than at the weather station.

In future experiments it is recommended that it should be possible to track down the measured DON values to the individual field and make regression and correlation analysis on field level to limit environmental variation. Another way would be to find fields in the region that are close to the weather station and resemble each other.

6.3.2. DON sampling uncertainties

It is often quite difficult to conduct a fair sampling of DON. The harvested oat is often very heterogeneous and it is difficult to obtain a representative sample from the sampling procedure. It is not at all impossible that a relative contaminated harvest might be missed since it is difficult to sample the contaminated parts of the harvest. On the other hand a harvest that is almost clean can be counted as heavy contaminated since by accident, a few but heavy contaminated kernels are sampled. The indices might in some instances actually be correct but have not been proven due to difficulties to sample the harvest. This error source alone is not enough to defend the prediction models and probably only affects a couple of samples though.

6.4. Summarizing discussion

None of the modeling strategies could estimate DON contamination correctly and therefore it is doubted that they can be used for prediction purposes. The reason why they failed to predict DON might be because it is difficult to conduct a correct statistical analysis due to environmental variability. This is because of the DON measurement strategy, where the distance between the fields and the weather stations leads to a risk of deviation between the weather station data and

the actual field conditions. However, this is probably not the only reason why the strategies failed. No sensitivity analysis was done on individual model or on entire strategies making it possible for small miscalculations to impact disproportionately much. In addition, most models need enhancement on several points and a spore model estimating the spore density might be necessary.

7. Final Conclusions

This thesis which goal is to “Try to predict Deoxynivalenol in oat with a modeling approach” can be concluded by the following statement:

- There is lack of evidence that this thesis´ attempts to create Deoxynivalenol predicting model strategies actually can predict DON contaminations in oat.

8. Other fields of usage for the leaf wetness module

The leaf wetness model is mainly constructed for *Fusarium* infections and DON production in oat. However the model can easily be used for other plant pathological reasons in oat or other small grain cereals without any major modifications. If it is used during early growth stages before the canopy is fully developed enhancements adjusting the throughfall and canopy capacity are needed.

For trees, bushes and larger crops like maize other models like the storm models by Gash et al. (1995) and Gash (1979) are better suitable. Those models include everything in the rain component but are more focused on the number of storm events and their effect on partly wetting and drying the canopy. These factors were not included in the leaf wetness model since the canopy structure differs greatly between a forest and a small grain crop, and the model also should be kept as simple as possible.

The dew component in the leaf wetness model is probably not suitable for big organic tissue like big fruits since those can store more heat and a thermal lag calculation needs to be considered. There is a dew model by Monteith and Butler (1979) that compensates for thermal lag and is therefore more suitable for that purpose.

9. Future

If any of these modeling strategies would be enhanced enough to predict DON contamination and FHB infections it could be used in an alarm system. For this future alarm system it is suggested that a couple of fields in the region that are representative for the majority of all fields are chosen. DON measurements and predictions should be conducted each year for calibrating and validating the model. Farmers that wish to subscribe for the service should be able to do it at their regional authorities or extension services. The service evaluates the farmers' field with help of GIS (Geographical Information Systems) tools and decides if they resemble the representative fields enough. If the fields do not reassemble the representative fields enough the differences could be accounted for with the help of GIS tools. When the model predicts risk of FHB infection and or DON contamination a sms, prerecorded telephone call or email is sent to the subscribing farmer. The farmer can then with the help from local extension services or regional authorities develop a countermeasure plan towards the FHB infection or the DON contamination.

Even if this kind of alarm system combined with biological (for example viral active components) or chemical fungicide sprays, plant defense priming agents and preventive fungal infection sprays is a strong weapon towards FHB infections, the alarm system must continually be updated. The FHB pathogens can adapt quickly towards the system. For example the pathogens could gain the ability to infect or produce DON at other environmental conditions and they can also become more aggressive and produce more DON. Therefore it is important to validate and calibrate the model strategy towards the disease to detect if the pathogen starts to alter its behaviour and then quickly compensate for it.

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Appendix 1 Leaf wetness model

Rain component

Leaf wetness (L_w) due to rain is strongly depending on the amount of precipitation (P) and evaporation (E). Basically, the model registers leaf wetness (L_w) when the precipitation (P) intercepts (I_{acc}) the canopy, illustrated in **figure 1** as boxed part 2. Not all precipitation (P) is registered as intercepted rainfall (I_{Acc}) due to the following two reasons:

- **Breached canopy capacity**. During heavy rainfalls, the amount of intercepted water (I_{Acc}) is more than the canopy can carry and the so-called canopy capacity (S) is breached. If that happens, the excess water will be drained from the leaves, illustrated in boxed part 4 in **figure 1**. In this model the canopy capacity (S) is set to 4 mm according to observations by Butler and Huband (1985).

- **Throughfall**. Some of the precipitated water simply misses the canopy and hits the soil surface directly or hits the stems and is pulled down by gravity (steam flow). This is called throughfall (F) and is illustrated in **figure 1** as boxed part 3. The throughfall factor (F) in wheat is around 40% according to a field experiment by Butler and Huband (1985) and is in this case considered to be the same for oats.

When the canopy gets wet the model will dry up the wetness by evaporation (E) which is illustrated in **figure 1** as boxed part 5. This evaporation process (E) for drying up the canopy is controlled by Penman's equation under the section for Dew formation. When all intercepted water (I_{Acc}) has been evaporated from the canopy the registration of leaf wetness (L_w) stops. The model can be summarized with **equation 1** and **2**.

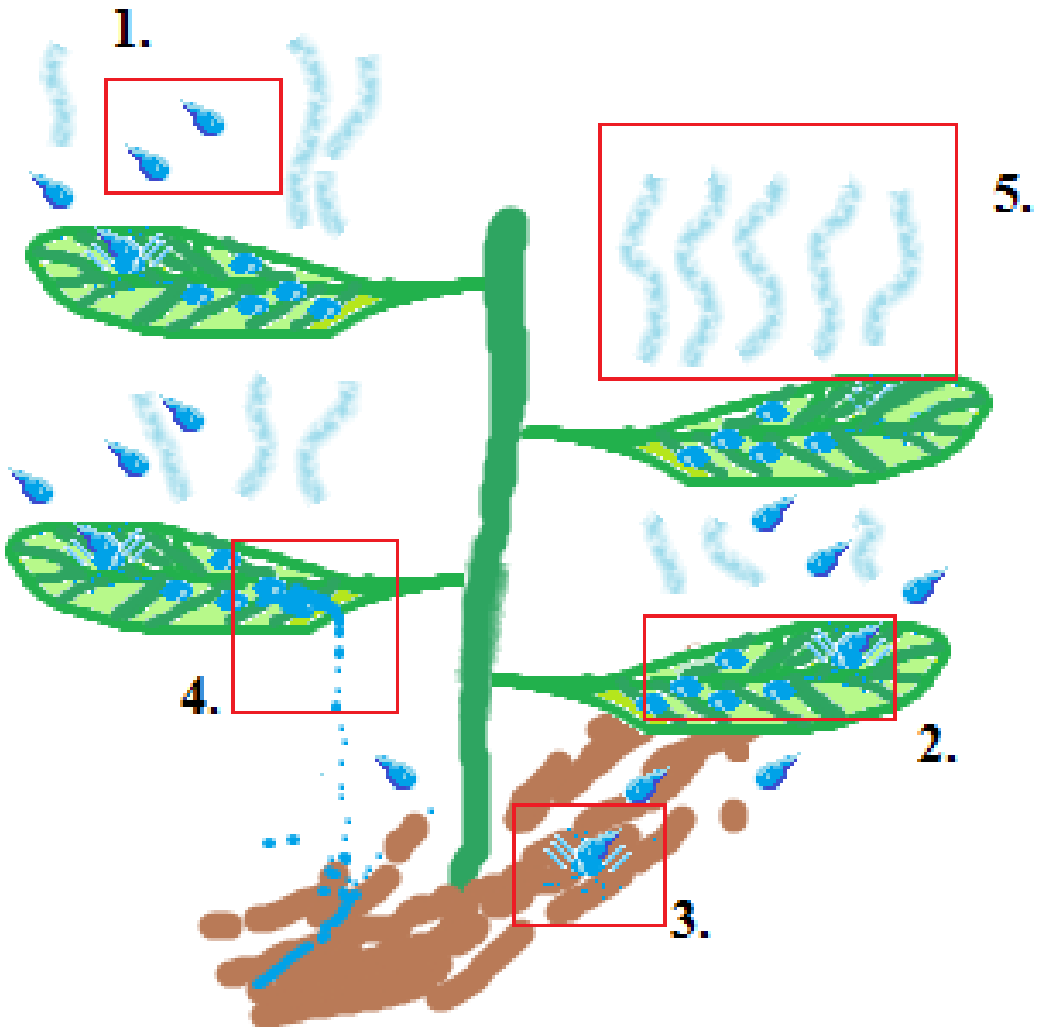

Figure 1. Basic overview of the rain component in the Leaf wetness v.1 model. The first boxed part illustrates precipitation that has not yet intercepted the plant or the soil surface (P). The second boxed part illustrates precipitation that is intercepting and have intercepted a plant leaf (I_{Acc}).The third boxed part illustrates precipitation that misses the plant and becomes throughfall (F).The forth boxed part illustrates a leaf where the canopy capacity has been breached and the excess water is being drained ($I_{Acc} \leq S$). The fifth box illustrates the evaporation of intercepted water.

$$I_{Acc} = \sum_{t \rightarrow t'} (P - F*P - E) \qquad I_{Acc} \leq S \qquad 1$$

where t = start of rainfall, t' = when canopy is dried after rainfall (h), F = the throughfall coefficient (%) and S = the canopy capacity (mm).

To avoid the risk of the module not showing any indications of leaf wetness (L_w) due to higher evaporation (E) than precipitation (P) during a rainfall, it classifies the precipitation (P) event alone as leaf wetness (L_w). Although, since the wetness in reality is drought up rapidly, a moment of leaf wetness has occurred (L_w), otherwise the weather station would not have registered any precipitation (P). How the model indicates leaf wetness (L_w) during periods of precipitation (P) is formulated in **equation 2**.

$L_w = 1$ IF $I_{Acc} > 0$ OR $P > 0$ ELSE $= 0$ 2

Fog component

When relative humidity (RH) is high, spontaneous leaf wetness (L_w) formation is possible. When relative humidity (RH) reaches above 90%, fog is formed due to the high water saturation leading to water droplet formations on particles in the air. The model gives an indication of leaf wetness (L_w) due to fog when relative humidity (RH) is 91% or higher, which is formulated in **equation 3**, since Monteith (1957) observed that leaf wetness (L_w) occurred when relative humidity (RH) spanned between 91-99%. The results from Monteith (1957) are confirmed by Sentelhas et al. (2008) who states that relative humidity (RH) is a valuable leaf wetness indicator according to regression analyses in USA, Canada, Italy and Brazil.

$L_w = 1$ IF $RH \geq 91$ ELSE $= 0$ 3

Dew formation- and evaporation- component

Dew formation works in the same way as when a glass of cold beverage is brought out in a warm summer day and small water droplets are condensed on the glass surface. This is a reversed evaporation process and happens when the surface temperature is cooler then the surrounding. This is rather tricky to predict since it is necessary to know how much cooler the surface temperature must be and how to predict this surface temperature.

Energy balance

Evaporation is an endothermic process meaning that heat which is a kind of energy is taken from the surroundings 2.4518×10^6 Joule is needed to evaporate one kg of water (L). This energy in turns comes from the sun. The model is therefore using the energy balance, formulated in **equation 4**. This equation describes how much of the energy from the sun is distributed to heat the canopy surface directly so called sensible heat (H) and to evaporate the transpiring water from the plants so called latent (LE). It is called latent heat (LE) because the energy needed for evaporation (E) is stored in the water until it condensates and the heat is transformed to sensible heat (H), because condensation is an exothermic process meaning that heat is spread to the surrounding.

The following equations (4 and 5) are taken from Karlsson & Karlberg (2004).

$0 \approx R_n - (H + LE)$ 4

where R_n = net radiation (W m^{-2}), H = sensible heat (W m^{-2}) and LE = latent heat (W m^{-2})

Radiation balance

The net radiation (R_n) in equation 16 is the sum of long way net radiation (L_n), incoming global short way radiation (R_s) mostly from the sun and how much of it is reflected by the crop ($R_s*\alpha$), this is described by the so called radiation balance in **equation 5**.

$$R_n=L_n+R_s-(R_s*\alpha) \quad (5)$$

where L_n = net long way radiation (W m^{-2}), R_s = global radiation (W m^{-2}) and α = the albedo.

The values for Global radiation (R_s) are in the model obtained from the weather files previously described. The amount of reflected global radiation (R_s) is regulated by the crop´s albedo (α). The albedo is a reflection coefficient and is in the model set to 0.25 according to a field measurement with solarimeters in oats by Impens & Lemeur (1969) and double checked by values from Sellers (1965 cited in Karlsson & Karlberg 2004).

The net long way radiation (L_n) term from equation 17 consists of incoming ($L_\downarrow$) and outgoing long way radiation ($L_\uparrow$), shown in **equation 6**. This describes how the earth emits long way radiation and how this radiation is absorbed by clouds and partly reemitted back to earth. The incoming long way radiation ($L_\downarrow$) is calculated according to a dew model by Madeira (2002), where the incoming radiation ($L_\downarrow$) varies due to cloudy and clear sky (cloudy sky, clear sky) and their respective temperature ($T_{cloud,}$ T_{clear}), formulated in **equation 7, 8** and **9**. Since there is no data about cloudiness from the weather stations when running the simulations, the amount of cloudiness and clear sky is constantly set to 50% in the model. The emission of long way radiation ($L_\uparrow$) is calculated according to Stefan Boltzmann´s blackbody law, described in **equation 10**. Since the oat fields are not perfect blackbodies the emissitivity (ε) of them are set to 0.98 according to a field experiment by Humes et al. (1994) and also double checked by values from Sellers (1965 cited in Karlsson & Karlberg 2004).

The following equations (18-21) are taken from Madeira (2002).

$$L_n = L_\uparrow+L_\downarrow \quad (6)$$

$$L_\downarrow=(\sigma*\text{clear sky}*T_{clear}^4)+(\sigma*\text{cloudy sky}*T_{cloud}^4) \quad (7)$$

$$T_{cloud}=T_a-15 \quad (8)$$

$$T_{clear}=T_a-20 \quad (9)$$

$$L_\uparrow= \varepsilon \sigma T_a^4 \quad (10)$$

where $L_\uparrow$ = outgoing long way radiation (W m^{-2}), $L_\downarrow$ = incoming long way radiation (W m^{-2}), σ = the constant of Stephan Boltzman (J m^{-2} s^{-1} K^{-4}), ε = emissivity, clear sky = percentage of clear sky (%), cloudy sky = percentage of cloudy sky (%), T_{cloud} = cloud temperature (C°), T_{clear} = the sky temperature (c).

Latent Heat

The latent heat term alone describes the energy necessary to evaporate water. Apart from the resistance variables the latent heat term is controlled by the difference between saturated canopy vapor pressure and ambient vapour pressure seen in **equation 11**. This difference between vapour pressure gives an indication if there is room for water to evaporate at the canopy surface. If the difference turns out to be positive it indicates that there is room and perspired water is evaporated. If the difference turns out to be negative, there is no room for evaporation on the surface and water is condensed from the ambient air. The greater difference the more water is evaporated or condensed.

The ambient vapour pressure in **equation 11** is the product of the relative humidity and the ambient saturated vapour pressure, formulated in **equation 13**. The relative humidity is obtained from the weather files. The saturated vapour pressure for both the canopy and the ambient air is calculated by the empirical Tetens´formula formulated in **equation 12**, where the canopy temperature for respective canopy surface and ambient air is used. The Tetens´formula was supposed to be most suitable for calculating saturated vapour pressure according to Anyadike (1984) who tested seven different empirical formulas for that purpose.

The following equations (23-25) are taken from Eckersten et al. (2004) and Monteith (1981).

$$LE=((Cp*\rho)(e_{cs}-e_a))/(\gamma*(r_a+r_c)) \quad 11$$

$$e_s = 0.611*10^{(7.5T/(T+237.3))} \quad 12$$

$$e_a=(RH \times e_{sa}) / 100 \quad 13$$

where Cp = the specific heat capacity for air (1004 J kg^{-1} K^{-1}), ρ = the density of moist air (1.2047 kg m^{-3}), e_{cs} = saturated canopy vapour pressure (hPa), e_a = ambient vapour pressure (hPa), γ = the psykrometer constant (0.67 hPa K^{-1}), r_a = the aerodynamic resistance (s m^{-1}) and r_c = the canopy resistance (s m^{-1}).

Stomata resistance

The stomata resistance (r_c) in **equation 11** describes the leaf transpiration. When the stomata are open they evaporate water which increases the latent heat (LE). When they close, they hinder evaporation and the latent heat (LE) becomes more dependent on the aerodynamic resistance (r_a). The stomata resistance (r_c) depends on gathered stomata conductance (g) in the canopy, which is described in **equation 14**. The gathered stomata conductance is calculated only for each layer in the canopy and therefore the leaf area index (LAI) value is necessary in **equation 14**. In the model the leaf area index (LAI) value is set to 4 according to Best & Harlan (1985). The stomata conductance itself is according to Lindroth (1985) correlating to the increasing conductance due to increasing global radiation and decreasing conductance due to decreasing vapour pressure deficit. The relation can be formulated according to the Lohammar equation in **equation 15**.

Equation nr 29-30 are taken from Lindroth (1985) who bases the equations on the work of Lohammar et al. (1980).

$r_c = 1 / (g*LAI)$ $r_c = 0$ IF $R_s>0$ OR $L_w=0$ ELSE $r_c = 1 / (g*LAI)$ 14

$g = R_s*g_{max} / ((R_s + a_{ris})(1+(e_{cs}+e_a) / a_{vpd}))$ 15

where g = stomata conductance in (m s^{-1}), LAI = leaf area index, g_{max} = the highest possible stomata conductance, a_{ris} = stomata response coefficient towards radiation and a_{vpd} = response coefficient towards vapour pressure deficit.

The highest conductance value (g_{max}) in **equation 15** describes the highest conductance possible and is 0.016 m/s according to field observations in oats by Müller et al. (1986). The a_{ris} coefficient describes the affect increasing levels of radiation have on stomata opening for photosynthesis. a_{ris} is set to 460 according to stomata conductance measurements in oat due to changes of photosynthetic active radiation flux by Fay & Knapp (1993). The coefficient a_{vpd} describes when the stomata conductance (g) is halved due to vapour pressure deficit. a_{vpd} is set to 7 according to measurements on stomata conductances due to vapour pressure deficit in oat in a work by Polley et al. (1992) and calculated stomata changes by vapour pressure deficit in wheat by Yuan & Luo (2012).

In theory, the stomata on the leaves are supposed to be fully opened during leaf wetness. However **equation 15** is not always capable to detect leaf wetness on its own. Therefore there is a rule in **equation 29** that sets stomata resistance to 0 during leaf wetness. There is also a rule in **equation 29** which sets stomata resistance to 0 if global radiation is zero or below, this rule is to avoid negative stomata values.

Sensible Heat

The sensible heat term (H) in **equation 4**, describes the amount of energy from the net radiation which heats the canopy surface. The term is then regulated by the difference between the canopy (T_c) and ambient temperature (T_a), as seen in **equation 16**. The term also depends like the latent heat term (LE) on the aerodynamic resistance (r_a). The formulation of the sensible heat term (LE) is found and described in both Eckersten et al. (2004) and Monteith (1981).

$H = (Cp*\rho(T_c-T_a))/r_a$ 16

Aerodynamic resistance

How well the sun manages to warm the plant surface and the plants´ perspired water is depends a lot on the current wind speed. If the wind speed is low, the plant surface temperature tends to rise since it is not mixed with the surrounding air. This is like when dropping pieces of ice in a glass of water, when spinning them around in the water make them melt faster and all the water gets cooler, when not spinning them around they take longer time to melt and only the top layer of the water gets cooler. This phenomenon is called the aerodynamic resistance and depends on the logarithmic wind law, which is formulated in **equation 17**. The law describes wind speed (u) differentiations depending on the height above the soil. The differentiations are due to turbulence

in the air formed by friction from the soil and canopy surface, which is illustrated in **figure 2** where the speed of a wind front is differentiating due to turbulence over an oat field. The formula therefore uses information of zero plane displacement (z_d) which is used to describe the height of the crop, and the roughness factor (z_o) which describes the roughness of the crop, the formulas are described in **equations 18** and **19**. The crop height (z_h) for oats is estimated to be 1 m.

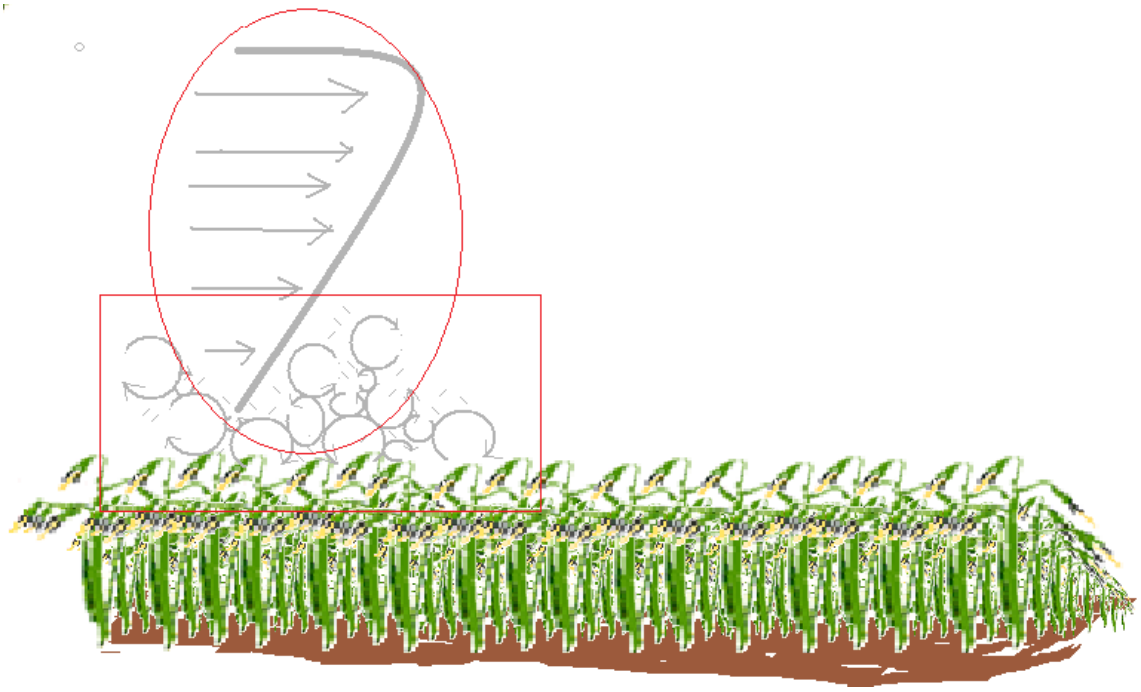

Figure 2, Illustration of the logarithmic wind law, where the wind front in the red circle gets weaker the closer to the oat crop, due to turbulence in the red squared area.

The following equations (23-25) are taken and reviewed by Eckersten et al. (2004), Lindroth et al. (2004) and Oke (1978).

$$r_a = (\ln((z - z_d) / z_o))^2 / (k^2 u) \quad \text{Where MIN } u=0.3 \qquad 17$$

$$z_d = 0.67 z_h \qquad 18$$

$$z_o = 0.1 z_h \qquad 19$$

where z = the height above ground where wind measurements take place (m), z_d = the zero-plane displacement (m), z_o = the roughness factor (m), k = von Karmans´ constant (0.41), u = measured wind speed (m s^{-1}) and z_h = the height (m).

If there is almost no wind, the aerodynamic resistance will increase towards infinity. If that would actually be the case the plant would be incinerated because the canopy temperature would also increase towards infinity. This does not happens since the warm leafs will form air turbulence by heating air at the leaf surface that will go upward, so called buoyancy forces (Monteith 1965). Therefore there is a rule that makes the wind speed to never undergo 3 m s^{-1}.

Canopy temperature

The canopy temperature (T_c) which is a necessary component for the sensible and latent heat terms (H and LE) is rarely measured at meteorology stations. Therefore the canopy temperature (T_c) is calculated by the energy balance in **equation 4**. In this thesis a combination between a graphical and a numerical solution has been used to obtain the canopy temperature (T_c).

According to a leaf-air temperature difference study by Eckersten & Kowalik (1986), the canopy temperature (T_c) seldom differs largely from the ambient air temperature (T_a). This knowledge is used when calculating the canopy temperature, which is done in the following way:

- Fictional canopy temperatures (T_c) are created, differing with 0.1°C from each other and differing 0°C to 10.5°C from the ambient air temperature (T_a), formulated in **equation 20**.
- The energy balance formula is formulated as a function depending on the fictional canopy temperatures as seen in **equation 21**. The fictional canopy function is equal to 0 as much as possible seen in **equation 21** and **figure 3**.
- The achieved canopy temperature is adjusted by halving excess or deficit from the energy balance and adding it to the sensible heat term.
- The final canopy temperature is calculated breaking it out from the sensible heat term seen in **equation 22**.

$T_{c1}= T_a-(10.6-(0.1*1))$, $T_{c2} = T_a-(10.6-(0.1*2))$, $T_{c3}=T_a-(10.6-(0.1*3)\ldots T_{cn} =T_a-(10.6-(0.1*n))$	Where MAX n=210	20
$f(T_{cn})=R_n-(((Cp*\rho(T_{cn}-T_a))/r_a)+(((Cp*\rho)(e_{cns}-e_a))/(\gamma*(r_a+r_c))))$, $e_{cns} = 0.611*10^{(7.5T_{cn}/(T_{cn}+237.3))}$	$f(T_{cn})\approx 0$	21
$(H/((Cp*p)/r_a))-T_a=T_c$		22

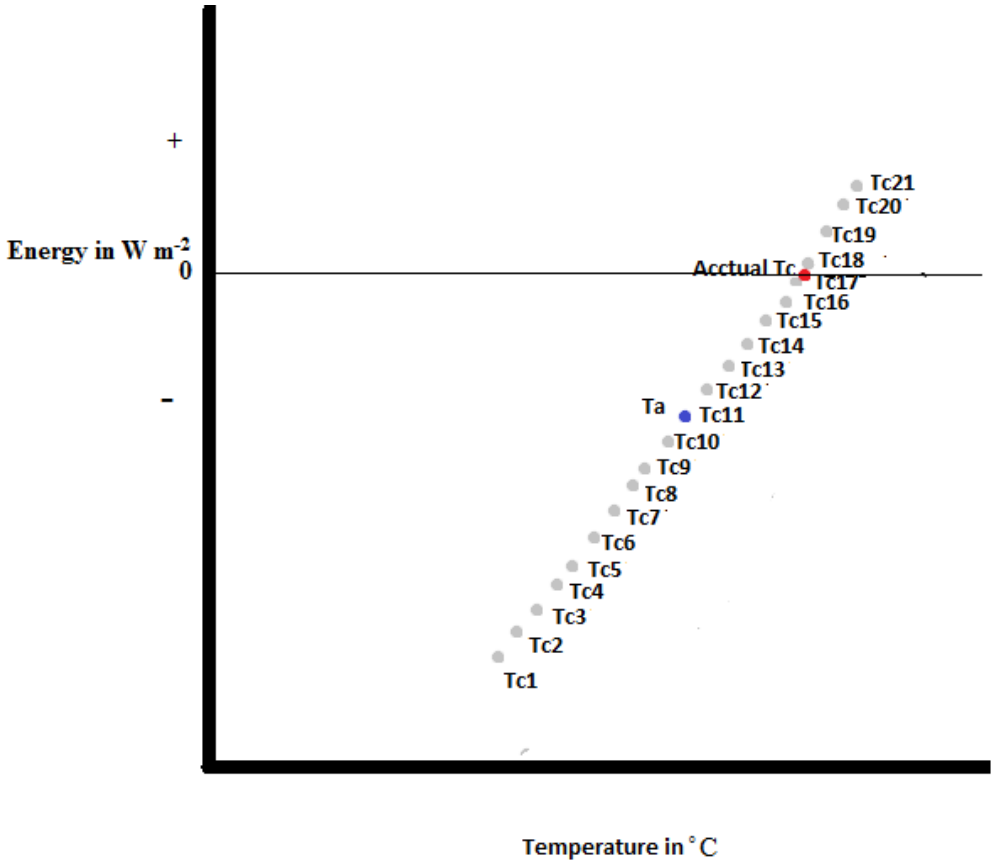

Figure 3. illustrating the graphical calculation of the canopy temperature. The canopy temperature is detected by screening after which fictional canopy temperature that can make the energy balance become equallent to 0 W m^{-2}.

Evaporation and Dew

The evaporation (E) and dew formation is calculated by Penman's equation, seen in **equation 23**. The equation combines all the terms of the energy balance and is therefore depending strongly on the radiation balance (**equation 5**), aerodynamic resistance (r_a), vapour pressure deficit (e_{cs}-e_a) and the slope of the saturated vapour pressure at the mean of both the canopy and ambient temperature (s) formulated in **equation 24**. When Penman's equation shows positive results, it is used as evaporation data for drying up the canopy in the rain model. When the equation shows negative results it means that dew is present, since that is the opposite from evaporation, seen in **equation 25**.

$$E=((R_n*s+((\rho*Cp*(e_{cs}-e_a))/r_a))*1/(s+\gamma))/L \quad (23)$$

$$s=(e_{cs}-e_s)/(T_c-T_a) \quad (24)$$

$$L_w=1 \text{ IF } E<0 \text{ ELSE}=0 \quad (25)$$

where s = the slope of the saturated vapour pressure (hPa $°C^{-1}$), L = the energy necessary for transformation of water from liquid to gas (2.4518 x 10^6 J Kg^{-1})

Appendix 2 Tooken list

α	albedo	
β_0	primary expected value	
$\beta 1$	slope of function	
$\Delta\chi/\Delta T_c$	change of absolute humidity due to canopy temperature	
ε	emissivity	
ρ	density of moist air	1.2047 kg m^{-3}
σ	the constant of Stephan Boltzman	J m^{-2} s^{-1} K^{-4}
γ	psykrometer constant	0.67 hPa K^{-1}
μ	mean leaf wetness matching ratio for respective method	
a	scaling parameter	
a	probability	
ANT	daily proportion of present anthers	Plants $Plants^{-1}$ (Total amount of $plants^{-1}$)$^{-1}$
ANText	proportion of extruding anthers	Plants $Plants^{-1}$ (Total amount of $plants^{-1}$)$^{-1}$
ANTdrop	propotion of dropping anthers	Plants $Plants^{-1}$ (Total amount of $plants^{-1}$)$^{-1}$
a_{ris}	stomata response coefficient towards radiation	
a_{vpd}	response coefficient towards vapour pressure deficit	
b	shape parameter	
clear sky	percentage of clear sky	%
cloudy sky	percentage of cloudy sky	%

Cp	specific heat capacity for air	1004 J kg^{-1} K^{1}
d	active leaf length	cm
d	difference for leaf wetness matching ratio for each simulation between the methods	
$\bar{\mathbf{d}}$	the median difference for leaf wetness matching ratio between the two methods	
E	evaporation	mm h^{-1}
$\mathbf{e_a}$	ambient vapour pressure	hPa
$\mathbf{e_{cs}}$	saturated canopy vapour pressure	hPa
$\mathbf{e_s}$	saturated vapour pressure	hPa
F	throughfall coefficient	%
g	stomata conductance	m s^{-1}
GIB	FHB risk index	
$\mathbf{g_{max}}$	highest possible stomata conductance	m s^{-1}
H	hypothesis	
H	sensible heat	W m^{-2}
$\mathbf{I_{Acc}}$	accumulated interception	mm
INF	proportion of plants that risk being infected under an infection event	Plants Total amount of plants-1
$\mathbf{Inf_c}$	infection score *F.culmorum*	
$\mathbf{Inf_g}$	infection score *F.graminearum*	

Inm_c	indication of *F.culmorum* infectious matter presence	0 OR 1
Inm_g	indication of *F.graminearum* infectious matter presence	0 OR 1
k	von Karmans´ constant	0.41
k_v	diffusion coefficient	$cm^2\ s^{-1}$
L	energy necessary for transformation of water from liquid to gas	2.4518 x 106 J Kg^{-1}
LAI	leaf area index	
LE	latent heat	W m^{-2}
Ln	net long way radiation	W m^{-2}
L_w	leaf wetness	0 OR 1
$L_\uparrow$	outgoing long way radiation	W m^{-2}
$L_\downarrow$	incoming long way radiation	W m^{-2}
n	number of simulations	
R	ccorrelation coefficient	
r_a	aerodynamic resistance	s m^{-1}
RH	relative humidity	%
Risk DON_c	DON risk index for *F.culmorum*	
Risk DON_g	DON risk index for *F.graminearum*	
Risk DON_{tot}	summarized DON risk index	
Risk inf_c	disease score for *F.culmorum*	
Risk inf_g	disease score for *F.graminearum*	

r_c	canopy resistance	s m^{-1}
R_n	net radiation	W m^{-2}
R_s	global radiation	W m^{-2}
P	precipitation	mm
PNG	proportion plants which panicles are emerging under a day	Plants Total amount of $plants^{-1}$
S	canopy capacity	mm
s	slope of saturated vapour pressure	hPa $°C^{-1}$
S_d^2	variation between the two methods leaf wetness matching ratio	
Spore $germ_c$	indication of conidospore germination	0 OR 1
Spore $germ_g$	indication of ascospore germination	0 OR 1
SP_{xy}	sum related to the xi values	
ST	susceptible tissue	Plants $Plants^{-1}$ (Total amount of $plants^{-1})^{-1}$
SS_x	sum related to the yi values	
SS_y	sum related to the yi value	
T_a	ambient air temperature	°C
T_c	canopy surface temperature	°C
T_{cloud}	cloud temperature	°C
T_{soil}	temperature 1 cm above soil surface	°C
u	measured wind speed	m s^{-1}

$\mathbf{z}$	amount of t-test scores	
$\mathbf{x}$	leaf wetness matching ratio for respective method and simulation	
$\mathbf{x_i}$	indices values	
$\overline{\mathbf{x}}$	mean index value	
$\mathbf{y_i}$	measured DON values	
$\overline{\mathbf{y}}$	mean measured value	
$\mathbf{z}$	height above ground where wind measurments take place	m
$\mathbf{z_d}$	zero-plane displacement	m
$\mathbf{z_h}$	height	m
$\mathbf{z_o}$	roughness factor	m