

Yield reduction of spring barley in relation to disease development caused by *Rhynchosporium secalis*

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Abstract. Effects of barley scald caused by *Rhynchosporium secalis* on grain yield were studied in three spring barley cultivars under field conditions using artificial inoculation over three years. The disease strongly reduced the green-leaf area duration compared with fungicide-treated leaves. At low infection level, *R. secalis* reduced the grain yield of barley by 3—5 %. Moderate and severe infection reduced the grain yields of susceptible cultivars by 10—12 %. Single-tiller analysis of yield components indicated that grain weight and ear weight were most affected, but the number of grains was only insignificantly reduced by the disease. Implications of these results for controlling scald disease in Finland are discussed.

Index words: *Rhynchosporium secalis*, spring barley, yield loss

Introduction

Rhynchosporium secalis (Oud.) Davis, the causal agent of leaf scald, is a serious pathogen of barley in many parts of the world (SHIPTON et al. 1974). When environmental factors favour scald development, yield losses of up to 35—40 % have been reported for spring barley (SCHALLER 1951, JENKINS and JEMMETT 1967, JAMES et al. 1968). *Rhynchosporium* is heavily dependent on rain and high humidity. The spores are dispersed by rain (POLLEY 1971, STEDMAN 1980), and mycelia on dead leaves can produce new spores within 24 h of wetting, but after drying there is no further sporulation until rewetting occurs (SKOROPAD 1962). RYAN and CLARE

(1975) have shown that, depending on the isolate, the optimal temperature for germ tube production and growth as well as for maximal lesion development immediately following inoculation, is 15—25°C. Leaf surface wetness for over 14 hours appears to give optimal conditions for lesion and symptom development (POLLEY 1971, RYAN and CLARE 1975). It is interesting, however, that in many barley growing areas *R. secalis* survives between cropping even when it is very hot and there is little rain (MAYFIELD and CLARE 1985). For example, in Australia (MAYFIELD and CLARE 1984) *R. secalis* in host debris survives summer temperatures of up to 20°C above those

that usually inhibit spore germination (RYAN and CLARE 1975), and the pathogen is subsequently capable of producing inoculum that infects barley. In Finland, I have found that *R. secalis* can easily survive and even grow in barley canopy in rainless and hot summer periods, apparently because the canopy is wet at night, and low light intensity provides a favourable compensation environment for scald development.

In the past twenty years, barley scald disease has become increasingly common in Finland. During that period the barley area in Finland has remarkably increased, and barley cultivation has become more specialized. The increased area and monoculture have at least partly contributed to the recent development of scald disease in Finland. In a disease survey carried out by MÄKELÄ (1974) in 1971—1973 30% of the fields were infected by *R. secalis*. However, even though scald seems to be a very important pathogen of barley in cool and wet years, no information is available of its impact on the yield.

The present study was designed to provide information of how *R. secalis* can influence yield and yield components of some commonly grown barley cultivars in Finland. The significance of these results for disease control is discussed.

Materials and methods

Yield reduction in barley caused by scald disease was studied in three years, 1983, 1985, and 1986. Three cultivars, susceptible Hankkija's Pokko, Ida, and moderately resistant or less susceptible Birger were selected for field tests. Experiments were carried out in a randomized block design with eight replications, half of which were inoculated with *R. secalis*. In 1983, experiment I was designed to study the edge effects of plot on disease induced yield loss. Therefore, 20 m² plots were used, but only the central part of the plot was used for yield harvesting. In the other experiments, plot size was 10 m². Normal fertilization levels and herbicide treatments were used.

All inoculated plots were surrounded by oat guard plots to prevent inoculum from spreading into control plots.

Inoculation was carried out by spraying *R. secalis* spore suspension onto test plots. In most cases, mixtures of two or three isolates of *R. secalis* were used. The isolates were collected from various parts of barley area in Finland and cultured on lima bean agar at 16°C for 10—14 days for spore production. Spore suspension of about 10⁶ spores/ml were used for inoculation.

Inoculations were made late in the evening in order to ensure high humidity for disease development. In 1983, inoculations were started at the beginning of flag leaf emergence, at a late stage of barley development. Occasional rains a few days after inoculations enhanced disease development so that only one inoculation was made. In 1985 and 1986, inoculations were started already at the three leaf stage, and three inoculations were made every fifth day.

Disease development was monitored by counting 60 plants per plot and estimating disease containing leaf area on three upper leaves. At the same time, green-leaf area duration was monitored on labelled tillers by visually estimating non-green leaf area because this trait was more accurate to estimate than green-leaf area.

In some cases, fungicide treatments (Bayleton, Tilt) were included in the trials in order to evaluate the effects of fungicides to control scald disease.

Before harvest, the 60 labelled tillers per plot were collected and subjected to yield component analysis. Grain yields and thousand grain weights were analyzed from whole plot data. Analyses of variance were computed from yield data.

Results

Disease development

Weather conditions during the growing seasons varied greatly (Table 1). In 1983 the

Table 1. Monthly mean temperatures and total rainfall in May–August in the years 1983, 1985 (Helsinki-Malmi airport), and 1986 (Helsinki, Kaisaniemi).

| | | Mean temperature °C | Rainfall mm |
|------|--------|---------------------|-------------|
| 1983 | May | 11.8 | 37.5 |
| | June | 13.9 | 55.3 |
| | July | 18.3 | 23.8 |
| | August | 15.8 | 52.4 |
| 1985 | May | 9.4 | 60.5 |
| | June | 13.7 | 66.3 |
| | July | 16.1 | 77.5 |
| | August | 16.3 | 95.4 |
| 1986 | May | 10.3 | 36.0 |
| | June | 17.0 | 29.0 |
| | July | 17.3 | 82.0 |
| | August | 14.6 | 160.0 |

weather was dry, and particularly in July the rainfall was exceptionally low. However, at the same time the temperature was higher than in most years, and *R. secalis* infection progressed first moderately, but slowed down towards the end of the growing period (Fig. 1).

In 1985 the weather was favourable for scald development. June and July were cool and rainy, and *R. secalis* progressed rapidly from the lower leaves to the upper parts of the canopy (Fig. 2).

Summer 1986 was exceptional in many respects. It rained very little in June, moderately in July, but both June and July temperatures were high compared with previous years. Low rainfall in June and the high temperatures in June and July apparently delayed the epidemic build-up of *R. secalis*, and several inoculations were needed to induce disease outbreak (Fig. 3).

In all experiments cv. Pokko was more severely infected by *R. secalis* than cv. Birger. Fungicide treatments reduced the disease progress into the canopy (Fig. 1).

Disease-induced green-leaf area destruction was monitored simultaneously with disease severity. The results indicate (Fig. 4) that leaf-area duration was inversely associated with disease progress. Birger appeared to prolong leaf area duration under disease pressure

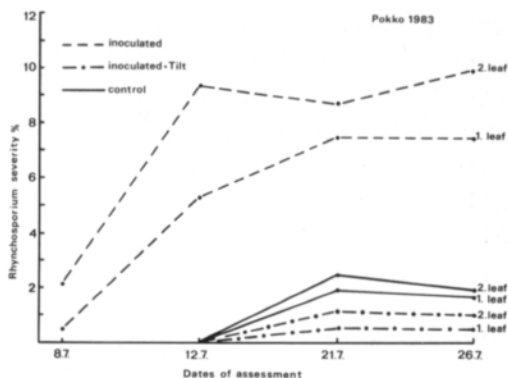


Fig. 1. Development of *Rhynchosporium secalis* on two upper leaves of cv. Pokko in 1983 under dry weather conditions.

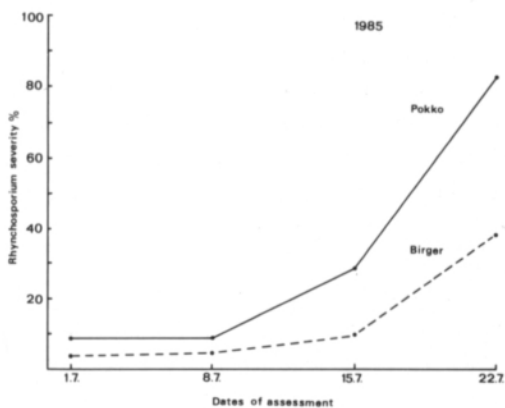


Fig. 2. Development of *R. secalis* on second leaves of cultivars Pokko and Birger in 1985 under favourable weather conditions for disease development.

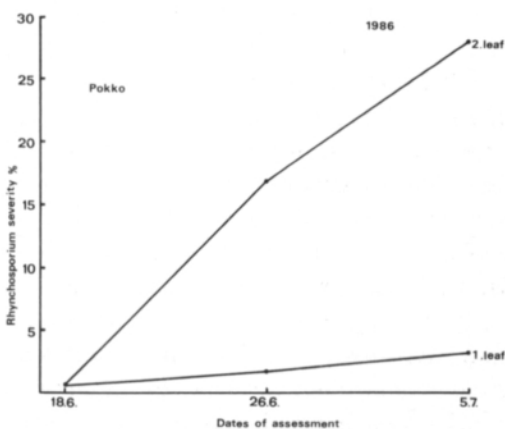


Fig. 3. Development of *R. secalis* on two upper leaves of cv. Pokko in 1986.

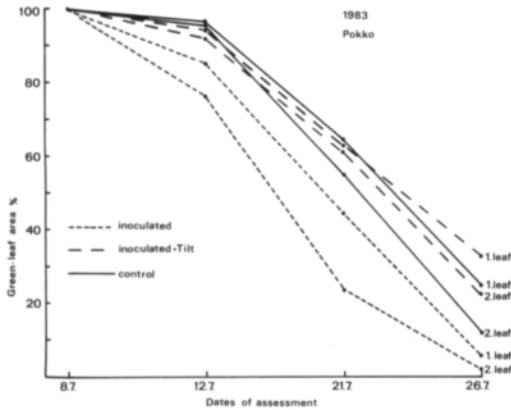


Fig. 4. Green-leaf area duration on two upper leaves of cv. Pokko in 1983 under disease stress caused by *R. secalis*.

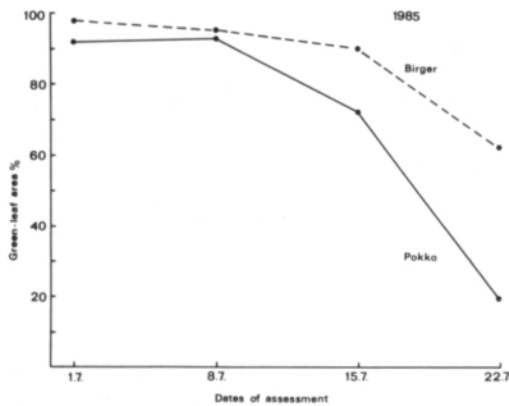


Fig. 5. Green-leaf area duration on second leaves of cultivars Pokko and Birger under disease stress in 1985.

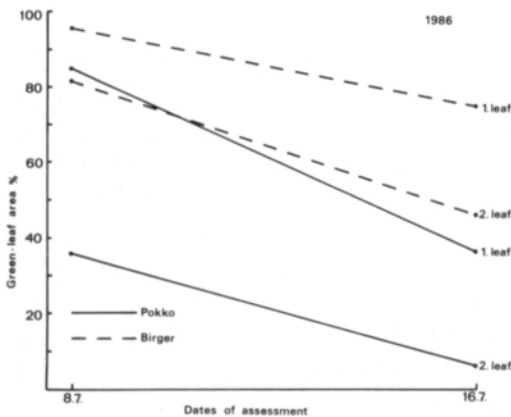


Fig. 6. Green-leaf area duration on two upper leaves of cultivars Pokko and Birger under disease stress in 1986.

better than the more susceptible Pokko (Figs 5, 6). Fungicide treatment delayed the senescence of leaves (Fig. 4). In 1985, the disease reduced green-leaf area more rapidly than in years 1983 and 1986, which was in accordance with the rapid progress of scald under favourable weather (Fig. 2).

Effect of *R. secalis* on grain yield

Despite the unfavourable weather for scald development in 1983, *R. secalis* induced 10–11% ($p < 0.01$ – 0.1) yield losses in Pokko in two experiments compared with the untreated control (Table 2). The plot size of experiment I was twice that of experiment II, and only the central part of plot I was harvested and used for yield measurement. In this way the edge effects were considered to be reduced. However, there was only a small difference in yield loss between these two trials.

In 1985, the weather was favourable for a rapid disease build-up, but *R. secalis* did not reduce the grain yield of Pokko more than 12% (Table 3). In the same trial, *R. secalis* did not cause any reduction in the grain yield of Birger. That was interesting because the disease was able to progress in Birger (Fig. 2). In Table 3, the results of Birger are based on two years' combined data (1985–1986) because the number of replications in both years was small.

In 1986, the weather was not suitable for

Table 2. Effect of *R. secalis* on grain yield of spring barley cultivar Hankkija's Pokko, in comparison with uninoculated controls in the year 1983.

| Treatment | Grain yield | | | |
|------------------------------------|--------------|-------|--------------|-------|
| | Experiment 1 | | Experiment 2 | |
| | kg/ha | ratio | kg/ha | ratio |
| Control | 3753 | 100 | 4856 | 100 |
| <i>Rhynchosporium</i> -inoculation | 3365 | 90 | 4309 | 89 |
| Significance | * | | ** | |
| L.S.D. _{.5%} | 332 | | 329 | |

*, ** significant at $P < 0.05$, $P < 0.01$ levels respectively.

Table 3. Effects of *R. secalis* on grain yield of spring barley cultivars Hankkija's Pokko and Birger, in comparison with uninoculated controls in the year 1985.

| Treatment | Grain yield | |
|------------------------------------|-------------|-------|
| | kg/ha | ratio |
| <i>Pokko</i> | | |
| Control | 4340 | 100 |
| <i>Rhynchosporium</i> -inoculation | 3807 | 88 |
| Significance | | |
| L.S.D. _{.5%} | | |
| <i>Birger</i> | | |
| Control | 4882 | 100 |
| <i>Rhynchosporium</i> -inoculation | 4822 | 99 |
| Significance | | |
| L.S.D. _{.5%} | N.S. | 758 |

N.S. = not significant

Table 4. Effects of *R. secalis* on grain yields of spring barley cultivars Hankkija's Pokko and Ida, in comparison with uninoculated controls in the year 1986.

| Treatment | Grain yield | |
|------------------------------------|-------------|-------|
| | kg/ha | ratio |
| <i>Hankkija's Pokko</i> | | |
| Control | 5156 | 100 |
| <i>Rhynchosporium</i> -inoculation | 4877 | 95 |
| Significance | | |
| L.S.D. _{.5%} | N.S. | 624 |
| <i>Ida</i> | | |
| Control | 4223 | 100 |
| <i>Rhynchosporium</i> -inoculation | 4090 | 97 |
| Significance | | |
| L.S.D. _{.5%} | N.S. | 599 |

N.S. = not significant; * significant at $P < 0.05$ level.

rapid disease spread, and *R. secalis* reduced the grain yields of susceptible varieties Pokko and Ida only 5 and 3 %, respectively (Table 4).

Effect of *R. secalis* on yield components

Infection was severe in experimental fields in 1983 and 1986 only at a late stage of barley

Table 5. Effects of *R. secalis* on 1000-grain weights of spring barley cultivars Hankkija's Pokko, Ida, and Birger, in comparison with uninoculated controls in the year 1986.

| Treatment | 1000-gw | |
|------------------------------------|---------|-------|
| | g | ratio |
| <i>Hankkija's Pokko</i> | | |
| Control | 49.7 | 100 |
| <i>Rhynchosporium</i> -inoculation | 47.3 | 95 |
| Significance | | |
| L.S.D. _{.5%} | * | 2.1 |
| <i>Ida</i> | | |
| Control | 54.1 | 100 |
| <i>Rhynchosporium</i> -inoculation | 53.6 | 99 |
| Significance | | |
| L.S.D. _{.5%} | N.S. | 1.0 |
| <i>Birger</i> | | |
| Control | 59.8 | 100 |
| <i>Rhynchosporium</i> -inoculation | 59.7 | 100 |
| Significance | | |
| L.S.D. _{.5%} | N.S. | 6.8 |

N.S. = not significant; * significant at $p < 0.05$ level.

development, and thus grain weight was the yield component principally affected by the disease. Measurements of thousand grain weights from whole plots in 1986 revealed significant reduction only in the grain weight of Pokko (Table 5), while the susceptible cultivar Ida suffered only slightly. The grain weight of Birger was not affected at all.

Single tillers were taken from the plots for more detailed analyses. The yield component data (Table 6) showed that disease induced significant reductions in the grain weight of Pokko. Grain numbers/ear and ear weights were also reduced but non-significantly. Infection reduced all yield components of Ida, but grain weight and ear weight were significantly reduced (Table 6). The results thus suggest that the grain weight data above, based on samples from whole plots, might be more liable to sample errors than the data based on single tillers.

Table 6. Effect of *R. secalis* on yield components of spring barley cultivars Hankkija's Pokko and Ida in comparison with uninoculated controls in the year 1986.

| Treatment | grains/ ear | ear weight | 1000-gw |
|--|----------------|------------|---------|
| | | g | g ratio |
| <i>Hankkija's Pokko</i> | | | |
| Control | 34.8 | 1.31 | 37.3 |
| <i>Rhynchosporium</i> - inoculation | 32.9 | 1.17 | 35.0 |
| Significance | N.S. | N.S. | ** |
| L.S.D. _{.5%} | 4.4 | 0.19 | 2.7 |
| <i>Ida</i> | | | |
| Control | 16.4 | 0.83 | 50.3 |
| <i>Rhynchosporium</i> - inoculation | 15.8 | 0.75 | 47.1 |
| Significance | N.S. | ** | *** |
| L.S.D. _{.5%} | 0.8 | 0.06 | 1.5 |

N.S. = not significant; **, *** significant at $P < 0.01$, $P < 0.001$ levels respectively.

Discussion

The Finnish climate is favourable for scald disease development. In most years the growing season is cool and wet, and later in summer dew provides humidity inside barley canopy for many hours, which ensures scald survival even in rainless periods. Finnish farming practice also favours the development of scald disease because barley fields are often small and surrounded by forests which keep them humid. Autumns are often wet, which makes effective ploughing difficult with the consequence of abundant debris remaining on soil. *R. secalis* can overwinter in plant debris (POLLEY 1971) and spread into new barley growth in the next season. This and infected seed are apparently the main sources of its inoculum in Finland. There are some clear indications that the increasing use of CCC to prevent lodging promotes the incidence of *R. secalis* (SHIPTON et al. 1974), and in southern Finland farmers have increasingly used CCC in recent years. All these factors and the fact that most currently grown barley cultivars

are susceptible to *R. secalis* (KARJALAINEN, unpublished) have contributed to the wide-scale incidence of scald disease in Finland in the past years.

The results reported here show a yield reduction of 5–12 % in the susceptible cultivar Pokko in relation to untreated control plots. A similar type of experiment was carried out by SKOROPAD (1960), who compared inoculated and fungicide treated plots. He found 10–12 % yield losses due to induced field epidemics caused by *R. secalis*. In California, SCHALLER (1963) reported crop losses of up to 35 % using isogenic lines. It is interesting to note that even during the severe epidemic of 1985, *R. secalis* induced yield losses of only 12 %, whereas under similar disease conditions *Septoria nodorum* can reduce the yield of susceptible wheats by 20 % (KARJALAINEN 1985). This might be due to the longer growing time of spring wheat in Finland compared to barley, and thus *S. nodorum* has a longer time to affect grain filling of wheat than *R. secalis* of barley.

Our cultivars appear to respond differentially to the disease. Birger, which was also infected but not to such extent as Pokko and Ida, did not seem to suffer any yield losses. It remains to be seen whether this is due to tolerance effect or not. In a previous glasshouse study ROWLING and JONES (1976) observed some evidence of tolerance after *R. secalis* infection.

In our study the yield loss of barley was apparently mainly due to disease-induced green-leaf area destruction. Thus the total photosynthetic area was reduced with the consequence that dry matter accumulation into grains was limited. This is in accordance with our observations, which showed that the 1000-grain weight was the main yield component reduced due to infection. In 1983, inoculation was made during flag leaf emergence, and it is known (TENG and GAUNT 1980) that such late inoculation principally reduces grain weight because the other yield components have already been formed. However, in 1986 inocu-

lation was made immediately at the three-leaf stage and repeated three times. In this experiment all yield components of susceptible Pokko and Ida were affected, but most of all the 1000-grain weight.

Our results confirm some previous glasshouse and field trials (ROWLING and JONES 1976, JACKSON and WEBSTER 1981) where *R. secalis* infection mainly lowered grain weight and the number of grains per ear. There is some evidence (GALLAGHER et al. 1975) that although photosynthesis post-anthesis generally provides the majority of the dry matter in the barley grain, under extreme conditions considerable part of the final grain weight can also be provided by retranslocation. Therefore early infection can also influence the final grain weight.

It is evident that the yield loss caused by *R. secalis* is not accounted for merely by the reduced amount of green-leaf area duration. *R. secalis* is a necrotrophic pathogen influencing the diseased barley plant in several ways. Abnormal stomatal behaviour occurs after the fungus has penetrated into leaves (AYRES 1972, BRANCHARD and LAFFRAY 1987). Increased transpiration of scald infected leaves has also been observed (AYRES and JONES 1975). In addition, *R. secalis* excretes toxic molecules, such as Rhyncosporoside-type compounds (AURIOL et al. 1978), which have been found to affect the loss of turgor in young plants. Recently, MARTIN (1986) showed that photosynthesis in barley leaves was reduced mainly at about the time of scald symptom appearance, but reduction was not observed before visible symptoms.

The above experiments show that the scald disease of barley can reduce grain yield by up to 3–12 % under moderate to severe infection conditions. Yield reduction is mainly due to lowered grain weight, which also implies that scald apparently reduces the hectolitre weight. There is large variation in the incidence of scald disease between different years, regions, and fields. The occurrence of scald is not uniform even within a single field, and

the spatial distribution of disease units in a pathosystem is the most important factor influencing field estimations of disease and yield loss (TENG 1983). Therefore, it is extremely difficult to predict how much scald can totally reduce Finnish barley yields because extensive field surveys of disease incidence would be required to address this question. JAMES et al. (1968) developed a yield loss — disease severity relationship equation, which based on the observation that there is a linear relationship between yield and the percentage of lamina area affected by leaf blotch on the flag and second leaves at growth stage 11.1. However, more recently it was demonstrated (KHAN and D'ANTUONO 1985) that third leaf disease amount correlates best with observed yield loss.

Recent advances in using remote sensing assessment method and their implications (NUTTER and CUNFER 1988) that they may predict yield losses caused by *R. secalis* relatively accurately might give an easier and more effective way to evaluate the national losses of barley crop induced by scald disease. It seems evident that for the past two decades, and even before that (MAKELÄ 1974), scald has been the major leaf disease of barley of significant economic importance in Finland. The most serious losses have occurred in wet summers when humidity has provided ideal build-up conditions for the disease.

An effective and the economically cheapest form of crop protection is to use disease resistant cultivars (DOODSON 1981). There are several barley cultivars and lines possessing major genes for scald disease resistance (WEBSTER et al. 1980), which can be incorporated into well-adapted barley backgrounds. However, breeding scald-resistant barleys in Finland is still at an early stage, and all cultivars currently grown in Finland are fairly susceptible to the disease (KARJALAINEN, unpublished).

Chemical control of leaf spot diseases of barley is relatively new in Finland, and two fungicides (Bayleton and Tilt) have been used

to some extent. The present study shows that fungicide treatments clearly prolong the green-leaf area duration with the consequence of increased grain weight compared to the untreated controls. Yield increase due to fungicide treatment in this experiment varied between 5–20 % compared to untreated controls. The current barley prices in Finland imply that the benefit due to fungicide control should be about 250 kg/ha or more (KURTO 1986).

The present study suggests that fungicide treatment is profitable in cool and wet years,

when scald reduces yield by 5–10 % or more, but it is unlikely to be profitable in dry, rainless summers. The rational use of fungicides to control scald disease requires a disease monitoring system which allows the right timing of control. Simple forecasting techniques used e.g. for *Septoria nodorum* monitoring (TYLDESLEY and THOMPSON 1980) might prove out to be valuable aids for predicting the optimal time for the control of scald disease because scald is strongly dependent on temperature, the frequency and amount of rain.

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SELOSTUS

Rengaslaikkutaudin vaikutus ohran sadontuottoon

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Tässä tutkimuksessa selvitettiin rengaslaikkutaudin (*Rhynchosporium secalis*) vaikutusta kahden alttiin (Pokko, Ida) ja yhden kohtalaisesti tautia kestävän (Birger) ohralajikkeen satoon ja eräisiin satokomponentteihin. Kokeet tehtiin Helsingin yliopiston Viikin koetilalla vuosina 1983, 1985 ja 1986. Olosuhteet taudin leviämiseksi vaihtelivat vuosittain. Taudin kehitystä seurattiin lehti-analyysillä arvioimalla ylimpien lehtien tautisuusindeksi ja vihreän lehtialan kesto. Sadonkorjuun jälkeen laskettiin jyväsato ja 1000-jyvän paino. Lisäksi määritettiin Pokon ja Idan satokomponentit.

Rengaslaikku on viileiden ja sateisten kesien tauti, ja siksi vuosina 1983 ja 1986, jolloin oli lämmintä ja kui-

vaa, se levisi kasvustossa hitaasti. Vuonna 1983 tauti alensi kahdessa kokeessa Pokon jyväsatoa 10—11 % ja vuonna 1986 Pokon satoa 5 % ja Idan 3 %. Vuonna 1985 kasvukausi oli sateinen ja viileä, ja tauti levisi aikaisin ohra-kasvuston yläosiin ja aiheutti Pokolle 12 % satotappion. Tauti alensi huomattavasti yksilöpuitujen tähkien jyvän painoa ja koko tähkän painoa muttei merkittävästi jyvien lukumäärää tähkissä.

Rengaslaikkutauti lyhensi voimakkaasti ohran lehtialan kestoja, jolloin yhteyttävä kokonaispinta-ala pieneni ja jyvän kuiva-aineen muodostus väheni. Tämän seurauksena jyvien koko pieneni. Tilt-käsittely hidasti tehokkaasti taudin kehitystä ja piti lehdet vihreänä pidempään.

Lippulehden kehittymisen jälkeen tehty Tilt-käsittely paransi eri kokeissa jyväsatoa 5—20 %. Tulokset viittaavat siihen, että taudin torjunta kannattaa sadevuosina, jolloin patogeenipopulaation kehitys voidaan pysäyttää

oikein ajoitetulla torjuntakäsittelyllä ja siten estää lehtien ennenaikainen kuihtuminen. Sen sijaan poutavuosina taudin kehitys kasvustossa on hidasta, eikä torjunta useimmiten kannata.