THE EFFECTS OF FOLIAR DISEASES AND IRRIGATION ON ROOT DEVELOPMENT, YIELD AND YIELD COMPONENTS OF WHEAT (*Triticum aestivum* L.)

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Studies were conducted on three field trials of wheat cv. Kopara to investigate the lack of compensation by later determined components of yield because of early disease constraints. The investigation was based on the hypothesis that early disease reduces root development and thus causes the plants to be water constrained at later growth stages when soil water deficits usually occur. The reduced root development and soil water deficits may reduce the ability of the plant to compensate for reductions in early determined components. The hypothesis was tested by the application of irrigation to alleviate water stress.

In a disease free crop, the possible phytotonic effects of the fungicides benomyl and triadimefon on wheat were investigated. These fungicides had no phytotonic effects on shoot, root growth, or yield under the prevailing conditions.

The effect of disease on root development was analysed by root length measurements. Disease present in the crop at any stage of growth affected root development. Root development in the upper zones of the soil profile was reduced more by disease compared to those zones below 35 cm. A full disease epidemic reduced root development more than an early or late disease epidemic. The early and late disease epidemics had similar effects on root length. Alleviation of early disease constraints enabled greater development of roots to offset any earlier

reductions.

Soil water deficits increased root development in the lower zones of the nil disease plants. The presence of adequate soil water from irrigation reduced the requirement for further root growth in all treatments.

In the 1981-1982 field trial a full disease epidemic reduced yield by 14% whereas an early disease epidemic reduced yield by 7%. The reduction in yield was attributed to a lower grain number. With irrigation the yield reduction in the full disease plants was 12% whereas in the early disease plants the reduction was only 2.4%. This indicated that plants affected by the early disease epidemic were water constrained. In this study, the results suggested that, for conditions prevailing in Canterbury, the supply of water at later growth stages increased grain weight in plants which were subject to early disease epidemics. This suggests that reduced root development caused by early disease and soil water deficits may prevent compensation by grain weight.

Water use was similar in all disease treatments. After irrigation the irrigated plants of all treatments used more water. Disease affected water use in relation to yield production however, and was better expressed by water use efficiency. Water use efficiency was reduced in the full disease plants. A stepwise regression analysis suggested that water use efficiency was affected directly by disease at later growth stages, and indirectly via an effect on total green leaf area at early growth stages.

This study partially proves the hypothesis that reductions in root development caused by an early disease epidemic may constrain the plants at later growth stages when water deficits usually occur. It was shown that the reduction in root development caused by disease could be counteracted by irrigation. In this respect, water served as a tool to study the effect of disease constraints on the yield of wheat.

A knowledge of cereal crop physiology, root growth and function is used to explain and discuss the observations made in this research programme. The results are discussed in relation to the way in which disease affects yield through its effect on root development. The

possible reasons for the continued effects of disease even after the control of disease at later growth stages is discussed. The economic use of fungicides and water in diseased crops are also outlined.

Suggestions for future studies on disease-yield loss relationships are provided. The repetition of these experiments in different sites and climatic regions could provide information which may be incorporated in disease-yield loss simulation models. This could then be used to predict root development and water requirements of diseased plants, and provide a basis for economic use of fungicides and water, and for better disease management programmes.

KEY WORDS : wheat, foliar disease, disease assessment, root development, fungicides, phytotonic, water use, water use efficiency, water deficit, stripe rust, speckled leaf blotch, yield, yield components.

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CHAPTER ONE

INTRODUCTION

1 The need to study disease effects on root development

Crop loss is a significant constraint world-wide to increased food productivity. It is estimated that pre-harvest losses caused by diseases, insects and weeds are about 35%, post-harvest losses are reported to be 20%, and consumer wastage is 10% of the total production (James, 1980). The pre-harvest and post-harvest losses in cereal grain for 1975 were estimated at 748 million tonnes (FAO, 1976). During the same year, the estimated world grain deficit was 37 million tonnes. This clearly illustrates that a shortage of world cereals may be overcome by reducing crop losses.

Diseases are a major causal factor in crop losses. The phenomenon of crop loss was recognised by farmers and scientists before the science of plant pathology was developed (Main, 1983). However, we are still attempting to understand, define and measure the impact of such losses upon agricultural productivity. Crop losses from plant diseases are a result of disease epidemics. The epidemics involve host, pathogen, environment, and human activities interacting with other factors in space and time (Browning et al., 1977). The science of plant pathology operates from the base positions of the disease triangle: environment, pathogen, and host (Brown and Morgan, 1980). Before 1853, it was believed that unfavourable weather caused disease, and this resulted in crop losses (Horsfall, 1983). A typical example was the Irish famine which occurred during the cold and wet weather between 1845 and 1846. In 1853, de Bary shifted the philosophy of disease to the pathogen base, with the evidence that plant disease was caused by a living entity called fungus. Since then, the emphasis of disease studies was on causal organisms (fungi, bacteria, nematodes). However, after 1950, the study of the influence of weather on plant diseases once again gained importance, and was related to the growth and development

of the pathogen. Van der Plank (1963) provided a base for the principles of quantitative epidemiology, which led to the development of disease epidemic simulations through the use of computers. Simulation models have often been based on incomplete biological knowledge (Brown, 1978), and thus they have not always provided useful information on the interactions between the host, pathogen and environment. However, they have helped to formulate control strategies to avoid part of the disease epidemic and thus crop losses. Link (1932) suggested that pathology was a biological discipline in the same sense as physiology, as they are both concerned with functions and processes of the pathogen and host respectively. To understand the functional disorders of the host in plant pathogen interactions, the branch of 'physiological plant pathology' was conceived. Now, the emphasis is on the mechanisms involved so that such studies may provide answers to the questions about disease effects on the host and the subsequent effect on yield.

Most studies of physiological plant pathology have concentrated on processes such as photosynthesis, respiration, translocation and their consequent effects on shoot growth. The study of roots in relation to host pathogen interactions has been confined mainly to crops where the roots are the source of economic yield (e.g., sugar beet (Beta vulgaris L.)). Exceptions to this are where roots themselves are affected directly by pathogens (e.g., take-all of cereals, damping off, root knot and cyst nematode problems). Root studies in relation to foliar disease have been limited and confined mainly to pot and glasshouse experimental conditions. Roots and shoots are integral parts of a plant, and are interdependent. Therefore, any interference with the physiology and function of one of these parts could affect the other, and the subsequent performance of the plant as a whole. In the field, where crop plants are exposed to variable soil and environmental conditions, foliar pathogens may have serious and unexplained effects on the growth and yield of a crop. Field studies on the effects of disease on both shoots and roots may provide further insight into disease yield-loss relationships and a probable explanation as to how disease causes yield reductions.

Yield loss assessment studies through experimental programmes are now interrelated with modelling to provide control strategies. With the aid of computers, modelling has developed into a practical tool for plant pathologists. Field trials have generally been used to formulate disease control strategies. Unfortunately, the empirical approach is only practical for testing a few possible alternative management and control practices. Empirical modelling is also useful for predicting epidemics. Crop disease management practices developed empirically have advocated excessive use of chemicals. The increasing production costs and concern about the long term impact of chemical control practices on the environment creates the need for a more detailed understanding of disease management systems. Until recently, plant pathologists have quantified yield loss directly with the amount of disease present in a crop through empirical models such as critical and multiple point models (James, 1974; James and Teng, 1979). These models do not explain how disease reduces yield nor interpret the interaction between disease and yield.

The development of plant physiology as a discipline has enabled the construction of explanatory plant growth, or mechanistic, models (Thornley, 1976). Unlike empirical models which represent a statistical summary of what is observed, mechanistic models provide an understanding of the processes causing the observed relationships among variables (Schoener, 1976). This has been made possible through computer simulations linking together several subroutines that describe separate physiological processes, for example, photosynthesis, transpiration, respiration and nutrient transport (de Wit, 1978). As plant stress relates to plant growth and yield, factors that cause stress, whether biological (e.g., weeds, insects or pathogens) or environmental (e.g., drought, nutrition), may impose physiological stress on the crop. Such factors which affect the physiological processes may be observed by measuring the effects on plant growth and yield. The mechanistic plant growth models have thus far not accounted for disease related plant stress (Rouse, 1983). Epidemiologists have also ignored the quantitative interactions between pathogen population, disease and host plant growth.

Increasing interest by plant pathologists in mechanistic yield-loss models has led to attempts to link disease models to physiological plant growth models. This link requires a quantitative understanding of the physiological effects of disease on plant growth. For some disease-yield loss systems, e.g., cereal rusts, there is sufficient knowledge to relate the effect of the pathogen to crop yield on a physiological basis. However, there is a lack of quantitative data to relate the percentage of visual disease symptoms to the degree of change in these physiological processes (Rouse, 1983). Although a fully quantitative approach may be limited, the measurement of plant growth variables such as total dry weight, green leaf area, and total leaf area measured as affected by disease and disease induced stresses (e.g., water stress) may be used in plant growth models. With regard to plant growth variables of diseased plants there is information on above ground plant parts (van der Wal and Cowan, 1974; Siddiqui, 1980; Lim, 1982). However, there is no information on root growth of field-grown cereals as affected by foliar disease which may be incorporated into plant growth models. This information may contribute towards yield loss studies in understanding the limitations to yield of cereals at different growth stages and their importance.

During the crop cycle, the growth of cereals and the development of components of yield may be affected by constraints, therefore reducing final yield. Cereals have a capacity for yield compensation, reflected in the negative correlations frequently reported between yield components (Evans and Wardlaw, 1976). For example, the later determined components of grain yield are known sometimes to compensate under favourable conditions for earlier losses or restrictions of growth and development. The compensation by different components may be limited by the genetic capacity of a cultivar. A reduction in plant density was reported to be compensated for by greater tillering (Bremner, 1969) and by increased grain number and grain weight (Kirby, 1969). A reduction in grain number per ear was reported to be compensated for by greater grain weight (Bingham, 1967; Rawson and Evans, 1970). A compensation in grain weight may be limited by genetic capacity as well as the amount of photosynthates available for grain filling (Bingham, 1967).

The production and reduction of primary yield components as affected by disease has been studied by Doodson et al. (1964a), Carver and Griffiths (1981, 1982), Lim (1982), and Gaunt and Thomson (1985). Disease epidemics during the early stages of crop growth reduced the yield components determined during those stages, and thus reduced grain number per unit area. If disease severity increases above threshold levels after anthesis, grain weight may also be affected because of a limited amount of photosynthate (Gaunt, 1978). Therefore the effects of early disease on components such as grain number and ear number may be compensated through later determined components (e.g., grain weight) especially if disease is controlled at later growth stages. However, it has been reported (Carver and Griffiths, 1981, 1982; Gaunt et al., 1982; Lim, 1982) that reduction in early determined yield components (e.g., grain number) from disease constraints were not compensated for by later determined components (e.g., grain weight). The lack of compensation in these experiments may be linked to limitations in photosynthates required for grain growth. Foliar disease-causing pathogens can affect the photosynthetic process (Misaghi, 1982) and also act as an alternative sink for photosynthates, reducing the amount available for plant growth. The growth of roots is dependent on photosynthates translocated from the shoot. The rate of root growth is initially exponential and then linear up to anthesis (Gregory et al., 1978b). When disease occurs during early growth stages, therefore, it may reduce root growth more than when disease occurs at later growth stages (e.g., Martin and Hendrix, 1974). A reduced root system cannot exploit the soil for its nutrients and water as much as a well-developed root system, and may result in plant water deficits earlier in soils where water supply is limited. As plant water deficits affect physiological processes (Kramer, 1963), plant growth and yield may be adversely affected.

It is hypothesised that early disease constraints may limit root growth which could, at a later stage, lead to water stress when soil water deficits occur, thus imposing a limit on photosynthesis. Knowledge of cereal yield physiology is used to study the effect of disease on plant growth, and to test the above hypothesis.

2 Cereal yield physiology at the whole crop level

Cereal crop production, under a given set of climatic and soil conditions without disease, weeds or other biological limitations, is dependent on resources such as light, water and nutrients. Light is an important environmental factor controlling crop growth and development, as it influences photosynthesis and thus dry matter production. The influence of light may vary depending on its duration and intensity. Limitation of light in the field through reductions in photosynthetically active radiation was reported to reduce the rate of crop growth and thus dry matter production (Willey and Holliday, 1971; Fischer, 1975). Such limitations to light may occur because of seasonal variation and from self-shading caused by high plant density and closed canopies. Water availability is one of the major factors limiting production in many areas of the world. In plants, limitations to water availability cause stomata in the leaf to close, thus reducing photosynthesis (Slatyer, 1973). Furthermore, water is a transporting medium and an important component of many biochemical processes. Water deficits, therefore, lead to reductions in a number of physiological activities such as nutrient uptake, carbohydrate and protein metabolism, transport of ions and metabolites, and general plant growth and development. These physiological constraints ultimately lead to a reduction in crop productivity. Nutrients are another important resource for crop development. An adequate supply of nutrients at all stages of growth is an important requirement for plants to be able to realise their yield potential (Sneep et al., 1979). Nitrogen is one of the major components of nutrients, besides phosphorus and potassium, and is required in large quantities for plant growth at all stages. For instance, responses to nitrogen have been obtained after heavy rainfall (Stephen, 1980) indicating that nitrogen is easily leached and thus needs to be replenished when lost from the soil. In soils with nitrogen deficiency and water deficits, the response to nitrogen is increased with irrigation (Drewitt, 1980). In winter sown cereals, rapid plant growth in spring creates an increased demand for nutrients and a large amount of nitrogen compared to the other elements. Edwards et al. (1983) reported that photosynthesis increased linearly with increasing nitrogen content of the leaf up to seven percent of leaf dry weight.

Nitrogen has been reported to increase leaf growth in wheat (Welbank and Widdowson, 1972; Welbank and Taylor, 1973). Increased crop productivity from nitrogen application may also occur through increased ear number (Dougherty et al., 1979) and spikelet number (Langer and Dougherty, 1976). Nitrogen and other minerals are also required for grain growth (Sneep et al., 1979). Limitations to nutrients could arise through direct competition from weeds and would depend on the competitive ability of the crop to extract nutrients from the soil.

A well-developed root system which can make use of water and mineral resources is an important factor in yield determination (Evans and Wardlaw, 1976). The rooting pattern of a plant depends on frequency of rainfall and on soil properties such as water holding capacity, porosity and nutrient status. In regions where rainfall is infrequent and low, the roots need to grow into a greater volume of soil and to reach depths where stored soil water may be present. However, there has been some controversy as to whether wheat requires an extensive root system, or a restricted root system that conserves water for the late season growth. An extensive root system may not necessarily mean greater use of water, as water use is dependent on evapotranspiration which is influenced by atmospheric demand. Winter wheat develops an extensive root system (Troughton, 1962) because of a longer growth period. This would enable the plant to cope with the increased demand for water late in the season. Canadian studies suggested that an extensive root system is preferable, but Australian research indicated that restricted root systems may reduce water use prior to anthesis, leaving more water available during the grain filling period (Passioura, 1972). A well-developed root system enables better exploitation of the soil for nutrients. Nutrient uptake by roots involves a metabolic process (Russell, 1977) and is dependent on metabolites from the shoot. The movement of nutrients to the shoot is influenced by the rate of transpiration (Russell, 1977). Therefore, effective root growth and function are dependent on the shoot.

In cereals the entire shoot is photosynthetically active, and uses light to produce photosynthates required for crop growth. With increases in light intensity, the rate of photosynthesis usually increases, but in a closed canopy, this increase is not linear (Edwards et al., 1983). This may be because the lower leaves are shaded by the

upper leaves, which intercept most of the light. In closed crop canopies, therefore, the effects of increased light duration or intensity are not beneficial to the entire plant, as the light in excess of that absorbed by the upper leaves is unable to reach the lower leaves. To use this resource effectively there must be maximum light interception by the entire shoot. This may be achieved by greater leaf area index (photosynthetic area) and suitable canopy architecture to intercept maximum light by all the leaves of the plant. If the plant has erect leaves, lower leaves would also be able to intercept light. The erectness of leaves depends on leaf angle which varies with genotype, especially after the vegetative period (Fischer, 1983). Leaves are usually erect when emerging from the subtending leaf sheath at the top of the canopy. However, in most wheat and barley (Hordeum vulgare L.) genotypes the leaf lamina bends, resulting in a convex (planophile) canopy in a dense field population (Fischer, 1983). This may reduce light interception by the lower leaves. In some species, usually ones with small leaves (e.g., rice (Oryza sativa L.)), the leaves resist bending, thus producing erect leaves and open canopies. This favours a greater interception of light by the entire canopy. Increasing photosynthetic area and erect leaf production are two growth characteristics that may contribute towards increased photosynthesis, thus increasing crop growth (Austin et al., 1980; Fischer, 1983).

The rate of crop growth (increase in dry weight per unit land area per unit time) determines the total biomass production of the crop (Thorne, 1974). The crop growth rate depends on the leaf area index (LAI) of the crop and the net photosynthetic rate (Thorne, 1974). Net photosynthetic rate depends on the season and the climatic region, whereas leaf area index varies with cultivar, and the effects of season and fertilizers (Watson, 1947, 1952). Differences in the photosynthetic rate per unit leaf area have been reported in wheat genotypes (Evans and Wardlaw, 1976) with higher rates of photosynthesis in older genotypes (Dunstone et al., 1973; Evans and Wardlaw, 1976) and a reduction in newly evolved genotypes. However, differences in such studies are complicated by the age of leaf chosen, light conditions during leaf growth, and by the varying photosynthate requirements of the growing regions of the plant (Austin and Jones, 1975; Evans and Wardlaw, 1976). Watson (1947) concluded that dry matter yield depended more on

differences in leaf area index than net photosynthetic rate. However, these two factors are not independent, and although net photosynthetic rate increased with increasing LAI and reached a peak at a value of nine (Watson et al., 1976) it declined between values of nine and twelve (Stoy, 1965; Watson et al., 1976). This decline was associated with greater plant density and low radiation, or low incident radiation on the lower leaves of the canopy (Puckridge and Donald, 1967). Fischer (1983) concluded, therefore, that the rate of crop growth depended on the amount of photosynthetically active radiation intercepted by the canopy under non-stressed conditions.

In cereals, the economic yield is the grain, and this is made up of various components determined throughout the growth period of the crop. Grain number is determined over a long period of time, whereas grain weight is determined during a relatively short grain filling period at a late growth stage. Brocklehurst (1977) suggested that grain weight is dependent on the number of endosperm cells, which is determined at anthesis. Thus, events which occur before the grain filling period also determine the ultimate weight of the grain; for example, the amount of photosynthates available at anthesis. During the grain filling period, there is an allocation of current and stored photosynthates to the grain, permitting grain growth (increase in dry matter) to occur when shoot (minus the ear) dry matter is either stable or declining. Crop growth rate and its components (leaf area index and net assimilation rate) may be more relevant and relate more significantly to grain number than to grain weight. Contribution to grain dry matter is made mainly by photosynthesis after anthesis (Evans and Wardlaw, 1976) during which leaf area index declines rapidly. This is contrary, however, to reports of the contribution made by stem reserves. Thorne (1974) suggested that stored stem reserves contributed 20% of cereal grain yield, whereas Gallagher et al. (1975, 1976a) reported that in six crops of wheat and barley, an average of 43% of grain yield was contributed by stored stem reserves. The percentage contribution by the stem reserves, however, depends on the amount of stress affecting the plant after anthesis (Evans and Wardlaw, 1976; Bidinger et al., 1977). In wheat, Bidinger et al. (1977) reported that an average of 12% to 22% of grain weight was contributed by stem reserves depending on the amount of soil moisture stress, with a greater

contribution under severe stress. Under non-stressed conditions, the contribution of current photosynthates to grain yield may be greater compared to that made by stored reserves. In cereals where photosynthate is limiting (i.e., source limited), increasing leaf area index at anthesis, lengthening the duration of the grain filling period, and delaying the onset of the decline in photosynthetic capacity may all increase yield (Austin, 1982), through an increased source of photosynthates for grain filling. Thorne (1974) suggested that in temperate climates, sink capacity (grain number and grain size) may affect the distribution of photosynthates, as has been found in sugar beet and potatoes (Solanum tuberosum L.) (Thorne and Evans, 1964; Nösberger and Humphries, 1965). Increasing the amount of photosynthate, therefore, may translate to further increase in yield only if there is sufficient sink potential (grain number and grain size) under non-stressed conditions.

There has been a large increase in world cereal production in the last 80 years, both by increasing potential yields through breeding and increased area under cultivation. The average yield of wheat and barley increased by 80% and 84% respectively between 1947 and 1977 (Silvey, 1978). Improvements in genotype contributed 50% and 31% of these increases in wheat and barley respectively (Silvey, 1978). Breeding strategies have incorporated measures to utilise the resources of light, water and nutrients to maximise yield. Such increases in yield were correlated with increased harvest index and not with total productivity (total biomas yield) per unit area (Singh and Stoskopf, 1971; Austin et al., 1980). The increase in photosynthate partitioning between the ear and the remainder of the plant (Jain and Kulshrestha, 1976) and the reduction in straw weight have contributed towards a higher harvest index. Reduction in plant height in new genotypes (semi-dwarfs), through a reduction in internodes, has enabled greater utilisation of nutrients, and reduced lodging in comparison to older genotypes. However, in addition to the reduced plant height and straw weight, Mackey (1973) reported that the root system of some new genotypes was also reduced. Reduction in root systems may be detrimental to crop growth when soil water is limiting. In wheat, deep rooting characteristics may be selected independently of shoot structure, and therefore deep root systems may be developed in the new

genotypes (Mackey, 1973).

To exploit the increased yield potentials of the recently evolved genotypes, and to produce maximum yield, the crop needs to be grown in an optimum environment with adequate water and nutrients. Furthermore, the crop must be free from weeds, pests and diseases which may interfere with the physiology of the crop. Zadoks and Schein (1979) defined yield as the measurable produce of a crop, and crop loss as the reduction in either quantity and/or quality of yield. Theoretical yield is that yield under the best conditions according to calculations based on plant and crop physiology, and which involves a high input of nutrients, pesticides, and growth regulators. This yield, however, is not attainable. Zadoks and Schein (1979) suggested that yield of a particular crop may vary between the lowest primitive yield and the highest attainable yield, depending on various factors such as genotype, environment, nutrient input, and crop losses. Between the two extreme yields are the economic, and actual yields. Primitive yield is that yield obtained when a crop is grown under the prevailing conditions, with no additional inputs. Attainable yield is that yield which is obtained with good crop husbandry when grown under optimal conditions, using all available technology, as, for example, in some experimental trials. This yield may not necessarily be an economical one, as the additional production costs may be greater than the returns. Economic yield is that yield obtained by management practices which aim to maximise returns. Economic yield is always lower than the attainable yield, because of diminishing returns with increased production. Actual yield is that obtained under prevailing crop husbandry practices through necessary inputs, and is generally higher in developed countries where the practices are better and more advanced compared to developing countries. The economic yield in developed countries usually approaches the attainable yield. Good management practices involve soil and water management, seed certification, fertiliser application, and weed, pest and disease control through proper use of chemicals.

Climatic conditions in New Zealand are generally suitable for cereal growth. The average actual wheat yield in New Zealand is about 4 t ha^{-1}, which is below the attainable yield (about 12 t ha^{-1}). Although the attainable yield is high, it is not realised because of various constraint factors. Under optimal management practices, the required

amounts of nutrients are supplied and are thus not limiting. The duration of sunshine in New Zealand is greater (average of 168 hours per month) compared to the United Kingdom (average of 125 hours per month), and thus may not be a limiting factor. One of the limitations to yield may be water, which depends on rainfall or irrigation. In some growth seasons, rainfall may be very low, and the crop may depend mainly on stored water in the soil profile. In situations where stored soil water is found at lower depths in the soil profile, the root system may not reach such depths, leading to crop water stress. Limitation to crop water requirement could, over an extended period of growth, reduce crop yield (e.g., Day et al., 1978). Nitrogen may be a limiting factor when there is high rainfall, as it may be leached. The supply of nitrogen may need to be replenished if nitrogen is lost from the soil profile during crop growth.

Another limitation to achieving attainable yield is disease. A full disease epidemic of powdery mildew (caused by Erysiphe graminis DC. f.sp. hordei) on barley (Lim, 1982) and speckled leaf blotch (caused by Mycosphaerella graminicola (Fuckel) Schroeter) on wheat (Gaunt and Thomson, 1985) were reported to reduce yield through reduced grain number and grain weight. Within a crop the potential yield is set by the different yield components during crop growth. This potential, however, may not be met because of factors which reduce the set potential. Potential yield reduction because of early disease constraints are normally not compensated for at later stages of growth (Lim, 1982) under Canterbury conditions. The lack of compensation may be attributed to the prolonged effects of disease affecting crop physiology through reduced leaf area (Lim, 1982; Gaunt et al., 1985), and root growth (e.g., Martin and Hendrix, 1974; Ayres and Zadoks, 1979). All these limitations to yield affect both grain number and grain weight.

3 Cereal yield physiology at the whole plant level

The physiology of grain yield is mainly concerned with photosynthesis and the use of its products in the plant (Austin and Jones, 1975; Bingham, 1976). The greatest individual component of grain yield is carbohydrate derived from photosynthesis, which constitutes 80% of the grain in chemical composition. It is well established that most of the carbohydrate in the grain comes from photosynthesis after anthesis (Evans et al., 1975; Bingham, 1978). However, the potential of the plant is largely dependent on earlier events, as yield potentials are determined before anthesis. Yield of cereals is the product of complex interactions of many primary plant characters (including genetic variability) with each other and with the environment.

The components of yield are ear number per unit area, grain number per ear and mean grain weight. These components are determined sequentially at different times during crop growth. Many weeks elapse between the first and the last component being fixed, and this may be influenced by weather or management at a critical time during the season (Austin and Jones, 1975). Yield components are generally inversely correlated with each other, and they vary considerably with the growing season at any particular location (Thorne, 1974; Simmonds, 1979). The growth of different parts of the plant which contribute towards final yield depend on their innate capacity for growth in a prevailing environment, and on the extent to which its early growth has been supported by the supply of materials from the remainder of the plant.

The potential number of ears per plant is partly dependent on the number of tillers produced. Tiller buds are produced at the axils of lower leaves, thus giving a hierarchy of main stem, primary and secondary tillers (Bingham, 1978). The initiation and growth of tiller buds is influenced by the supply of photosynthates (Fletcher and Dale, 1974) and by the growth regulator gibberellin (Kirby and Faris, 1972). Initially, tillers are dependent on photosynthates produced by the main stem (Lupton, 1966), especially from the subtending leaf on the main stem (Fletcher and Dale, 1974). Therefore, any climatic factor, nutrient status or cultural practice that may adversely affect the

photosynthesis of this leaf may reduce tiller growth and development.

Tiller production can be genetically controlled (Thorne and Blacklock, 1971; Langer, 1972). Within a genotype, tiller production is thought to be controlled by an apical dominance system (Leopold, 1949) and also by auxins (Suge and Yamada, 1965). The duration and rate of tiller production is influenced by light intensity (Aspinall and Paleg, 1964). High light intensity favoured tillering (Friend, 1965a). Low temperature favoured increased tillering (Friend, 1965a; Rawson, 1971; Brooking, 1979), whereas high temperature reduced tillering by increasing apical dominance (Friend, 1965a). Increased nutrient supply enhanced tillering (Aspinall, 1966; Langer, 1966). Nitrogen applied at early stages of growth stimulated tillering, especially in winter wheat (Fischer and HilleRisLambers, 1978; Scott, 1978; Dougherty et al., 1979). The rate of tillering increased with increases in leaf appearance on the main stem under a favourable environment during the vegetative phase of plant growth (Langer, 1972). Kirby and Riggs (1978) reported that the pattern of tillering was dependent on ear development. There was a relationship between the time of tiller emergence and the number of emerged leaves on the main stem, and the growth to maturity of the tiller depended on its size when the maximum primordium number was initiated at the apex.

Tiller production can also be affected by plant density, and Puckridge (1969) reported that a high plant density inhibited tiller production. Conversely, a lower plant density usually results in increased tillering (Gallagher et al., 1976b). Kirby (1967, 1969) showed that there was little change in final yield where plant density varied over a wide range, because of the compensatory negative correlation between plant density and tillering capacity of the cereal crop.

Tiller number reaches its maximum at the onset of stem elongation, following floret initiation (Jewiss, 1972). Tillers formed after this time usually fail to develop ears and subsequently die (Cannell, 1969). The growth of the emerged tillers initially depends on photosynthate produced by the main stem (Quinlan and Sagar, 1962; Lupton, 1966) and normally become independent of it after they develop three mature leaves, and when adventitious roots develop at their base

(Evans et al., 1975). Although tillers may become independent, however, they are still capable of integrated activity, and under adverse conditions, both nutrients and photosynthates may be induced to move between adjacent tillers (Wardlaw et al., 1965). Later formed tillers compete with the main stem and primary tillers for photosynthates at early stages of growth. These are considered a waste of plant resources, as the metabolites of these tillers are not usually translocated to the rest of the plant when they are dying (Donald, 1968; Rawson and Donald, 1969). Consequently, the size and final yield of the main stem and primary tillers may be reduced, as observed by Jones and Kirby (1977) and Kirby and Jones (1977).

The potential and final tiller number varies with cultivar, even when grown under similar conditions (Kirby, 1967; Bingham, 1978). Within a cultivar, the maximum potential number of tillers may not be realised because of death of both tiller buds and developing tillers. This may be because of an unfavourable environment (Wardlaw, 1974) and constraints to the supply of photosynthate (Friend, 1965a; Puckridge, 1968). Many tiller buds that are formed on the main stem do not develop (Kirby and Faris, 1972); this may be attributed to either nutrient stress (Aspinall, 1961) or water stress (Iljin, 1957), both of which affect the apices of developing tillers, resulting in their death. The extension and growth of tiller buds (Kirby and Faris, 1972; Gallagher et al., 1976b), and further development of the tillers (Thorne and Blacklock, 1971; Gallagher et al., 1976b) is influenced by photosynthate supply. From shading experiments, Fischer (1975) reported that the survival of developing tillers was reduced because of reduced photosynthesis. The rapidly growing tillers may be in direct competition with other meristems of the plant, and therefore tillers in an unfavourable position for photosynthate supply may die. Many of the emerged tillers produced by a plant may also be lost because of water stress (Barley and Naidu, 1964). In pot (Langer and Ampong, 1970) and field experiments (Day et al., 1978), tiller survival was reduced by prolonged soil water deficits from emergence to anthesis. As tiller survival is reduced in cereals, the majority of the final yield of a plant is accounted for by the high grain number and high grain weight of the main stem, coleoptile tiller and first two primary tillers (Cannell, 1969).

Grain number is the most complex component of yield, as it is determined by the number of spikelet primordia produced and surviving, the number of florets per spikelet which survive to anthesis, and the number of florets which set grains (Kirby, 1974). This component of yield is affected over the longest period of crop growth, i.e., the period of ear development from spikelet initiation through to several days after anthesis of each floret (Gallagher, 1979).

When active growth starts in the spring, the shoot apex elongates in preparation for ear differentiation. At this stage, leaf primordia are seen at the base of the shoot apex. Spikelet primordia are initiated by the shoot meristem at a constant rate, but often more rapidly than that for leaf primordia (Kirby and Faris, 1970; Kirby, 1973, 1974). The initiation of spikelet primordia may be considered as the end of the vegetative phase of growth (Austin and Jones, 1975), and is indicated by the appearance of double ridges on the spike (Bonnett, 1966). Development of spikelet initials occurs in acropetal and basipetal succession from the middle of the spike (Bonnett, 1966).

The spike is considered as the inflorescence in wheat and barley (Bonnett, 1966). The barley spike is indeterminate, and the maximum number of spikelets initiated is between 30 and 40 (Kirby and Faris, 1970). The wheat spike, on the other hand, is determinate, with the axis ending in a terminal spikelet (Bonnett, 1966). Between 15 and 25 spikelets are initiated (Rawson, 1970, 1971; Wall and Cartwright, 1974), depending on cultivar and environmental conditions. The duration of spikelet initiation is important in both barley and wheat, as it is positively correlated with the number of spikelets produced (Aspinall, 1966; Kirby and Faris, 1970; Rawson, 1970, 1971; Wall and Cartwright, 1974; Allison and Daynard, 1976). The duration is influenced by photoperiod and, in winter wheats, by vernalization. Rawson (1971) found that vernalization and extended photoperiod reduced the duration of spikelet initiation, which led to lower spikelet numbers, even though the rate was increased. A similar response to temperature was reported by Friend (1965a), Halse and Weir (1974) and Rahman and Wilson (1978). Increased nitrogen levels at the beginning of the reproductive phase increased the rate of spikelet primordium production and extended its duration, thus increasing the number of spikelets (Langer and Liew, 1973). The interaction of environmental factors also influenced

spikelet initiation within a cultivar (Aspinall and Paleg, 1963; Friend, 1965a; Bonnett, 1966; Puckridge, 1968; Langer and Dougherty, 1976; Kirby and Jones, 1977). For example, the response to photoperiod is dependent on temperature (Austin and Jones, 1975). High temperature and long photoperiod reduced the number of spikelets initiated (Aspinall and Paleg, 1963; Paleg and Aspinall, 1964; Friend, 1965b; Rawson, 1971; Lucas, 1972). High light intensities and low temperatures increased the number of spikelets initiated, thus increasing spikelet number per ear. At high plant densities, however, there was no increase in the number of spikelets per ear because of self-shading of leaves (Puckridge, 1968). The number of spikelets initiated is often correlated with the numer of leaves produced by the plant (Rawson, 1970; Wall and Cartwright, 1974), and this interrelationship is influenced by genetic and environmental factors, vernalization and the time of sowing (Aspinall, 1966; Rawson, 1970, 1971; Scott and Dennis-Jones, 1976).

After the maximum primordium number is reached on the barley apex, some of the distal spikelets cease to develop further and die. This is referred to as die-back or spikelet abortion (Bonnett, 1966; Kirby and Faris, 1970). Kirby and Faris (1970) suggested that competition for nutrients in the developing ear may account for spikelet abortion because of rapid growth of the central and basal primordia, which limits the supply of photosynthate to the primordia at the apex. This limitation may also be caused by the rapidly growing stem which is in a favourable position for the supply of photosynthates, and, therefore, reduces the photosynthate supply to the spikelets (Bingham, 1972; Kirby and Jones, 1977).

In wheat, however, not all spikelets develop to bear florets. Those at the base of the ear may be rudimentary, depending on cultivar and environment (Langer and Dougherty, 1976). Furthermore, fewer spikelets are formed in wheat than in barley, and the undeveloped spikelets are usually confined to the basal and terminal positions of the spike (Bingham, 1978). Further abortion of spikelets may occur because of die-back or abortion of florets in a spikelet.

Concurrent with spikelet differentiation, each spikelet primordium develops and differentiates into florets (Bonnett, 1966; Langer, 1972; Kirby, 1974). The initiation of florets begins a few

days before the end of the spikelet initiation phase, and overlaps with the formation of the terminal spikelet (Brooking, 1979). Floret initiation begins in the middle spikelets and proceeds acropetally and basipetally within a spike (Bonnett, 1966; Kirby, 1974). This sequence of development among spikelets is maintained throughout spike development, including anthesis and grain growth (Bonnett, 1966). In wheat, the maximum number of florets initiated in a spikelet is between eight and nine and occurs over a period of several days (Langer and Dougherty, 1976). The rate of floret initiation is influenced by temperature (Brooking, 1979), and this response may be further influenced by other environmental conditions. Williams (1960) reported that, with long day length, at 17°C, there was an interval of 1.5 days between the initiation of successive floret primordia. This interval may be reduced with increasing temperature up to an optimum level of 30°C (Rahman and Wilson, 1978).

Floret development begins soon after initiation and continues until anthesis (Brooking, 1979), by which time the number of potentially fertile florets per ear is determined (Evans et al., 1972). Following the differentiation of florets in wheat, many potential florets do not grow to anthesis (Bingham, 1978). This factor is more important in wheat than spikelet development. The development of potentially fertile florets depends on the physiological events that occur during the period from floret initiation to anthesis (Langer and Dougherty, 1976). The number of potentially fertile florets in wheat is increased by increased light intensity at any stage before ear emergence. The amount of photosynthates available to the ear is also important for floret development (Gifford et al., 1973; Anon, 1974). Austin and Jones (1975) suggested that a high concentration of photosynthates may be needed to overcome transport resistances in the ear, and to sustain the flow of photosynthates into the developing florets, especially in the distal spikelets. The competition for photosynthates between vegetative and reproductive structures (Langer and Dougherty, 1976) may limit the supply of photosynthates, thus preventing full development of florets (Bingham, 1972; Langer and Dougherty, 1976). Furthermore, as floret development occurs during part of the rapid stem elongation phase, photosynthate supply becomes an important interacting factor between the vegetative and reproductive organs (Brooking, 1979). The rate of floret

development may be increased by the supply of nitrogen between double ridge and floret initiation (Hanif, 1970; Langer and Liew, 1973). In addition to the importance of photosynthates and nutrients, floret development and viability is sensitive to water stress (Salter and Goode, 1967).

Within a spikelet, florets develop acropetally, and those florets which ultimately produce grains reach anthesis at the same time. Therefore, those florets further from the middle of the spikelet develop at a faster rate than the florets positioned in the middle. The distal florets which fail to set grains develop at a slower rate than those that produce grains (Langer and Hanif, 1973). This may be attributed to a well-developed vascular system in the central florets in a spikelet compared to the distal florets. In wheat, only the lower three florets are linked with the main vascular supply of the spikelet, while the distal florets are connected by sub-vascular elements (Hanif and Langer, 1972). The basal three florets, therefore, are better positioned within the spikelet for supplies of photosynthate, and may develop more than the distal florets. Under optimal conditions, however, especially with high nitrogen, more distal florets may set grain (Langer and Dougherty, 1976). In contrast, an unfavourable environment may limit the number of directly linked florets from setting grain to one or two (Hanif and Langer, 1972). The inhibition of other florets within a spike in barley (Nicolls and May, 1963), or within a spikelet in wheat (Hanif, 1970; Walpole and Morgan, 1970, 1973; Langer and Hanif, 1973) may be caused by correlative inhibition, as Hanif (1970) and Langer and Hanif (1973) observed that fertilization in the basal florets coincided with the beginning of degeneration in the potentially fertile ones. Hanif (1970), and Langer and Hanif (1973), suggested that events such as fertilization, followed by grain setting in the basal florets, exert a controlling influence on the developmental pattern of the potentially fertile florets. Hanif (1970), Walpole and Morgan (1973) and Langer and Dougherty (1976) suggested that this may be because of a restricted supply of photosynthates and plant growth regulators. Some florets may not remain viable until anthesis as microsporogenesis and pollen development are especially sensitive to water stress (Aspinall et al., 1964; Salter and Goode, 1967). Failure of floret fertilization because of structural deformities such as lack of receptive stigma, or

constraints to processes governing pollen meiosis (de Vries, 1971) or failure of pollination because of adverse conditions, results in reduced grain set (Wardlaw, 1974). Salter and Goode (1967), and Wardlaw (1974), observed that water is required during anthesis for expansion of lodicules, growth of the filament and dehiscence of the anthers, which are all dependent on turgidity of the tissues. Bingham (1966) and Fischer (1973), reported that a lack of water reduced pollen meiosis and that this process was also reduced by low light intensities and high temperatures. A poor supply of photosynthates to the filament prevented dehiscence of anthers, resulting in male sterility. Therefore, an adequate supply of photosynthates is required for fertilization and grain set (Evans, 1978). The position of the floret in the spikelet also influences anthesis (de Vries, 1971), as the two basal florets opened (Obermayer, 1916), while the more distal ones did not. Walpole and Morgan (1973) observed a similar trend and attributed it to dominance of some florets, possibly because of inhibition by plant growth regulators.

The final component of grain yield to be determined is the individual grain weight. This is determined through several phases of grain growth from anthesis to maturity (Evans et al., 1975), and may not be limited by genetic potentials and post-anthesis grain filling events alone. The potential size may be influenced by events before or immediately following anthesis (Evans et al, 1975). There is a correlation between time of floret initiation, ovary development and potential grain size (Kirby, 1974, 1977). Therefore, the potential grain size may be influenced by the size of the developing ovary before anthesis (Kirby, 1974; Dougherty et al., 1975). Following fertilization, initial growth predominantly occurs in the maternal epicarp and endocarp of the grain coat (Rijven and Cohen, 1961; Jennings and Morton, 1963). Free endosperm nuclei are formed after fertilization, at a rate depending on temperature (Hoshikawa, 1961), and this is followed by cell wall formation. The endosperm cell number increases rapidly following cell wall formation (Evans et al., 1975), and can vary from 100,000 to 150,000 endosperm cells (Brocklehurst, 1979). The number of endosperm cells produced may be influenced by temperature, with an increase in the rate of production at high temperatures, but a reduction in the duration (Hoshikawa, 1961;

Wardlaw, 1970). The final number of endosperm cells may be influenced by the supply of photosynthates available to the endosperm (Brocklehurst, 1977), the internal resistance to the transport of photosynthates (Bremner and Rawson, 1972), and by plant growth regulators (Wheeler, 1972, 1976). Low light intensity can cause a reduction in final endosperm cell numbers because of a reduction of dry weight accumulation by both stem and ear (Wardlaw, 1970). Water stress, however, increased the rate of endosperm cell division and initial rate of grain growth (Asana and Basu, 1963; Wardlaw, 1971). This increase may be associated with a compensatory mechanism for reduced seed set, caused by water stress during anthesis and fertilization (Wardlaw, 1971). However, with continued water stress, the duration of grain growth was reduced because of premature cessation of growth, resulting in reduced grain weight (Asana et al., 1958; Aspinall, 1965; Wardlaw, 1971).

The ultimate size of the mature grain is a function of the rate and duration of grain growth (Sofield et al., 1977b). It has been suggested that the rate and duration of grain growth is influenced by environmental conditions, and cultivars (Sofield et al., 1977a). Welbank et al. (1968) reported that an increase in light intensity reduced the duration of grain growth. An increase in temperature generally causes an increase in the rate of grain growth, whilst decreasing the duration (Brooking, 1979). Low temperatures during grain maturation may result in a high individual grain weight (Wardlaw, 1970, 1974). The effect of temperature on grain growth is further influenced by light intensity (Spiertz, 1977; Evans, 1978). Spiertz (1977) found that the rate of grain growth during the linear phase of grain filling was increased by increased light at high compared to low temperatures, whilst the duration was reduced, whereas Evans (1978) reported that the rate of grain growth was reduced by low light and temperature conditions whilst the duration was increased.

The period of grain growth involves grain filling, which is the process of starch synthesis from photosynthates translocated to the endosperm cells. In wheat and barley, the flag leaf and parts above this generally provide the main source of photosynthates for grain growth (Wardlaw, 1968; Rawson and Hofstra, 1969). Flag leaf photosynthesis fluctuates with the demand for photosynthates by the

developing grain (Evans and Rawson, 1970), thus the production of photosynthates fluctuates with demand (Neales and Incoll, 1968). The variations in photosynthesis and changes in the rate of grain growth with time result in a change in the relative supply of photosynthate from different organs (i.e., ear, flag leaf, penultimate leaf, leaf sheath) to the grain throughout their development (Buttrose, 1963). The pattern of photosynthate supply to the grain also depends on environmental conditions. Low light intensity and water stress, which decrease the total supply of photosynthates, result in the remobilisation of photosynthates from the lower parts of the plant (Wardlaw, 1967). Remobilisation of stored pre-anthesis photosynthates to the grain was reported by Asana and Basu (1963), Asana and Joseph (1964), and Bidinger et al. (1977). Under severe water stress conditions in the field, Gallagher et al. (1975, 1976a) reported that an average of 43% of the photosynthates in the grain was translocated from stored pre-anthesis photosynthates. Under prolonged water stress conditions during grain filling, less current photosynthates are available for grain growth, because of reduced photosynthesis (Fischer, 1973; Slatyer, 1973). Remobilisation of stored pre-anthesis photosynthates may be greater under prolonged water stress conditions than in low water stress seasons (Aspinall, et al., 1964; Fischer, 1973; Day et al., 1978). Individual grain weight, therefore, may not be affected because of compensatory translocation from stored photosynthates. Post-anthesis shading affected grain growth and reduced individual grain weight by limiting the post-anthesis photosynthate supply (Fischer, 1975). Wardlaw (1971) suggested that plant growth regulators may be involved in controlling grain growth. He reported that wheat roots were extremely sensitive to water stress during grain filling, and postulated that reduced cytokinin production caused by water stress (Itai and Vaadia, 1965, 1971) may be an important factor in premature senescence of the grains.

Grain filling commences with the deposition of starch soon after the free endosperm nuclei are isolated by cell walls (Wardlaw, 1974), and is correlated with total sugar levels in the endosperm (Jenner, 1970, 1974). Spiertz (1977) reported that sugar uptake by the grain was affected by temperature and light intensity. With an adequate supply of sucrose available to the grain, starch synthesis was reported to be

independent of light. However, temperatures between 15°C to 30°C increased the rate of starch synthesis (Jenner, 1968). The rate of grain filling is limited by starch synthesis within the grain (Jenner and Rathjen, 1975). The movement of sucrose to the grain continues until the moisture content of the grain decreases to about 40% (Miller, 1939), and the sucrose continues to be converted to starch while desiccation of the grain occurs (Spiertz, 1977).

It has been suggested that as the grain reaches its maximum size, vascular connections become limiting to sucrose flow, since transport within the grain is achieved by diffusion and not by well-developed vascular tissue. Hence grain growth ceases (Langer and Hanif, 1973; Kirby and Rymer, 1975). The cessation of grain growth, however, may not be entirely the result of a lack of photosynthate supply to the grain, as other factors may regulate the duration of its growth (Evans and Rawson, 1970; Rawson and Evans, 1971; Jenner and Rathjen, 1972a, 1972b; Sofield et al., 1977a). Jenner and Rathjen (1972a, 1972b) suggested that there is a rate-limiting stage in the accumulation of starch in the wheat grain which is responsible for restricting grain weight. Plant growth substances may also be responsible for regulating grain growth, as cytokinin has been reported to influence the process of starch accumulation and its duration (Michael and Seiler-Kelbitsch, 1972), and may also influence the cessation of grain growth (Wardlaw, 1971).

4 Constraints to yield

4.1 The effects of disease constraints on physiological processes

4.1.1 The effect of disease constraints on photosynthesis

Environmental stresses and pathogens adversely affect plant growth by interfering with rates and balances of physiological processes, especially photosynthesis, respiration, translocation, plant growth regulators and water relations. Pathogens often initiate or accelerate a sequential and complicated series of metabolic imbalances, rather than a simple change in only one process, such as photosynthesis

(Kozlowski, 1978).

Carbohydrate metabolism in plants is controlled largely by photosynthesis and respiration, and has been reviewed by Wheeler (1975), Daly (1976), and Kosuge (1978). Infection of plants by pathogens is known to alter one or more of the processes of photosynthesis. Daly (1976) reported that the apparent rate of photosynthesis of diseased beans was reduced, in contrast to respiration. Reduction in photosynthesis of plants infected by necrotrophic pathogens is caused by injury to tissues and loss of effective photosynthetic area (Daly, 1976). In diseases caused by biotrophic pathogens, however, nutritional interactions between pathogen and host suggest that changes in photosynthesis are metabolically regulated in the early stages of infection (Daly, 1976). This implies that changes in the rate of photosynthesis are important for pathogenesis and are not solely a result of injury to the photosynthetic apparatus. A reduction in the rate of photosynthesis is a characteristic response to rust and mildew infections (Allen, 1942; Scott and Smillie, 1966; Black et al., 1968; Magyarosy et al., 1976; Mignucci and Boyer, 1979; Ellis et al., 1981; Gordon and Duniway, 1982). Reduction in photosynthetic rates may be associated with changes in the level of chlorophyll (Allen, 1942; Scott and Smillie, 1966), chloroplasts (Dyer and Scott, 1972), chloroplastic RNA (Callow, 1973), and auxins and amino acids (Zscheile, 1974) of leaf tissues. Alteration in the photosynthetic rate of diseased tissues may also be as a result of a change in CO_2 uptake, by changes in stomatal behaviour (their opening and closure), and in the conductance of CO_2 in the mesophyll (Duniway and Slatyer, 1971; Hall and Loomis, 1972; Gordon and Duniway, 1982).

Little is known about the effect of disease-causing organisms on photosynthesis at the biochemical level (Buchanan et al., 1981). Montalbini and Buchanan (1974), and Magyarosy et al. (1976), reported that a reduction in the rate of photosynthesis in leaves infected with obligate parasites was partly because of a parasite-induced block in the non-cyclic electron chain. Buchanan et al. (1981) suggested that infection by obligate parasites altered the content of certain carriers involved in the electron transport chain, thereby reducing the rate of non-cyclic transport. Infection can also alter the products of photosynthesis through the inhibition of photosynthetic CO_2

assimilation, resulting in the production of amino acids instead of sucrose (Montalbini and Buchanan, 1974; Magyarsoy et al., 1976). A reduction in the activity of enzymes is also reported to lead to the production of organic acids (Magyarosy et al., 1976).

Buchanan et al. (1981) concluded that changes in photosynthetic rates in plants infected with obligate parasites stemmed from the diverse effects of mycelial growth during the course of infection. Differential effects of the biochemical reaction associated with photosynthesis may be observed depending on the stage at which infection occurs. However, the consequences of infection may be complicated by the different responses that pathogens elicit in susceptible and resistant plants.

4.1.2 The effect of disease constraints on respiration

A rise in respiration rate occurs at some stage in all disease infected plants, except for plants systemically infected with virus (Allen, 1953; Merrett and Bayley, 1969; Daly, 1976). For example, Daly (1976) reported that the rate of respiration was higher in rust-infected cereals than in non-diseased cereals. The initial increase in respiration before sporulation of the pathogen was attributed to host activity, and following sporulation, was associated with the presence of hyphae (Bushnell, 1970) and spore production (Bushnell, 1970; Daly, 1976). A detailed study by micro-respirometry of rust-infected host tissues revealed that host activity did not extend far from the infected areas (Bushnell, 1970), contrary to that previously reported by Bushnell and Allen (1962). The increase in respiration rate in regions at or beyond the tips of hyphae of rust fungi was negligible (Bushnell, 1970). Continued respiration at an increased rate, either by the host or the pathogen, would result in utilisation of photosynthates and depletion of reserves (Kosuge, 1978).

4.1.3 The effect of disease constraints on translocation

The effect of disease on respiration or photosynthesis, or both, is generally reflected in an altered movement of photosynthates within and between diseased tissues, and between diseased and non-diseased organs of the same plant (Koslowski, 1978). Accumulation of photosynthates at infection sites is common in diseases caused by biotrophic fungi (Doodson et al., 1965; Livne and Daly, 1966; Edwards, 1971). In some cases, accumulation of photosynthates in infected sites may be caused by the inability of the infected leaves to export photosynthates (Doodson et al., 1965; Livne and Daly, 1966). This may be attributed to an altered transport of photosynthates to the pathogen because of a concentration gradient between pathogen and host, resulting from utilisation of substrates by the growing pathogen (Livne and Daly, 1966). Daly (1976) suggested that the maintenance of a gradient through the separation of carbon by formation of fungal carbohydrates or increased synthesis of host starch may account for the retention of photosynthates in infected leaves. Neales and Incoll (1968) suggested that the accumulation of photosynthates increases the diffusive resistance in the leaf by reducing stomatal aperture. This may result in the reduction of photosynthesis in the infected leaves, and increase the need for the supply of photosynthates from other non-infected areas of the plants. Gaunt and Manners (1971b) reported that photosynthates were exported from uninfected shoots of wheat to shoots infected with loose smut (Ustilago nuda (Jens) Rostr.). They also observed a reduction in the rate of photosynthesis and an increase in the rate of respiration in infected plants. An increase in photosynthetic rate in uninfected leaves (Livne and Daly, 1966), and the increase in the rate of respiration in infected leaves, may be correlated with the movement of photosynthates (Daly, 1976). Allen (1942) and Bushnell (1967) reported abnormal starch accumulation in infected leaves, which was attributed to the conversion of accumulated photosynthates to starch (Inman, 1962; Schipper and Mirocha, 1969). Starch formation in host cells appears to be in excess of that required by the pathogen for its development, and in such cases, it may function as a source of carbon during sporulation in tissues when photosynthesis becomes inhibited (Bushnell, 1967).

Altered translocation may contribute as much to the economic damage of plants as the more obvious mechanisms of reduction in photosynthetic capacity, or loss of carbon through an increased respiration rate (Daly, 1976). For example, a reduction in root systems of cereals infected by rusts (Doodson et al., 1964a; Martin and Hendrix, 1967) or mildew (Last, 1962) may be attributed to the retention of photosynthates in infected leaves (Doodson et al., 1965). In smut-infected cereals there was also a decrease in root development (Gaunt and Manners, 1971a). Yield reductions caused by heavy infections of rust on the flag leaves, which supply the major portion of photosynthates to the developing grain (Rawson and Hofstra, 1969) may, therefore, be attributed to the retention of photosynthates.

Altered patterns of carbon translocation are of potential importance in understanding the effect of pathogens on the growth and development of the host in relation to yield and yield components of cereals. However, more quantitative details about pathways, as well as mechanisms, are required to fully understand the process (Daly, 1976).

In long term infections, a cumulative effect of disease on host growth may affect yield. Yield loss is often analysed by its effects on individual yield components, i.e., tillers m^{-2}, grains per ear and individual grain weight. Therefore, it is important to study the effect of disease on yield site potential, apical development, the time of infection, and the duration of disease (Gaunt, 1980).

4.1.4 The effect of disease constraints on yield potential

The retention of photosynthates by infected leaves decreases the amount of photosynthates available to growing regions of a plant, and this may have a detrimental effect on its growth and yield. As each yield component is determined at different crop growth stages, an analysis of disease and its effect on apical development and yield site potential may indicate the time at which disease constrains the yield. The potential number of tillers and florets is determined during the early stages of plant growth and development before stem elongation (Kirby, 1974). Therefore, any constraints during this period may affect the potential yield, especially when later determined yield components

(e.g., grain weight) do not adequately compensate for the earlier reduction in yield potential.

Brooks (1972) suggested that the number of fertile tillers produced per plant in barley may be reduced by powdery mildew infection during early crop growth. Although glasshouse trials showed that fewer tillers were produced by plants infected at early growth stages with powdery mildew (Carver and Griffiths, 1981), this effect was not observed in the field (Carver and Griffiths, 1982; Lim, 1982). Similar effects to glasshouse-grown barley were obtained with glasshouse-grown wheat infected with stripe rust (caused by Puccinia striiformis Westend.) (Doodson et al., 1964a). In the field, however, tiller production was not affected by early infection with speckled leaf blotch (Gaunt and Thomson, 1985). Thus, the number of tillers produced by field-grown cereals may not be affected by early disease. This may be because the pathogen does not manifest itself before the tiller bud initiation phase, or because disease severity during this phase is below threshold levels.

The physiological constraints imposed by disease on apical growth have been studied by Doodson et al. (1964a), Carver and Griffiths (1981, 1982), Lim (1982), and Gaunt and Thomson (1985). The apical development of certain barley cultivars (cv. Manapou, cv. Zephyr) was reported to be increased by disease (Lim, 1982), but Gaunt and Thomson (1985) reported that apical development of wheat was reduced by disease. In barley (Carver and Griffiths, 1981; Lim, 1982) and glasshouse-grown wheat (Doodson et al., 1964a), the production of spikelet and floret primordia was not affected by disease. This may be attributed to infection occurring after spikelet initiation, or a low disease severity during this period. However, Gaunt and Thomson (1985) reported that in field-grown wheat, spikelet primordium production was reduced by disease because of a slower rate of production. Floret primordium production was also affected by disease, resulting in a lower number of florets per ear.

The production of spikelet and floret primordia in wheat is affected by early disease, depending on the time and duration of infection. This may be explained by the competition for photosynthates between the pathogen and the apical meristem, as the pathogen acts as an

additional sink, depriving the apex of its requirements for normal development (Daly, 1976). Furthermore, it is possible that the host-parasite interactions may create imbalances in plant growth regulators which may affect apical development. As roots are major synthesisers of plant growth regulators (Itai and Vaadia, 1965, 1971), they may also be affected by foliar disease. This effect, however, is not yet well understood.

Kirby (1974) observed that all yield components, except grain weight, are initiated before growth stage 31 (Zadoks et al., 1974). Depending on the timing and severity of the epidemic, the initiaton of these components may be affected. The continued presence of disease may also affect further development of these components, and thus reduce the potential yield.

4.1.5 The effect of disease constraints on yield potential reduction

It has been established that powdery mildew reduces the final number of fertile tillers in glasshouse and field-grown barley (Last, 1962; Rea and Scott, 1973; Carver and Griffiths, 1981, 1982; Lim, 1982). Speckled leaf blotch (Williams and Jones, 1972; Gaunt and Thomson, 1985) and stripe rust (Doodson et al., 1964a) on wheat also reduced the number of fertile tillers. Fertile tillers may be lost even after ear emergence, depending on the severity and duration of infection. This may be attributed to the competition for photosynthates between the pathogen, main stem and tillers. The hierarchical system of tiller production (Austin and Jones, 1975) does not favour the lower order (secondary) tillers for the limited source of photosynthates. Disease may also affect the final number of tillers by reducing the growth and development of tiller meristems through imbalances in plant growth regulators. As tillers are capable of producing their own root systems (Sallans, 1942), and providing nutrients and water for their requirements, it is possible that root production and the growth of each tiller may be affected by foliar disease. The performance of these tillers, therefore, may be reduced, and their dependency on the main shoot increased. The loss of these tillers contributes towards a reduction in the number of potential grain bearing ears.

Disease may further reduce grain site potential by retarding the development of spikelet initials. Barley plants produce an indeterminate spike, and after the attainment of maximum primordium number, some of the distal spikelets cease to develop further, and eventually die (Kirby, 1974, 1977). Disease has been shown to have a major effect on the degeneration of spikelets in barley, even at low infection levels (Lim, 1982). The degeneration of spikelets may be further increased by disease (Carver and Griffiths, 1981, 1982; Lim, 1982), thus contributing to a reduction in potential grain number. In contrast, the wheat spike is determinate, and produces fewer spikelets than barley. The die-back of spikelets is less variable, and confined to the basal and terminal spikelets only (Bingham, 1978). Although disease retarded the development of spikelets in glasshouse trials, the final spikelet number was not affected (Doodson et al., 1964a). In field-grown wheat, however, Gaunt and Thomson (1985) reported that disease reduced the development of spikelets at all positions. The effect of spikelet die-back in both barley and wheat may be explained by a reduced supply of photosynthates for the growing apex, caused by the altered translocation of photosynthates to the sink created by disease (Edwards and Allen, 1966). Disease may also reduce the amount of plant growth regulators (e.g., cytokinins, gibberellins and auxins) produced by the roots that are required for the growth and development of the shoots.

A prolonged disease epidemic may have a detrimental effect on further development of the spikelets, resulting in infertile spikelets. This may be attributed to floret abortion, failure of fertilization of potentially fertile florets, and/or abortion of mature grains. Early and late disease (i.e., before and after growth stage 50) has been shown to increase the number of infertile spikelets in barley (Lim, 1982). In wheat, Mains (1930) found that severe rust infection resulted in sterile spikelets at the basal and terminal ends of the spike, attributed to florets being affected within the spikelets. An early disease epidemic in wheat was reported to reduce floret number (Doodson et al., 1964a), and was attributed to a reduction in the number of floret primordia surviving to form potentially fertile florets. Gaunt and Thomson (1985), however, observed that many florets were competent to set grain (i.e., potentially fertile), but failed to do so. They suggested that

the lower number of grains set may be causally related to the slower rate of floret development of diseased plants at early growth stages. Therefore, early disease epidemics above threshold levels may have a prolonged effect on the development of yield components, attributed to indirect effects of disease on crop growth, such as leaf area development and reduction in photosynthate supply.

Early foliar infections have also reduced root development in glasshouse studies (Last, 1962; Brooks, 1972; Martin and Hendrix, 1974), and if this occurs under field conditions, this may induce water stress in soils where water is limiting. The production of growth regulators such as cytokinins, which have important regulatory effects on photosynthetic activity (Michael and Seiler-Kelbitsch, 1972), may also be reduced by a reduced root system.

Growth regulators have also been shown to affect floret fertility in wheat (Bingham, 1966). Reduction in plant growth regulators in diseased plants may therefore have effects on their growth and function which have not yet been investigated.

Final grain number may be further reduced by disease because of grain abortion (Lim, 1982). This may be caused by a reduction in photosynthetic area in the flag leaf (Lim, 1982; Gaunt and Thomson, 1985), which is a major contributor to grain filling. In wheat, the distal grains within a spikelet are not directly connected to the vascular system (Hanif, 1970). In a late disease epidemic, therefore, they may be disadvantaged when competing for a limited source of photosynthates, resulting in grain abortion.

A late disease epidemic has been known to affect potential grain weight, as the full potential of the grain is not realised because of limitations to the supply of photosynthates (Carver and Griffiths, 1981, 1982; Lim, 1982; Gaunt and Thomson, 1985). Disease may also have an effect on grain size, as endosperm cell division is influenced by cytokinins (Wheeler, 1972) which may be reduced by disease.

The effect of disease on yield potential (production and reduction) provides a better understanding of the sensitivity of the crop responses to disease. It is evident that some growth stages are more sensitive to disease than others, depending on the time at which

yield components are initiated, and the duration of their growth and development.

4.2 Water constraints

Cereal crops are subjected to a variety of environmental stresses during their growth, including abnormal temperatures, unfavourable chemical and physical soil conditions, water stress and various diseases and insect pests. Water deficit, which produces water stress, reduces plant growth and crop yield to a greater extent than all the other stresses combined (Kramer, 1983). Water deficit or water stress refers to situations in which reduction in plant water potential and turgor interfere with the normal functions of the plant. The water potential at which this occurs depends on the stage of crop development and the physiological and morphological functions (e.g., cell enlargement and division, stomatal closure) under consideration. The responses of various processes to water stress have been summarised by Hsiao et al. (1976). Water deficits vary in intensity from small decreases in water potential to permanent wilting and death by dehydration.

In order to understand the effect of water deficits on plant growth, it is necessary to understand the significance of water in plant processes. Water is the major constituent of physiologically active tissue, and is essential in photosynthesis and in hydrolytic processes such as starch digestion. It acts as a solvent in which salts, sugars and other solutes move from the soil into plant cells and within the plant. Water is also essential for the maintenance of turgidity necessary for cell enlargement and growth, and for gaseous exchange through the stomata, which is kept open by turgid guard cells. Kramer (1959, 1963) suggested that many plant processes may be affected by water deficits.

4.2.1 The effects of water constraints on physiological processes

Water deficits in plants reduce net photosynthesis (Vaadia et al., 1961; Kramer, 1963; Boyer, 1976); mechanisms involved have been reviewed by Hsiao (1973). In wheat (as in other species), when stresses were of several days duration, the observed reduction in net photosynthesis was attributed to the closure of stomata, restricting gaseous exchange (Fischer and Kohn, 1966; Wardlaw, 1967; Frank et al., 1973). The reduction in photosynthetic rates under stress through stomatal closure may be related to the enhanced abscisic acid concentrations generally observed (Wright, 1969). Cytokinins may also be important as they are known to promote stomatal opening. Water deficits may result in reduced cytokinin production in roots, and therefore reduce the supply to shoots (Itai and Vaadia, 1965, 1971). Slatyer (1973) suggested that although there was a decline in net photosynthesis from stomatal closure because of short periods of water deficits, the photosynthetic system was not affected. However, Kramer (1983) suggested that water stress severe enough to cause stomatal closure simultaneously causes injury to the photosynthetic system. Accumulation of photosynthates in the leaf may occur when growth is reduced by water deficits, through a failure of either the translocation system to remove photosynthates from the source, or of the growing regions to utilise photosynthates (Wardlaw, 1967), which reduces photosynthesis.

Translocation of photosynthates is reduced in plants subjected to water deficits (Hartt, 1967). Bunce (1982) reported that in water-stressed maize (Zea mays. L.), day-time translocation of photosynthates from leaves was reduced and night-time translocation increased. This was attributed to alleviation of stress in the night when transpiration was reduced. Wardlaw (1967, 1969) concluded that reduction of translocation in water-stressed plants is caused by reduced growth which reduces sink activity. Slatyer (1969) and Wardlaw (1969) suggested that the phloem conduction system remains functional under moderate or severe stress, and therefore the main reason for a reduction in translocation is a change in photosynthesis and utilisation of photosynthates.

The partitioning of photosynthates among various organs determines their survival and later performance. The effect of water deficits on photosynthate partitioning to the different organs has been neglected. If water deficits occur when the plants are young, there may be a reduction in photosynthates partitioned to the roots, which may reduce root development. At later growth stages, especially when soil water is limiting, this may reduce the amount of water taken up by the roots. A better knowledge of water stress and partitioning is required so that the contribution of photosynthates towards sinks such as grain components is known. This may provide a better understanding of yield potential reduction in water-stressed plants.

4.2.2 The effect of water constraints on yield potential reduction

Grain yield and water use are linearly related, and therefore water deficits may affect both yield and water use (Day et al., 1978). As yield reduction is attributed to the reduction in yield components, the production, growth and development of yield components may be affected by water stress at certain stages of crop growth (Aspinall et al., 1964; Husain and Aspinall, 1970; Slatyer, 1973; Day et al., 1978).

Husain and Aspinall (1970) found that water stress reduced leaf primordium production and subsequent leaf growth in barley plants grown in a controlled environment. They also reported a reduction in the number of expanded leaves 18 days after sowing. Cell size was reduced and therefore leaf area was also affected (Kramer, 1969). Plant water deficits usually hasten leaf senescence (Asana, 1962; Fischer and Hagan, 1965), and consequently reduce leaf area after ear emergence. The reduction in leaf area was suggested to contribute towards a decline in total photosynthetic area (Hsiao, 1973).

The growth and development of tiller buds in pot-grown barley has been reported to be suppressed by water stress during the vegetative stage of growth (Aspinall et al., 1964). Day et al. (1978) reported reduced tiller production when field-grown barley plants were stressed in early stages of growth (i.e., spikelet initiation stage). In wheat, total tiller number was reduced by water deficits during the period of

rapid leaf expansion (Slavik, 1966). When early plant water stress was alleviated, tillering was enhanced, and this compensated for reduced tillering in the early stages. However, continued water stress (up to ear emergence) reduced late tiller production and thus the total number of productive tillers (Aspinall et al., 1964; Day et al., 1978). Up to 70% of the tillers produced on plants in the field may be lost because of water and nutrient stress during crop growth. Begg and Turner (1976) suggested, therefore, that the number of surviving tillers is dependent on water availability, with the rate of tiller death increasing with increasing water deficits.

In barley (Husain and Aspinall, 1970; Day et al., 1978), and wheat (Oosterhuis and Cartwright, 1983), prolonged water stress during the period of spikelet initiation decreased the number of spikelet primordia initiated. Distal primordia initiation has been shown to be sensitive to water stress (Husain and Aspinall, 1970), confirming similar work reported by Nicolls and May (1963). If a short period of water stress is alleviated, the rate of primordial initiation becomes rapid in order to compensate for the earlier reduction in spikelet number (Husain and Aspinall, 1970). They suggested that water stress during the differentiation of the apex may affect the distribution of regulatory factors such as nutrients and plant growth regulators. Thus the supply of essential substances may be monopolised by existing primordia. Consequently, the reduction in yield, through a decrease in the number of grains per ear, reported by Asana et al. (1958), may be associated with water stress before anthesis (Aspinall et al., 1964). A similar effect on field-grown barley was observed by Day et al. (1978), and was attributed to reduced potential spikelet number.

Further reductions to potential grain number may occur through reduced spikelet survival. As the rates of cell division and enlargement are sensitive to water stress (Hsiao, 1973), the rate of development of spikelet initials may also be reduced. If water stress is relieved before ear emergence, spikelet development may proceed at a faster rate (Fischer, 1973). However, prolonged water deficits from emergence to anthesis may result in under-developed spikelets and thus less grains per ear (Day et al., 1978). In wheat, water stress was reported to affect spikelet survival during the internode elongation period (Oosterhuis and Cartwright, 1983).

Water deficits during development and differentiation of the apex affected the rate of floret primordium production (Husain and Aspinall, 1970). The production of new primordia was more sensitive to water stress than the development of existing primordia. Since spikelet development appears to be affected less by water stress than floret primordium initiation, prolonged stress during this phase may reduce the potential number of grains per ear. This may also be a reason for the reduction in grain number reported by Day et al. (1978) for water stress from emergence to anthesis.

The processes from floret initiation to fertilization (e.g., floret development) may be sensitive to water deficits (Slatyer, 1973) and thus cause a reduction in grain number per ear or in the number of fertile ears. Fischer (1973) reported a reduction in the number of grains per spikelet related to water-stressed wheat plants during the period 15 to 5 days before emergence of the inflorescence, which was attributed to a reduction in the number of fertile florets (Oosterhuis and Cartwright, 1983). They also observed that floret death occurred at the terminal ends of the ear. The death of florets during this period may be attributed to the induction of male sterility by water stress (Bingham, 1966; Saini and Aspinall, 1981), which affects meiosis in the gametes (Bingham 1967). Water stress during anthesis has been reported to affect fertilization (Wardlaw, 1974) thus reducing the potential grain number.

Individual grain weight has been reported to be affected by water stress during the period of grain growth, development and filling. Asana and Joseph (1964) reported that water stress which occurred before anthesis and extended to grain development increased the rate of endosperm cell division. Wardlaw (1971) suggested that this may be a response to compensate for a reduction in potential grain number, and thus grain weight may be increased. Prolonged water stress, however, reduced the amount of photosynthates produced, and the translocation of photosynthates from the leaves, thus limiting the amount of photosynthates available for grain filling (Wardlaw, 1967). This may explain the reduction in individual grain weight and final yield obtained by Day et al. (1978) from plants which were water stressed during the period of grain growth and development. Although the potential endosperm cell numbers are increased under water stress, the

potential grain weight may not be realised because of a reduction in photosynthate production and supply (Aspinall, 1965). Furthermore, this may result in shrivelled grains (Swain and Melville, 1973; Brooks et al., 1982).

The effect of water stress on cereal root systems has been ignored in most studies. Water deficits reduce dry matter production of plants and also change the partitioning of photosynthates among organs (Kramer, 1983). Root:shoot ratios are usually increased by water stress, probably because more water deficits develop in the transpiring shoots and persist longer than in roots. The absolute weight of the roots, however, may not be increased (Kramer, 1983). The reduction in root function associated with water deficits results in reduced cytokinin production (Itai and Vaadia, 1965, 1971). Reduced cytokinin levels during grain filling are an important factor in the premature senescence of grains (Wardlaw, 1971). Alteration in root functions owing to water deficits may, therefore, lead to yield reductions.

5 The growth and function of roots

5.1 Anatomy

During their growth and maturation, roots undergo anatomical changes which affect their permeability to water and solutes (Kramer, 1983). The root apex consists of the root cap, the meristematic region, the region of cell elongation and the region of differentiation and maturation (Esau, 1941). The root cap is composed of loosely arranged cells, and plays no role in absorption (Barlow, 1975). The meristematic region consists of thin walled cells almost completely filled with cytoplasm. The high resistance to movement of water and/or solutes through the cytoplasm reduces absorption through this region (Kramer, 1981). The movement of photosynthates into the mersitematic region is by diffusion and this may limit the growth in the apical region. Behind the root apex is the zone of rapid cell elongation and expansion, and here growth occurs by cell division (Byrne, 1974). The area where cells and tissues are differentiated merges with this zone, and differentiation often continues after root elongation ceases (Byrne,

1974).

The cells behind the zone of enlargement are differentiated into the epidermis, cortex and stele, which constitute the primary structures of the root (Percival, 1921; Byrne, 1974; Kramer, 1983). In a transverse section through a wheat root, there is an outer epidermis and a broad zone of cortex which is limited internally by a well-defined endodermis. Inside the endodermis is a layer of parenchymatous cells, forming the stele, among which the phloem and xylem are arranged radially. The outermost layer of the stele forms the pericycle (Percival, 1921).

Root hairs, which develop from the endodermis layer, are composed of cellulose, which allows the absorption of water and solutes by osmosis. There is a continued process of root hair production near the root apex, and death on the older portions of the root (Percival, 1921).

With the ageing of the root, the epidermis ceases its absorptive function, and the layer of cortical cells within the exodermis undergoes suberization of their walls, forming a protective layer. When the cortical cells die, the vascular cylinder is protected by the endodermis for a large part of the plant's life.

5.2 The absorption and transport of water and ions

The absorption of water and minerals occur through those regions of the root which offer the least resistance (Kramer, 1983). Little absorption takes place through the meristematic region because of the high resistance offered by the protoplasm, and the absence of xylem. The suberization of the hypodermis also reduces the absorption of water, thus absorption mainly occurs in a region behind the root tip where root hairs are present. Studies by Clarkson et al. (1975), however, suggest that absorption also occurs far behind the apex. Ions enter suberized roots primarily by diffusion and by mass flow, through lenticels and openings caused by the death of lateral roots (Kozlowski, 1978; Kramer, 1983).

The movement of water across the root (radial movement) to xylem vessels occurs by diffusion and mass flow (passive movement), and by active transport (Kramer, 1983). Movement occurs through different pathways, i.e., through the vacuoles, the cytoplasm (symplast), and the cell walls (apoplast). There is still a lot of uncertainty about whether movement of water across roots is through the vacuoles, symplast, or apoplast (Kramer, 1983), and more information is required regarding the conductivity of these three pathways. The apoplast is highly permeable and allows unrestrained movement of water and solutes. However, the free space does not extend beyond the endodermis because the Casparian strips are a barrier to ion diffusion. Kramer (1983) suggested that ions accumulate in the cytoplasm and move from cell to cell (symplastic movement) through the plasmodesmata, and that this pathway is considered the most important pathway for ion movement. The active transport of ions across cell membranes occurs against concentration gradients, and requires metabolic energy (Mengel, 1974; Kozlowski, 1978). The most common concept of active transport is the carrier theory (Epstein and Hagen, 1952). The energy for active transport is provided by respiration (Russell, 1977), and allows the movement of ions from a zone of lower to higher electrochemical potential. This movement of ions into the xylem increases their concentration in the xylem sap and creates an osmotic potential for water movement into the xylem.

The movement of water into the plant from the xylem occurs by a driving force created by transpiring leaves (Slatyer, 1967; Kramer, 1969; Kozlowski, 1972, 1978), and is dependent on water potential gradients. Therefore, the transfer of water from the soil through the plants to the atmosphere is considered in terms of potential gradients (Russell, 1977). These water potentials may be affected by resistances to flow of water across roots, through the plant, and to the atmosphere (Kramer, 1983). The resistances are mainly offered by stomata and the roots, and under normal conditions, may be influenced by the atmospheric demand and the soil water status (Kramer, 1983). However, soil and plant water stresses (Kramer, 1969), and diseases (Buchanan et al., 1981; Misaghi, 1982) which affect host physiology, may also affect the water potential of a plant.

5.3 The production and function of plant growth regulators

Other important functions of roots include anchorage of plants, conversion of inorganic nitrogen into organic nitrogen compounds (Obroucheva, 1975), and synthesis of growth regulators such as cytokinins (Itai and Vaadia, 1965), gibberellins (Phillips and Jones, 1964), auxins (Batra, et al., 1975), and abscisic acid (Lenton et al., 1968). Cytokinins were reported to be produced in the meristematic region of the roots (Feldman, 1975), and abscisic acid in the root caps (Elliott, 1977). Batra et al. (1975) observed that auxins were produced by germinating seeds and in the basal regions of roots. The rate of production of plant growth regulators and their concentrations in the plant may be influenced by the developmental stages of plant growth (Skene, 1975). The production of plant growth regulators by roots may be influenced directly or indirectly by environmental factors (Skene, 1975). Stress to the roots may also influence physiologic changes in the plant. Andreenko et al. (1964) reported that low pH around the rhizosphere affected the production of cytokinin by maize roots. The production of cytokinins may be reduced by water stress (Itai and Vaadia, 1965; Vaadia and Itai, 1969) and saline and osmotic stresses (Vaadia and Itai, 1969). Water-logging has been reported to reduce the production of gibberellins (Reid et al., 1969) and cytokinins (Burrows and Carr, 1969) by the roots. Reduction in the production of plant growth regulators may affect the normal functions of the plant, as they play an important role in the growth and differentiation of the various tissues and organs. For example, auxins and gibberellins stimulate cell expansion (Leopold and Kriedemann, 1975), while cytokinins promote growth by cell division (Skoog et al., 1967). Plant growth regulators are also involved in nutrient uptake and transport. These processes are stimulated by indole-acetic-acid (Davies and Wareing, 1965), and cytokinins (Ilan, 1971), and inhibited by abscisic acid (Collins and Kerrigan, 1973). Increased levels of absicisic acid found in water-stressed plants and water-logged plants stimulated the closure of stomata (Wright and Hiron, 1972). This conserved water in the plants during the water-stressed period, and counteracted the effects of reduced efficiency in water uptake during water-logging. Abscisic acid is reported to inhibit growth by reducing cell enlargement

(Leopold and Kriedemann, 1975). It also promotes senescence and ethylene production (Osborne, 1973). Ethylene also initiates abscision of leaves (Leopold and Kriedemann, 1975). It has been reported that plant growth regulators influence the formation of root primordia (Haissig, 1971) and the growth and differentiation of roots (Kozlowski, 1978). Although plant growth regulators individually influence specific plant processes, they also have interactive influences, which may be synergistic or antagonistic (Kozlowski, 1978). For example, Procházka (1981) reported that cytokinins and gibberellins promoted lateral bud growth of the shoots of pea seedlings (Pisum sativum L.) owing to the presence of indole-acetic-acid in the shoot apex.

5.4 Structure of the cereal root system

The roots of cereals arise from two sources. The first formed source is the seminal axis which appears from the coleorhiza, and the second formed source is the nodal axis, which appears from the coleoptile and stem nodes (Gregory et al., 1978b). The root initials in an embryo are comprised of a radicle and two pairs of lateral rootlets which originate from the hypocotyl (Percival, 1921). A sixth root is often produced later from the base of the plumule (Percival, 1921), as well as a third pair of roots which appear behind the second pair of laterals. A young cereal root system under favourable conditions may therefore contain three to eight roots (Percival, 1921; Weaver, 1926; Ellis, 1976; Gregory et al., 1978b). These constitute the primary or seminal root system (Percival, 1921; Weaver, 1926). The primary roots produce first order laterals which grow horizontally and they, in turn, produce second and third order laterals (May et al., 1967; Hackett, 1968).

The first pair of roots of the nodal root system arises from the nodal axis (Gregory et al., 1978b). This nodal root system is also referred to as the secondary or adventitious root system (Percival, 1921; Weaver, 1926; Ellis, 1976; Gregory et al., 1978b). The development of this root system coincides with the appearance of tillers (Percival, 1921), but this may vary with the time of sowing. In spring-sown wheat, the secondary root system develops one to two months

after sowing (Weaver, 1926), whereas in winter-sown wheat, the nodal axes are produced three to four months after sowing, and the roots develop slowly until late spring (Gregory et al., 1978b). As many as ten nodal root axes may be produced from nodes 1-5 and up to six root axes from nodes 6-7 (Gregory et al., 1978b). Many of these nodes are below the soil surface, except the seventh node which is above the soil surface because of internode elongation. Nodal roots may branch after elongation beyond 10 cm (Gregory et al., 1978b). The tillers develop their own nodal root system (Boatwright and Ferguson, 1967) and roots are produced on nodes 1-5, with one axis per node (Gregory et al., 1978b). The root system of each tiller normally meets the requirements of the shoot to which it belongs (Boatwright and Ferguson, 1967). Tiller roots, however, are mainly restricted to the top 40 cm (Gregory et al., 1978b). Therefore, under conditions of soil water stress, or when the moisture in the surface layers is depleted, tillers may depend on the main shoot which is able to extract moisture from greater depths, as it has a longer seminal root system (Gregory et al., 1978b). There may be no functional difference between the two different root systems of cereals, although they show distinct distribution patterns (Percival, 1921).

The volume of soil explored by root systems of field-grown cereal crops in a silt loam soil was studied by Weaver (1926), who reported vertical root growth to depths greater than 185 cm and horizontal growth to 60 cm. Kirby and Rackham (1971) observed barley roots at 200 cm depth. In a sandy soil, wheat roots were observed at 396 cm (Troughton, 1962), but, in contrast to Weaver (1926), horizontal growth did not exceed 30.5 cm. It is possible these differing observations are a consequence of soil type. Differences in the total amount of roots produced by a plant may vary depending on genetic and environmental conditions (Russell, 1977). In general, winter cereals develop a greater root system than spring cereals (Weaver, 1926; Troughton, 1962), which may be owing to the longer growth period. Evidence suggests that in spring wheats, a deeper root system may develop if the vegetative period is prolonged (Asana and Singh, 1967). Singh and Dastane (1970) reported that the depth to which roots penetrated may also depend on the genotype of wheat. However, Lupton et al. (1974) and Wilhelm et al. (1982) found no consistent differences

between the rooting depth of tall and dwarf wheats, and suggested that soil and climatic conditions influenced rooting depth and distribution more than the genotype of the plant.

Under field conditions, most of the cereal root system occurs in the upper 0-15 cm layers of the soil (Welbank and Williams, 1968; Kirby and Rackham, 1971). In a silt-sandy loam soil, Welbank and Williams (1968) observed that to a depth of 60 cm, 78%, 12%, and 10% by weight of roots was between 0-15 cm, 15-30 cm, and 30-60 cm respectively. Root growth of field-grown wheat has been reported to increase exponentially in dry weight up to five months after sowing, and then increase linearly until anthesis (Gregory et al., 1978b). Other workers have reported a reduction in root dry weight between anthesis and harvest for barley (Welbank and Williams, 1968). Root weight declined or remained constant after anthesis, but the growth of roots continued up to harvest (Gregory et al., 1978b). Aung (1974) suggested that during the vegetative phase of growth, shoot and root growth increase concurrently, but the shoot grows at a faster rate. With the onset of the reproductive phase, root growth is reduced, possibly because photosynthates are monopolised by the shoot and the developing grain (Aung, 1974). Thus the pattern of root development is influenced by the physiological stage of the shoot.

5.5 Factors affecting growth and development of the root system

The growth and development of the root system is dependent on the shoot system and its environment, and also on soil properties such as texture, water holding capacity and nutrient status (Evans and Wardlaw, 1976; Russell, 1977). During seed germination, the root initials depend on reserves in the endosperm for their growth (Percival, 1921). The extent of their development and the initial depth to which they grow is influenced by soil texture and sowing depth (Percival, 1921). When sown near the soil surface, seminal roots may reach a depth of 20-30 cm. If the grain is sown at greater depths, the seminal roots may grow poorly, as most of the endosperm reserves are then utilised to enable the shoot to reach light (Percival, 1921). The subsequent growth of the root system is dependent on the shoot for photosynthates (Hicks, 1928) and is in competition with the shoot for available metabolites

(Aung, 1974). At the seedling stage of growth, the partitioning of photosynthates to the roots is high relative to the shoots, and steadily declines throughout development (Mann, 1957; Bruinsma and Schuurman, 1966). The partitioning of photosynthates between roots and shoots may also be influenced by genetic factors. The development of high yielding cereal cultivars has been accompanied by a progressive decline in the weight of roots relative to the shoots (Evans and Dunstone, 1970), probably because greater amounts of photosynthates are invested in shoot dry matter which may contribute towards yield. Variations in the distribution of photosynthates between roots and shoots may also be caused by other factors such as plant density, light, nutrients and temperature (Russell, 1977). Kirby and Rackham (1971) reported that the dry weight of barley roots increased with increasing plant densities (from 50 to 800 plants m^{-2}), but in smaller increments as densities were increased beyond a certain number (from 200 to 800 plants m^{-2}). A greater proportion of this increase was observed in the 0-40 cm layer of the soil profile. An increase in light intensity generally results in an increased shoot:root ratio (Troughton, 1962). Conversely, decreases in light intensity reduced both shoot and root weight, but the roots were affected more than the shoots (Brouwer and de Wit, 1969). Competition between shoots and roots for photosynthates increased when the supply was restricted by low light intensity (Neales and Davies, 1966; Osman, 1971). Under these conditions, the shoots may have an advantage over the roots, as they are nearer to the source of the limited photosynthates (Brouwer and de Wit, 1969).

Root growth is greatly influenced by the soil environment. Some of the stresses roots experience in the soil include mechanical impedance to root penetration, an imbalanced nutrient status, unfavourable pH, unfavourable temperatures and inadequate water supplies (Russell, 1977). Root growth was reported to be restricted by increasing bulk density (Veihmeyer and Hendrickson, 1948). However, the wet bulk density of a soil may vary with water content, influencing the soil strength (Russell, 1977). Thus root penetration may be influenced to a greater extent by a change in soil strength rather than bulk density (Taylor and Gardner, 1963). This has been shown to be a controlling factor in the growth of maize roots (Barley, 1962, 1963). A restricted root system attributed to increased soil strength may reduce

the potential exploration of water and nutrients (Taylor, 1974). This may be detrimental to crop growth, especially if the roots are confined to the upper zones of the soil in which the supply of water and nutrients is limited (Ellis et al., 1977).

A favourable soil nutrient status has been shown to reduce the size of the root system relative to the shoot (Troughton 1962; Welbank and Williams, 1968), especially with respect to nitrogen and phosphorus. Hackett (1968) also found that an increased phosphorus supply reduced root:shoot ratio, but potassium increased the ratio. In contrast, a deficiency in phosphorus was shown to increase root growth (Marshall and Wardlaw, 1973).

Root growth is also affected by soil pH (Arnon and Johnson, 1942), and low pH's reduce root elongation (Burström, 1956). In general, a pH lower than 4 and greater than 8 has been shown to reduce root growth (Moore, 1974).

Variations in temperature may affect both root and shoot growth, although they experience different temperature regimes (Russell, 1977). Within the root system itself, differences in temperature may occur. Walker (1969) has shown that changes in soil temperature can induce significant effects on root growth. Root meristematic activity is most affected by unfavourable temperatures, and low temperatures can restrict branching of the root axis (Garwood, 1968).

The growth of roots may also be influenced by the water status of the soil. It has been shown that roots of dicotyledonous plants accumulate photosynthates under soil water deficits (Vartanian, 1981) and when soil water deficits are alleviated, the roots rapidly elongate and produce more root hairs (Vartanian, 1981). It has been suggested that soil water stress in cereals favours root growth relative to shoot growth (Evans and Wardlaw, 1976). The most important characteristic of the root system of crops is the ability of the root axes to extend sufficiently so that they maintain contact with soil zones where the water potential remains adequate under dry conditions (Weaver, 1926). During short periods of soil water deficits, cereals may induce development of new nodal roots which may contribute to the recovery of the plant after alleviation of water stress (Russell, 1977). It is often reported that variation in soil water status is the major cause of

differences in distribution of roots, particularly the depth to which they penetrate in the soil (Russell, 1977).

5.6 Relationship between root function and yield

The role of roots in yield determination is closely related to their effectiveness in nutrient and water uptake, but many other factors interact to control the distribution and function of nutrients in plants (Evans and Wardlaw, 1976). Roots also synthesize plant growth regulators (page 40), which are essential for shoot growth and function. For maximum attainable crop yields, an optimum root system is necessary. Russell (1977) suggested that an optimum rooting system is one in which "there is a minimum diversion of metabolites to the roots which is compatible with them providing adequate water, nutrients, and growth substances to shoots". This may be relevant when plants are grown in a uniform and favourable environment throughout crop growth. Under field conditions, however, this does not occur, and optimum rooting strategies depend on climate and soil, and an optimum root system should be able to adequately support the plant during periods of stress. For example, where crop growth is largely dependent on stored water, growth and penetration of roots throughout development may be beneficial in maintaining water uptake (Salim et al., 1965; Hurd, 1968). By the amputation of wheat roots in the field, Sallans (1942) reported that once roots are establised, they supply water and nutrients to the plants independently. The primary root is the most important individual root, and is as important as the first pair of laterals in its contribution to yield (Sallans, 1942; Passioura, 1972). The contributions of the nodal roots, while individually small, in combination were found to be greater than those of the primary root system. The increased production of roots by unrestricted tillering in the presence of adequate water may not make a significant contribution to a higher grain yield. These tillers may use more water, and not produce grain bearing ears (Jones and Kirby, 1977). Consequently, the restriction of tillering, which may lead to a reduced root system, could improve water use efficiency where water is limited. Passioura (1972) reported that a single primary root of wheat contributed to improved yield even when water was limiting.

The contribution of different roots towards water uptake in the soil profile may vary depending on the rooting pattern (Asana and Singh, 1967; Pearson, 1974). The climate, which determines atmospheric demand (Gregory et al., 1978a), and soil water potential (Russell, 1977), also influences water uptake. The soil zone where roots absorb water most readily is that where the water potential is highest (Russell, 1977; Unger et al., 1981). When the soil is close to field capacity, absorption normally occurs near the soil surface. Roots, however, can absorb water at similar rates from both near the soil surface and from lower regions of the soil profile, if root densities and soil water contents are similar (Pearson, 1974). Under normal field conditions, Long and French (1967) observed that the lowering of water potential in the top 30 cm occurred mainly from evapotranspiration, and from deeper layers by water extraction by roots. However, in a dry season when surface layers of soil are rapidly depleted of water, wheat roots at lower depths were reported to play an important role in water extraction, and, in some cases, contributed up to 20% of the transpired water (Gregory et al., 1978b). Long and French (1967) suggested that the quantity of water which roots absorb from different zones of the soil may be inferred from the manner in which soil water is depleted. They considered that movement of soil water reflected both water uptake by different roots from different depths, and its movement through the soil which is created by gradients in water potential. However, Gregory et al. (1978a) showed a poor correlation between the proportion of roots in a particular layer and the water extracted from that layer. This was attributed to the movement of water to the layer immediately above, which was induced by the lowering of water potential caused by root water extraction in that layer.

Although the amount of roots present in the soil may affect the amount of water which is made available to the plant, some plants conserve water or use it more efficiently than others (Todd et al., 1962). Therefore, it is difficult to correlate yields with total root length or total dry weight of roots (Hurd, 1968). Furthermore, reduced water use efficiency may not directly relate to dry matter production, nor to the extent of the root system. An extensive root system may avoid yield reductions when there is an inadequate supply of water, but may not in itself contribute to higher yields (Hurd, 1968). In a field

crop which depends entirely on stored water, an extensive root system developed early in the growth cycle may possibly lead to a shortage of water late in the season. In such a situation, it is difficult to establish what role root growth may play in yield determination. An extensive root system, however, may be able to exploit a greater volume of soil (Pearson, 1974), especially when soil water is replenished from time to time.

The atmospheric demand for water during hot and windy days is high, and therefore diurnal water stress may occur in both the root and shoot (Slatyer, 1967), even if a plant has an extensive root system. A reduced root system may compound the stress, as it cannot exploit a greater volume of soil for available water (Unger et al., 1981), which may lead to prolonged water stress. Water stress may have a detrimental effect on plant growth depending on the stage of crop development (Salter and Goode, 1967; Slatyer, 1973), and the duration of stress (Day et al., 1978) (see pages 34-37). It has been suggested that root growth is less affected by stress than shoot growth (Evans and Wardlaw, 1976; Unger et al., 1981). However, with increasing stress the rate of root extension is reported to progressively decrease, and the area of roots actively absorbing water is also reduced (Slatyer, 1973). Root synthesis of plant growth regulators which are essential for shoot growth (Vaadia and Itai, 1969) may be reduced under water stress situations (Itai and Vaadia, 1965; Livne and Vaadia, 1972), thus affecting shoot development (Vaadia and Itai, 1969). A reduced root system may, therefore, compound the effects of water stress on shoot growth.

5.7 The effects of foliar diseases on root growth and development

Foliar pathogens induce modifications in host metabolism. Some known responses are changes in respiration (Daly, 1976), temporary increases in photosynthetic rates (Yarwood, 1967; Martin and Hendrix, 1974; Aust et al., 1977), and qualitative and quantitative changes in primary metabolites (Samborski and Shaw, 1956; Daly et al., 1961; Lunderstädt, 1966; Misaghi, 1982). Changes in root morphology and physiology occur in response to foliar infection (Martin and Hendrix,

1974) but it is not known if these changes are caused by metabolites transported to the roots. Foliar infection causes reduced root growth, measured by dry weight (Last, 1962; Doodson et al., 1964a; Brooks, 1972; Gough and Merkle, 1977; Ayres and Zadoks, 1979), and length (Martin and Hendrix, 1974; Walters and Ayres, 1981). From studies of powdery mildew on barley seedlings grown in solution culture, Walters and Ayres (1981) reported that both root length and branching of main, seminal and nodal root axes were reduced. However, while root length may be altered by disease, the weight is not necessarily reduced (Martin and Hendrix, 1974).

Reduction in the root length of wheat caused by stripe rust (Martin and Hendrix, 1974) was attributed to reduced cell division in the root apex, and reduction in the endodermis and stele components (pericycle, xylem, phloem and pith parenchyma) (Martin and Hendrix, 1974). Although it is not certain what causes these reductions in cell division, Crapo and Ketellapper (1981), and Walters and Ayres (1981), associated this with a reduction in photosynthates. A reduction in root mitotic index was reported in response to a reduction in metabolites caused by disease (Martin and Hendrix, 1974). Doodson et al. (1964a) reported that the dry weight of the root system was correlated with the amount of photosynthates distributed to the roots, which was greatly reduced in diseased plants. However, the ratio of photosynthates partitioned to the roots and shoots was not affected by disease (Walters and Ayres, 1981). Therefore, the reduction in root growth may be related to the reduction in the amount of photosynthates available for root growth and development. It is not certain, however, whether a reduced root system would eventually determine the photosynthate requirement for its own growth and development, or whether a reduction in the amount of photosynthates determines the size of the root system. The involvement of plant growth regulators may also play an important role in photosynthate partitioning. Kende (1971) suggested that cytokinins are involved in the mobilisation of metabolites in healthy plants. However, the involvement of cytokinins in alterations to translocation in diseased plants has not been studied. Plant growth regulators are also involved in cell division and elongation of apical meristems (see pages 40-41). In response to disease, changes in plant growth regulatory activity occur (Bhambota and Kaul, 1966; Mertz, 1967,

1968; Dekhuijzen, 1976; Pegg, 1976a, 1976b, 1976c; Misaghi, 1982). It is postulated that the reduction in root cell division reported by Martin and Hendrix (1974) may be associated with changes in plant growth regulatory activity. However, in foliar-diseased plants, the involvement of plant growth regulators and their influence on root growth has not been studied.

The rate of root growth in wheat is greatest before the onset of the reproductive phase (Martin and Hendrix, 1974; Gregory et al., 1978b), and may be related to the partitioning of photosynthates to roots. At the seedling stages of growth, a greater proportion of photosynthates produced are partitioned to the root system, but this steadily declines throughout crop development (Mann, 1957; Bruinsma and Schuurman, 1966). As pathogens cause an overall reduction in photosynthesis (Montalbini and Buchanan, 1974; Magyarosy et al., 1976) and reduce the amount of photosynthates transported to the roots (Walters and Ayres, 1982), an infection occurring in the early stages (seedling stage to tillering) of crop growth may reduce the amount of photosynthate required for the establishment of an extensive root system. Infection occurring late in crop growth may affect the formation of new roots and possibly the function of the existing root system. The later the infection of the plant, however, the less the overall reduction of the root system (Hendrix and Lloyd, 1970; Martin and Hendrix, 1974).

The significance of reduced root growth caused by disease at early growth stages, and the subsequent effect on plant growth has not been investigated adequately. A reduced root system caused by foliar disease during early growth (seedling stage to tillering) may be of greater importance in later growth stages than the loss of photosynthetically active leaf tissue itself. In glasshouse studies on the effect of foliar disease on root growth, sufficient water is usually supplied to minimise soil and plant water deficits and to maintain maximum vigour of the host. In the field, however, sufficient water is often not present in the soil to avoid deficits. Therefore a reduced root system caused by disease may not be able to explore the soil for water as efficiently as the root system of a healthy plant, resulting in plant water deficits. This would be more important in seasons where prolonged water deficits occur, as the ability of those plants with a

smaller root system to explore the soil would be reduced compared to plants which have a well-developed root system.

The study of disease yield-loss relationships of cereals has often ignored the role played by roots. In order to quantify the losses caused by disease through an understanding of cereal physiology, it is important to consider the contribution made by roots, as they are an integral part of a plant, and their activity is interrelated with shoots. Such a study would provide a better understanding of causal yield loss relationships. If fundamental principles of crop physiology were adopted in such a study, it would provide a basis for comparison with various genetic and environmental backgrounds. A causal approach would provide the input required for mechanistic models which may help in formulating better disease management/control strategies.

6 Objectives of the research programme

The effects of foliar disease on root growth, yield, and yield components of wheat were studied under field conditions. Cereal yield physiology was used as the basis for understanding the effects of different disease epidemics, which were imposed by the use of several fungicide spray regimes. The effects of fungicide sprays in the absence of disease were studied to establish whether fungicides produced any stimulatory or inhibitory effects on plant growth. The hypothesis that a reduced root system (caused by early disease) would lead to water stress late in crop growth was also tested. Later determined yield components often do not compensate for a reduction in early determined yield components affected by disease. An investigation into possible reasons for this lack of compensation was based on the above hypothesis.

CHAPTER TWO

THE EFFECTS OF FUNGICIDE SPRAYS ON ROOT DEVELOPMENT, YIELD AND YIELD COMPONENTS OF WHEAT

1 Introduction

Many of the cereal cultivars grown today have high yield potentials. To exploit these potentials, and to achieve maximum yield, a high nutrient input is required. This has increased the use of nitrogen, resulting in greater foliage production, and leaf area index of cereal crops (Welbank and Taylor, 1973). Simultaneously, the incidence of diseases has increased (Jones and Clifford, 1983). The increased knowledge of insect pest and disease problems and the economic incentives of higher yields have resulted in an increased use of pesticides. In the past decade, fungicides have become a standard input for intensive cereal production throughout Europe (Jenkins and Lescar, 1980; Cook, 1981). Fungicides are commonly applied as scheduled prophylactic treatments, based on the disease risk associated with cultivar choice, previous cropping and general crop husbandry. For example, in 1979, a total of about 6.5 million hectares under cereal cultivation in eleven countries in Europe received at least one foliar fungicide application, with possibly a third of the area receiving two applications (Jenkins and Lescar, 1980). This included 50%, 27% and 29% of the areas under cereal production in the United Kingdom, France and Germany respectively. In France in 1980, this increased to 40% of the area under cereals (Lescar, 1981). The application of fungicides resulted in variable cereal grain yield responses (Lescar, 1981), depending on disease severity, efficacy of the fungicide and number of applications. Positive responses to yield by fungicide application were attributed to control of specific diseases. Jenkins *et al.* (1972) reported an increase in yield in sprayed plots when foliar disease severity was negligible. Griffiths and Scott (1977) suggested that disease control alone may not have accounted for the yield increase.

They hypothesised that fungicide application may have stimulated plant growth, and used the term 'phytotonic', meaning the stimulatory effect on plant growth independent of disease control, to describe the phenomenon.

Phytotonic effects were reported on coffee in East African countries (Gillett, 1942; Griffiths, 1971). Significant phytotonic effects have not been reported in field-grown crops, but Griffiths and Scott (1977) and Peat and Shipp (1981) reported a phytotonic effect on cereal plants under glasshouse conditions. These experiments cannot be equated with field experiments, however, as light, temperature, air and soil environment, plant density, rooting depth and number and type of phylloplane organisms are not similar to those in the field. As field-grown plants differ from those grown in the glasshouse; it is important to establish field experiments under disease free conditions to investigate whether similar phytotonic effects occur to those obtained in glasshouse experiments. Field experiments to test the efficacy of fungicides are normally conducted in high disease risk areas, reducing the possibility of a disease free crop. Griffiths and Scott (1977) concluded that the evidence for phytotonic responses to fungicides was inconclusive, but suggested three possible ways in which the effects may be produced. First, the fungicides may directly affect plant growth; second, control of even small amounts of recognised diseases may, at critical times, have large effects on yield; third, control of phylloplane organisms and weak pathogens may, in certain circumstances, lead to yield increases.

Direct effects of fungicides on plant growth have been reported for methyl benzimidazole carbamate (MBC) chemicals in cereals (Staskawicz et al., 1978). It is now established that benzimidazole fungicides have cytokinin-like properties (Dimond and Rich, 1977) which prevent senescence. Mukhopadhyay and Bandhopadhyay (1977) reported that carbendazim acted as a weak cytokinin. Staskawickz et al. (1978) confirmed the cytokinin-like activity of carbendazim and reported retention of chlorophyll by retarding the breakdown of chlorophyll in oat (Avena sativa L.) leaves. Peat and Shipp (1981) reported that benomyl produced a cytokinin-like effect on cereals. Benzimidazole fungicides, therefore, may have a direct biochemical effect on senescence, and this may be the mechanism by which senescence is delayed

by carbendazim plus zineb (Dickinson and Walpole, 1975), benomyl (Ellen and Spiertz, 1975), and zineb (Jenkins et al., 1972). The increase in green leaf area from delayed senescence, however, may only contribute to yield increases in a source limited situation as, for example, if the supply of photosynthates was limited at grain filling and was not sufficient to realise the grain filling potential of the crop.

Fungicide sprays of benomyl, carbendazim, ethirimol, tridemorph, triadimefon and captafol were reported to increase yield in the presence of negligible amounts of disease (Griffiths, 1981; Priestly, 1981). The yield increases in fungicide treated plants were related to a longer green leaf area duration before anthesis compared to untreated plants, which increased the amount of stored stem reserves (Griffiths, 1981). The importance of green leaf area before anthesis is emphasised by studies on light interception (Monteith, 1977), shading (Willey and Holliday, 1971), defoliation (Williams and Hayes, 1977) and photosynthate storage (Gallagher et al., 1975), indicating that reductions could reduce yield in proportion to the reduction in green leaf area. Disease epidemics early in crop growth were reported to reduce maximum leaf size (Lim, 1982; Gaunt and Thomson, 1985), emphasising the importance of reduced green leaf area contributing to yield reduction. Based on the developmental physiology of cereal yield, it was suggested that certain stages of growth may be very sensitive to disease (Lim, 1982; Gaunt and Thomson, 1985), and even small amounts of disease at the critical stages of plant development may reduce differentiation of apical meristems, thus reducing yield potential. Pathogens may also influence yield potential when they infect plants during critical stages of crop growth by interfering with host metabolism before symptom expression (i.e., during the incubation period). Infection by foliar pathogens early in the development of cereals may also induce changes in growth regulatory activity (Vizárová and Minarčic, 1974). Griffiths and Scott (1977) suggested that if these changes coincide with inflorescence differentiation, they could modify yield potential. Fungicides applied at these critical stages when disease severity is very low, therefore, may result in yield increases.

Dickinson (1981a) provided an alternative explanation to the cytokinin effect of fungicides on green leaf area. Cereals are colonised by a number of saprophytic fungi involved in tissue decomposition (Hudson, 1968), and their population was thought to increase only at the onset of senescence. Dickinson (1973) and Skidmore and Dickinson (1973), suggested that these phylloplane organisms may accelerate senescence, and thus the fungicidal control of saprophytic populations may influence senescence. Other evidence (Dickinson, 1981b), however, suggests that a major area of the leaf would senesce naturally before the saprophytes became active, thus the control of saprophytes by fungicides may not necessarily lead to retention of green leaf area by delaying senescence. Dickinson (1981a, 1981b) suggested that saprophytic fungi may also behave as weak pathogens, becoming more active in colonising host tissues as the crop senesces. These facultative parasites may be of greater importance than previously recognised, and their direct control by fungicides may increase green leaf area and possibly yield. Fungicide treatments may, therefore, both reduce the number of organisms and prolong the functional life of leaves. The above explanations of yield increases from fungicides by the control of facultative pathogens, however, cannot be considered to be phytotonic effects because they are not independent of disease.

If phytotonic effects occur in field-grown cereal crops, they will be acting within the balance of photosynthate production (source) and the number and size of yield site potentials (sink). Although Yoshida et al. (1972) suggested that an increase in either sink or source may lead to substantial yield increases, it should be understood that this can only be true when there is a correlative balance between the two systems. For example, if there is an increase in green leaf area (which contributes towards greater photosynthate production) from fungicide application, a yield increase will occur only if there is a greater yield site potential to utilise the additional photosynthates produced. An increase in photosynthate production beyond sink capacity could lead to correlative inhibition (Neales and Incoll, 1968), resulting in leaf senescence. The grain weight component of yield is closely related to green leaf area duration after anthesis (Thorne, 1974). Consequently, an increase in green leaf area before anthesis will not directly affect grain weight. Increased stem reserves laid down before anthesis may

contribute significantly to yield if constraints to photosynthesis are present during grain filling (see Chapter 1, Section 4.2.2). Individual grain weight may be dependent on endosperm cell number which is associated with photosynthate supply at anthesis (Brocklehurst, 1977, 1979). Brocklehurst (1979) suggested that plant growth regulators such as cytokinins may regulate the rate of cell division in the endosperm. As certain fungicides possess cytokinin-like effects, it is possible that they may affect endosperm cell division. Any increase in yield obtained by application of fungicides in the absence of disease, and which is attributed to increased grain weight, therefore, indicates the relief of a source limited system by phytotonic effects.

At present, there is no consistent evidence for phytotonic effects from field trials in cereals. There have been no studies on the effect of fungicide application on root growth. The shoot and root are dependent on each other for growth and development (Russell, 1977), and their activities are proportional to one another (Charles-Edwards, 1982). Therefore, for a better understanding of phytotonic effects, it is important to study both above and below ground components of plant growth. Such studies are reported in this chapter for field-grown wheat.

2 Materials and Methods

Wheat seed (cv. Kopara) treated with captan (100 g a.i. per 100 kg seed) was sown on 24th June 1980 on a Templeton silt loam soil at the research farm, Lincoln College. The seed was sown at 150 kg ha^{-1} with a row spacing of 150 mm using a Duncan seed drill (Duncan P and D Limited, Christchurch). Single superphosphate (8% P) was applied at a rate of 250 kg ha^{-1} at sowing. The land was previously occupied by field beans (*Vicia faba* L.).

On 20th August 1980, at G.S. 12 (Zadoks *et al.*, 1974) four randomised blocks of treatment plots (12.0 x 10.0 m) were marked out, with an access path (0.5 m) bordering each plot. The plots were separated by a 2.0 m buffer zone which was untreated to provide an adjacent source of inoculum. Each replicate block consisted of four

treatments - full, early, late and nil sprays designed to give nil, late, early and full disease epidemics respectively. The plant population was assessed in five randomly placed 0.1 m^2 quadrats for each plot at G.S. 12 (22 August 1980).

Herbicide (MCPA, 900 g a.i. ha^{-1} and dicamba, 100 g a.i. ha^{-1}) was applied to the entire trial by helicopter at G.S. 23 (20 September 1980). Benomyl (250 g a.i. ha^{-1}) was applied at G.S. 12, 23, 25, and 33 to control the development of speckled leaf blotch. Triadimefon (250 g a.i. ha^{-1}) was applied at G.S. 40 and 82 to control the development of brown rust (caused by Puccinia recondita Rob. ex Desm.) and speckled leaf blotch. Fungicide sprays were applied from each side of the plots by a tractor mounted 6.0 m boom with hollow cone nozzles (D2, 25) set 380 mm apart. The fluid to the boom was supplied from a rear mounted tank at the rate of 300 l ha^{-1} at 3.45 X 10^5 N m^{-2} pressure. The full and early spray treatments received three sprays of benomyl at early growth stages (Table 2.1), and a single spray of benomyl plus two sprays of triadimefon were applied at later growth stages to the full and late spray treatments.

Growth stage and disease severity were assessed ten times during the growth season (Table 2.1). On each occasion, ten plants were sampled randomly from rows at least one metre from the edge of the plots to avoid plants influenced by edge effects and interplot interference (James and Shih, 1973; James et al., 1973). Up to G.S. 33, plants from the full spray and early spray treatments, and from the late spray and nil spray treatments, were pooled for assessment. As disease severities were very low throughout the season, only full and nil spray treatments were sampled for disease assessment from G.S. 33 until harvest. Percentage leaf area occupied by disease symptoms was recorded on all green leaves of the main stem using standard area diagrams (Anon, 1972). The leaf lamina was removed and the area determined using an automated leaf area meter (Licor Model 3100, U.S.A.). When the maximum leaf area was attained for each leaf position, the area was no longer measured to avoid inaccuracies associated with senescence. Total green leaf area was calculated as described by Lim and Gaunt (1981).

Soil cores of 50 cm length were extracted at four growth stages (Table 2.1) from the full and nil spray treatments. Analysis was carried out only at G.S. 12 and 89, as disease was not present in significant amounts throughout crop growth. The cores were extracted from one metre inside the plots (to avoid edge effects) at diagonally opposed ends, using a modified post hole machine (Welbank and Williams, 1968) designed and constructed by the Department of Agricultural Engineering, Lincoln College. The equipment consisted of a coring tube inserted into a machine driven auger. Within the coring tube were two half sleeves (50 mm diameter) which collected the soil core. Once the desired depth was reached with the auger, the core of soil within the half sleeves was transferred intact to polyvinyl chloride tubes and transported to the laboratory for analysis. As root length in the early stages of growth was short and inter- and intra-plot variations were expected to be small, only two cores per plot were extracted in the first sampling, whereas three cores per plot were extracted in the later sampling. The soil cores were cut into 10 cm increments to a depth of 20 cm, and then at 15 cm increments to 50 cm depth. The segments of corresponding depths of each soil core from a plot were pooled for root extraction. Soil samples were washed and the roots separated from the soil (Böhm, 1979) using an automated washing system and were then stored in 5% formaldehyde at 5°C (Meyer and Göttsche, 1971). Floating organic debris was removed, and root length was measured by the grid intercept method (Marsh, 1971). Only live roots were measured, and were distinguished by their pale orange colour and intact stele (Gregory *et al.*, 1978b). The root lengths were calculated per unit area and per unit volume of soil. The total root length in the entire core is presented as L_A cm cm^{-2}, where L_A = length in cm per unit area of soil (cm^2). The root length in the individual zones of a core is presented as L_V cm cm^{-3}, where L_V = length in cm per unit volume of soil (cm^3).

At maturity, five quadrats (0.1 m^2) were sampled randomly on each side of the 10.0 x 1.5 m central strip. The total number of plants and the number of ear bearing and non-ear bearing tillers m^{-2} were determined. A central 10.0 x 3.0 m strip was harvested by header (Walter and Wintersteiger Universal Seed Master, Austria), and the yield was determined at 14% moisture. One thousand grains were randomly counted from the harvest of each plot by a numigral seed counter (39

Table 2.1: Time of plant and root samplings, and fungicide applications in 1980-1981 field trial on wheat cv. Kopara.

Date	Growth stage[1]	Assessment of plant development and disease severity	Fungicide applications[2]			Root sampling
			Full spray	Early spray	Late spray	
22/8	12	-	x[3]	x	-	-
26/8	12	x	-	-	-	-
28/8	13	-	-	-	-	x
1/9	22	x	-	-	-	-
8/9	23	x	-	-	-	-
11/9	23	-	x	x	-	-
15/9	24	x	-	-	-	-
22/9	25	x	-	-	-	-
25/9	25	-	x	x	-	-
29/9	32	x	-	-	-	x
20/10	33	x	-	-	-	-
23/10	33	-	x	-	x	-
4/11	40	-	x	-	x	-
24/11	69	x	-	-	-	-
2/12	71	x	-	-	-	-
16/12	79	x	-	-	-	-
19/12	82	-	x	-	x	-
4/1	89	-	-	-	-	x

(1) Decimal growth stage (Zadoks, Chang and Konsak, 1974).

(2) Benomyl (250g a.i. ha^{-1}) was applied at growth stages 12, 23, 25, 33, and triadimefon (250g a.i. ha^{-1}) was applied at growth stages 40 and 82.

(3) x denotes time of sampling or fungicide application.

R.J.J. Rousseau 75001, Paris), dried at 80°C for 24 hours, and the moisture content and individual grain weight determined.

3 Results

3.1 The effect of fungicide application on disease epidemic

Symptoms of speckled leaf blotch first appeared at G.S. 24 (Table 2.2) as scattered chlorotic areas on the lower leaves. Pycnidia did not develop from the primary infection, and therefore secondary infection did not occur. No disease was present at G.S. 33, and speckled leaf blotch was not present at subsequent growth stages. Signs of brown rust appeared at G.S. 64, and reached low severity by G.S. 79. Fungicide sprays applied throughout the season reduced disease severity, but there were no significant differences between treatments up to G.S. 71. At G.S. 79, there was a greater amount of disease in the unsprayed plants compared to the sprayed plants. After G.S. 79 until harvest, there were no signs or symptoms of disease, thus no disease analysis was done.

3.2 The effect of fungicide application on total green leaf area

The mean percentage green leaf area (Table 2.2) was influenced by senescence from G.S. 23 onwards, but there were no significant differences between treatments. Maximum leaf area was reached after all leaves attained maximum size, between G.S. 33 and G.S. 69 (Table 2.2). There were no significant differences in total leaf area attributable to disease or fungicide sprays. Disease also had no effect on leaf dry weight (Table 2.2).

Total green leaf area, calculated as a weighted mean from the percentage disease severity and total leaf area measurements, was not affected by treatments because of low disease severities and similar total leaf areas on all plants. Variance was low at all growth stages except G.S. 71 (Table 2.2).

Table 2.2: The effect of fungicide application on disease development, leaf area, and leaf dry weight on main stems of wheat cv. Kopara in 1980-1981 field trial.

Variable	Fungicide application[1]	Growth Stage[2]									
		12	22	23	24	25	32	33	69	71	79
Mean % disease severity[3]	Full	0.00	0.00	0.00	0.02	0.03	0.06	0.00	0.00	0.02	1.26
	Nil	0.00	0.00	0.00	0.02	0.07	0.23	0.00	0.10	0.09	2.26
F test P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	NS	S
Mean % green leaf area[4]	Full	100.0	100.0	93.8	98.9	98.9	88.8	79.1	78.8	77.6	60.1
	Nil	100.0	100.0	90.6	98.9	98.5	90.0	78.9	76.6	76.5	59.7
F test P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Total leaf area (cm^2)[5]	Full	1.5	4.4	5.5	10.0	15.0	20.6	49.4	43.6	56.9	51.5
	Nil	1.4	4.0	5.1	9.5	14.3	21.4	47.1	40.9	51.2	46.5
F test P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Total green leaf area (cm^2)[6]	Full	1.5	4.4	5.9	10.6	15.9	20.8	46.6	37.2	46.7	30.0
	Nil	1.4	4.0	5.3	10.3	15.0	21.1	45.0	36.4	41.4	27.3
F test P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Leaf dry weight (mg)[7]	Full	26	45	60	87	116	150	308	339	353	290
	Nil	23	44	57	88	106	152	300	312	326	268
F test P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	NS	NS

(1) Benomyl (250g a.i. ha^{-1}) was applied at the following growth stages : 12, 23, 25, 33. Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 40, 82.

(2) Decimal growth stage (Zadoks et al., 1974).

(3) Simple arithmetic mean of percentage disease severity on all leaves present on the main stem.

(4) Simple arithmetic mean of percentage green leaf area on all leaves present on the main stem.

(5) Total leaf area of all leaves present on the main stem.

(6) Total green leaf area adjusted for maximum leaf size on the main stem (see Chapter 2, Section 2).

(7) Total leaf dry weight of all leaves present on the main stem.

3.3 The effect of fungicide application on root development

Root length, measured before fungicide application, was variable but no significant differences between treatments were observed in all zones except the 0-10 cm zone (Table 2.3). In this zone, there was less root length in those plants designated to be sprayed. When root length was measured at G.S. 89 after six fungicide applications, there were no significant differences between treatments except in the 20-35 cm zone (Table 2.3). In this zone, the root length in sprayed plants was 12% less than the unsprayed plants.

3.4 The effects of fungicide application on yield and yield components

Yields for the mechanically harvested plants, measured in tonnes per hectare, were low relative to yields in trials in other seasons, but were high compared to mean commercial practice. There were no significant differences attributable to spray treatments, where yield was only 1.4% greater than the unsprayed treatments (Table 2.4). Similarly, there was no effect on individual grain weight, measured on the mechanically harvested samples, nor on the number of plants m^{-2} measured from quadrat samples. Because of the lack of significant yield differences, the number of grains per ear was not counted. When the grain number per ear was calculated from grain dry weight, ear number m^{-2}, and individual grain weight, there were no significant differences between treatments.

4 Discussion

Disease severity in the sprayed and unsprayed plants was mostly not significant and very low. Severity reached a maximum of 2.3% and 1.3% at G.S. 79 in the unsprayed and sprayed plants respectively, and did not exceed 0.23% and 0.01% at other assessments. Disease severities in other wheat trials (Gaunt et al., 1985) and in commercial crops in Canterbury were also low in the same season,

Table 2.3: The effect of fungicide application on root length at growth stages 21 and 89 of wheat cv. Kopara in 1980-1981 field trial.

Growth stage[1]	Fungicide applications[2]	Soil profile Zone (cm) 0-10	10-20	20-35	35-50	0-50
21						
	Full spray	0.69[3]	0.67[3]	0.29[3]	0.20[3]	21.0[4]
	Nil spray	0.96	0.83	0.36	0.18	26.0
	F test $P \leqslant 0.05$	S	NS	NS	NS	NS
89						
	Full spray	7.69	4.30	1.55	0.94	157.3
	Nil spray	8.64	4.89	1.78	1.17	179.6
	F test $P \leqslant 0.05$	NS	NS	S	NS	NS

(1) Decimal growth stage (Zadoks et al., 1974).

(2) Benomyl (250g a.i. ha^{-1}) was applied at the following growth stages : 12, 23, 25, 33.

Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 40, 82.

(3) Root length per unit volume (L_V) of soil ($cm\ cm^{-3}$).

(4) Root length per unit area (L_A) of soil ($cm\ cm^{-2}$).

Table 2.4: The effects of fungicide application on yield and yield components of wheat cv. Kopara in 1980-1981 field trial.

Variable	Fungicide applications				F test $P<0.05$
	Full[1]	Early[2]	Late[3]	Nil	
Plant number m^{-2}	275	285	260	270	NS
Ear number $plant^{-1}$	2.40	2.40	2.50	2.48	NS
Grain number ear^{-1} (calculated)	31.4	30.2	30.4	29.8	NS
Individual grain weight (mg)	40.8	40.8	40.8	40.8	NS
Header yield ($t\ ha^{-1}$) at 14% moisture content	5.73	5.73	5.62	5.65	NS
Percentage increase in yield relative to nil spray treatment	1.42	1.27	-0.58	-	

(1) Benomyl (250g a.i. ha^{-1}) was applied at the following growth stages : 12, 23, 25, 33.

Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 40, 82.

(2) Benomyl (250g a.i. ha^{-1}) was applied at the following growth stages : 12, 23, 25.

(3) Benomyl (250g a.i. ha^{-1}) was applied at growth stage 33, and triadimefon (250g a.i. ha^{-1}) was applied at growth stages 40 and 82.

possibly because prevailing environmental conditions did not favour the development of speckled leaf blotch. By comparison, a severe disease epidemic in 1979/1980 reached a maximum severity of 21% in unsprayed plants, causing an 18% yield reduction (Gaunt and Thomson, 1985).

During the season, fungicide applications had no effect on either green leaf area or leaf area, contrary to Griffiths and Scott (1977), Cook (1981), Dickinson (1981a), Griffiths (1981), Jordan (1981) and Priestly (1981), who all found that fungicide applications did produce phytotonic effects.

The growth of shoots and roots are interdependent, and therefore any effects on shoot growth may also affect roots. Compared with shoots, roots are poor competitors for photosynthates (Wardlaw, 1967; Rawson and Hofstra, 1969), and less photosynthates may be partitioned to roots when plants are constrained, for example, by disease (Doodson et al., 1964b) or low temperature (Brouwer, 1966; Welbank, 1972). However, in the absence of disease constraints, there may be an increase in the amounts of photosynthates produced, and thus root growth may increase by greater partitioning of photosynthates to the roots, when compared to a diseased plant where partitioning is less. In this experiment, root growth was not affected by foliar fungicides applied throughout the season.

Fungicides themselves have been suggested to influence early crop growth. Early determined yield components, such as the number of tillers per plant, spikelet number per ear and potential grain number per ear, may be influenced by fungicides (Griffiths and Scott, 1977; Peat and Shipp, 1981). In this trial, the growth of existing tillers was not affected by fungicides, contrary to the studies of Peat and Shipp (1981). An increase in tiller number can lead to yield increases only when those tillers produce grain bearing ears. In Peat and Shipp's (1981) trial, the tillers initiated by fungicide application produced ears only when plant densities were below the optimum. Although an increase in the number of tillers was observed in those plants which received an application of benomyl, the concentration of benomyl applied was two and one half, five and ten times that usually applied in the field. A response to benomyl may not be obtained in the field at the recommended rate of application. In this trial, tiller bud initiation

was not measured; therefore, it is not certain whether benomyl did have any effect on this process. Fungicides had no effect on individual grain weight, in contrast to the results of Jenkins et al. (1972) and Griffiths and Scott (1977). There was no positive yield response to fungicide application during the season. The absence of significant effects on the number of plants per unit area, number of grains per ear (calculated), the individual grain weight, or yield suggests that the other yield components (spikelet number per ear and grains per spikelet) were also not affected. It is concluded that the absence of yield response to fungicide applications in the absence of disease indicated that fungicides had no phytotonic effect on crop growth throughout the crop cycle.

The results obtained for this trial apply to a given set of conditions of crop species (wheat), cultivar (Kopara), soil (Templeton silt loam), climate (temperate) and agricultural practices. These conditions differ from those under which similar trials (e.g., Jenkins et al., 1972) were reported. Although the studies of Jenkins et al., (1972) were conducted in the field, they did not conduct regular and detailed analysis of disease assessment in the early growth stages of the crop. Before the 1970's, disease assessment was mainly confined to the post ear emergence period. This may be because yield measurements are related to grain weight, which is partly influenced by effective green leaf area and photosynthetic rates after ear emergence. However, the validity of single leaf assessment at later growth stages has been criticised (Lim, 1982). Percentage disease severity on a single leaf does not necessarily indicate the amount of plant infection (Gaunt, 1978). As many of the grain yield components are determined in early growth stages (Kirby, 1974), any constraints to plant growth, for example, disease, would result in a reduced final yield (Lim, 1982; Gaunt and Thomson, 1985; Lim and Gaunt, 1985). Leaf area index is often used as one of the variables for assessing crop growth (Evans and Wardlaw, 1976). Other plant parts containing chlorophyll, however, are also capable of photosynthesising and contributing towards growth, for example, stem, leaf sheath, young caryopsis and awns. For a more meaningful estimation of disease on plant growth, therefore, it is important to consider the total green area of a plant, as this would give a better estimate of the effect of disease on the ability of the

plant to grow and develop (Lim, 1982; Lim and Gaunt, 1985). This method of total green area measurement may also be applied to fungicide trials in the absence of disease, to relate to the green area of the crop, and its contribution towards final yield.

Under the agronomic and climatic conditions in which this trial was conducted, four applications of benomyl and two applications of triadimefon had no effect on green leaf area, tiller number, individual grain weight and yield of wheat. It was concluded, therefore, that benomyl and triadimefon had no phytotonic effects. Thus, in the trials discussed in Chapters 3 and 4, it is assumed that yield responses from fungicide applications in the presence of disease were attributable to the control of disease alone, and not to any possible phytotonic effects.

CHAPTER THREE

THE EFFECTS OF SPECKLED LEAF BLOTCH ON ROOT DEVELOPMENT, YIELD AND YIELD COMPONENTS OF WHEAT

1 Introduction

Recent changes in agronomic practices have led to an increased disease risk in wheat crops. For example, the undersowing of wheat with pasture species leads to increased amounts of stubble remaining over the winter months (Sanderson, 1978), thus providing sites for the overwintering of pathogens. The introduction of dwarf and semi-dwarf high yielding cultivars (Scott, 1973; Nelson et al., 1974; Tavella, 1978) provides a more suitable microclimate for the development of diseases (Scott and Benedickz, 1977, 1978, 1979), and continuous cropping of wheat also enables the pathogen to perpetuate between successive crops (Harrower, 1974; Brokenshire, 1975; Holmes and Colhoun, 1975).

In the past fifteen years, speckled leaf blotch, caused by Mycosphaerella graminicola (Fuckel) Schroeter (imperfect state Septoria tritici Rob. ex Desm.), and glume blotch, caused by Leptosphaerella nodorum Muller (imperfect state Septoria nodorum Berk.), either singly or together, have been recognised as diseases of major importance in all wheat growing areas of the world. Reviews of Septoria diseases of wheat, including speckled leaf blotch, were published by Shipton et al. (1971), and more recently by King et al. (1983). The first reports of yield losses caused by speckled leaf blotch were from Italy (Cavara, 1893) in the 1890's. The pathogen was reported in North, Central and South America, Britain and Europe, India , Australia and New Zealand during the first half of the 20th century. The pathogen causing speckled leaf blotch has now been identified in over fifty countries (Anon, 1970). Severe outbreaks of speckled leaf blotch have occurred in both high and low rainfall areas, for example, Brazil (Mehta et al., 1979), Britain (Williams and Jones, 1972), Romania, Israel (Eyal, 1981),

Australia (Brown and Paddick, 1980), and New Zealand (Hampton, 1975).

Severe epidemics of speckled leaf blotch may cause 30-50% yield losses (Caldwell and Narvez, 1960; Eyal and Ziv, 1974; Mehta, 1976). Yield losses from the disease have been reported in Britain (Cooke and Jones, 1971; Jones and Odebunmi, 1971; Williams and Jones, 1972; King, 1973, 1977), Israel (Eyal and Ziv, 1974; Ziv and Eyal, 1978), Australia (Shipton, 1968; Brown et al., 1978; Brown and Paddick, 1980) and New Zealand (Wenham, 1959; Hampton and Close, 1976; Sanderson, 1976b; Chan and Gaunt, 1982).

Speckled leaf blotch was first reported in New Zealand by Cunningham (1927). Before 1947, the disease was considered to be of minor importance. In view of the increasing economic importance of the disease in other parts of the world, Wenham (1959) studied the disease under New Zealand conditions. In Canterbury, one of the major wheat growing areas of New Zealand, the disease was recorded in 43 out of 56 wheat crops surveyed in 1948 (Anon, 1953). Speckled leaf blotch has since been prevalent each year, particularly during periods of prolonged cool, wet weather. Most wheat crops in Canterbury are sown from April to June, and harvested by March. Therefore, the fungus must perpetuate for three to four months in the absence of actively growing crops. Pycnidiospores of the fungus have been reported to be viable for at least five months in pycnidia on wheat straw left in the field (Wenham, 1959). Infected volunteer plants and viable spores in pycnidia on decaying leaves and stubble were thought to be the only source of primary inoculum. In 1972, however, Sanderson (1972) discovered the perfect state of Septoria tritici, and suggested that initial infection was from ascospores. The presence of ascospores has also been recorded in Australia (Brown, 1975). Sanderson (1976a) and Sanderson and Hampton (1978) reported that ascospores from pseudothecia formed on standing stubble were the primary source of inoculum for autumn sown wheat in the Canterbury region. Symptoms of the disease were not evident until July-August, two to three months after sowing, although inoculum is present from the time of crop emergence (Sanderson and Hampton, 1978). The first expression of disease symptoms is chlorosis, especially at the leaf tip, which may gradually spread down the leaf. Malcolm (1978) suggested that this chlorotic effect may be because of a phytotoxin produced by the pathogen. Pycnidia are produced later within discrete

water-soaked lesions (Wiese, 1977). The lesions coalesce, and later become necrotic. The disease is assumed to spread to newly formed leaves and to other plants within the crop by splash dispersal of pycnidiospores. There is an apparent decline in disease severity in spring (Gaunt et al., 1985), when a phase of rapid plant growth occurs before the newly formed leaves become infected and symptoms are expressed (Sanderson, 1978). At this stage, the disease is confined to the lower, partly senescent leaves. During early summer, there may be an increase in disease severity. This second epidemic is dependent on conducive environmental conditions, as the pathogen requires 5-25^{o}C for both pycnidiospore germination (Gheorghies, 1974) and mycelial growth (Gheorghies, 1979), and a long period (20 hours) of high humidity for infection (Holmes and Colhoun, 1970). Therefore, a second epidemic may not occur in dry summers because of a lack of infection (Sanderson, 1978).

Canterbury accounts for 53.6% of the total area sown with wheat in New Zealand (Logan, 1983). The yield of wheat in New Zealand varies from 2.0 t ha^{-1} to almost 10.0 t ha^{-1} (Langer, 1979). In 1980 the national average yield was 3.56 t ha^{-1}, and Canterbury, in comparison to other wheat growing provinces, recorded the lowest average yield, of 3.08 t ha^{-1}. The average wheat yield in Canterbury could be increased when grown with good management on heavy soils and/or with irrigation. Constraints such as disease (Hampton and Close, 1976; Sanderson, 1978), and soil moisture (Scott et al., 1977) often limit yield. Speckled leaf blotch has been reported to cause yield reductions of up to 40% (Sanderson, 1978).

The amount of yield reduction caused by disease depends on the growth stage at which initial infection occurs (Williams and Jones, 1972; Jones and Rowling, 1976). In a full disease epidemic of speckled leaf blotch beginning at G.S. 22, Williams and Jones (1972) reported a yield reduction of 24% in cultivar Opal, attributable to a combination of lower grain number and individual grain weight. This suggests that the presence of disease during early crop growth (from G.S. 22 to G.S. 33) constrained yield by reducing the potential grain number. Disease in the later stages of crop growth (from G.S. 59 to harvest) constrained yield by reducing the source capacity, for example, a reduction in the amount of stored and current photosynthate at grain

filling required to realise the grain weight potential. As leaf and tiller primordia are formed early in the crop growth cycle, disease could only reduce the number of leaves and tillers produced if infection occurred early (between seed germination and G.S. 12). Under Canterbury growing conditions, leaf and tiller primordia are produced before disease symptom expression, therefore, the number of these vegetative structures produced may not be affected.

A disease epidemic between G.S. 22 to G.S. 33 (early disease) was reported to reduce yield only by reducing the grain number per ear. This was attributed to a lower number of spikelets and grains per spikelet produced (Gaunt and Thomson, 1985), which was associated with reduced apical development. Speckled leaf blotch did not affect the duration of spikelet primordium production, but development was reduced, resulting in a lower number of spikelets per ear. The mean number of floret primordia per ear was also reduced by speckled leaf blotch, and was attributed to both a reduced number of floret primordium per spikelet in all spikelet positions, and a lower number of spikelets per ear (Gaunt and Thomson, 1985). The reduction in grain number by early disease, however, was partly compensated for by an increase in individual grain weight during the later growth stages in the absence of disease. This increase suggests that there was a limitation to the potential number of grains (sink limitation), thus more photosynthates were available for grain filling. A late disease epidemic (from G.S. 33 to G.S. 75) was reported to reduce yield by reducing individual grain weight only, which suggests a source limited system during grain filling.

Under suitable environmental conditions, speckled leaf blotch symptoms can be present in the crop as early as G.S. 14, approximately eight weeks after sowing (Sanderson and Hampton, 1978). This enables the build-up of inoculum to provide an early disease epidemic. With the rapid growth phase of the plant and the production and expansion of new leaves in spring, however, there is an apparent decline in disease severity (Gaunt et al., 1985). The disease at this stage is confined to the lower leaves, but it eventually increases in severity as it spreads to the upper leaves. Sanderson (1978) distinguished these two periods of high disease severity as two epidemics. By applying prophylactic fungicidal sprays during the apparent period of disease decline, the

disease may be controlled on the upper leaves before infection takes place, therefore creating a situation where the crop is free of disease in the later stages of growth. Similarly, by controlling the disease only in the early stages, a disease free crop during this period may be created.

Reduced root development in wheat caused by foliar pathogens has been documented for rusts (Johnston and Miller, 1934; Bever, 1937; Doodson et al., 1964a; Martin and Hendrix, 1967; Hendrix and Lloyd, 1970) and speckled leaf blotch (Gough and Merkle, 1977). Gough and Merkle (1977) studied the effect of speckled leaf blotch infection on root growth of wheat under glasshouse conditions, and reported that the disease caused a reduction in root dry weight. The extent of root reduction depends on the growth stage at which infection initially occurs, and on the duration of the disease (Martin and Hendrix, 1974). Almost all studies of disease effects on root growth have been conducted on pot-grown plants in glasshouses, growth cabinets, or mist culture. The results from these studies cannot be applied directly to field conditions, as field-grown plants differ from those grown in glasshouses.

The objective of the following trial was to study the effect of foliar disease on root growth in the field under four different disease epidemics, i.e., full, early, late, and nil disease.

2 Materials and Methods

On 15th May 1979, a crop of wheat (cv. Kopara) was sown on a Templeton silt loam soil in a commercial cropping area on the research farm, Lincoln College. Plots (12.5 x 6.0 m) were marked out surrounded by a 0.5 m access path. There were four replicate blocks in a randomised block design, each block being separated by a 10.0 m untreated buffer zone, and containing the following treatment plots:

1. Nil disease (full spray)
2. Late disease (early spray)
3. Early disease (late spray)
4. Full disease (nil spray)

The treatments were created by different spray regimes as indicated above. In Chapters 3 and 4, treatments shall be referred to as the disease treatments.

A mixture of benomyl and mancozeb (250 and 1600 g a.i. ha^{-1}) was used to control the development of speckled leaf blotch. Fungicide sprays were applied by a hand carried 3.0 m counter balanced boom from each side of the plots to give full coverage. The boom was equipped with 7 hollow cone nozzles (D2, 25) and supplied with fluid from a CO_2 pressurised reservoir, carried on the back, which delivered 300 l ha^{-1} at 3.45×10^5 N m^{-2} pressure. Disease was controlled throughout the growth season by the application of nine sprays at the following growth stages; 22, 23, 24, 25, 33, 41, 57, 61 and 75. Two treatments received sprays before or after G.S. 33, to control early and late phases of the disease epidemic respectively. The fungicides were selected for their efficacy (Thomson et al., 1981) and minimal phytotonic effects based on previous tests in controlled environment facilities (Gaunt, unpublished).

At G.S. 22 (8th August) when lesions were first observed in the untreated plots, twenty-five plants were sampled randomly at weekly intervals from rows six and nine on each side of the plots (each plot containing a total of 60 rows) to avoid influence by edge effects and interplot interference (James and Shih, 1973; James et al., 1973). A 3.0 m strip was left undisturbed for final harvest analysis. Ten plants per treatment were subsampled and analysed for disease severity, green leaf area, leaf area and total green leaf area (see Chapter 2, Section 2).

Soil cores were extracted, as described in Chapter 2, Section 2, at G.S. 61 (26 November) and at G.S. 91 (11 January). In the first sampling, only the nil disease and full disease treatments were sampled. Two soil cores per plot were extracted, one within and the other between rows of the plants. Each soil core (65 cm long) was washed, the roots extracted, and root length measured, as described in Chapter 2, Section 2. As root length in cores extracted within and between rows did not differ, subsequent cores were extracted between rows. In the second sampling, all treatments (nil, late, early and full disease) were sampled (three cores per plot), similar zones pooled, and root length

per zone determined (see Chapter 2, Section 2).

At maturity, whole plants in ten 0.1 m^2 quadrats were removed from the central 1.5 m strip of each plot. The total numbers of plants and tillers with grain bearing ears were determined on each sample. From a 25 plant subsample, detailed measurements of yield components (spikelet number per ear, grain number per spikelet and individual grain weight) were made. The central strip was machine harvested, weighed, and the yield determined at 14% moisture.

3 Results

3.1 Development of the disease epidemic

The first symptoms of speckled leaf blotch were observed at G.S. 22, 75 days after emergence, as light green leaf mottling followed by senescence and the development of pycnidia. On the full disease plants, disease was present at moderate to high severities from G.S. 24 (24 September) to G.S. 31 (1 October), and was maintained at moderate severities until G.S. 71 (5 December), when disease increased up to maturity. On the nil disease plants, natural senescence (i.e., not disease induced) was usually first observed one week after the appearance of symptoms on the disease plants. Total death of leaves on the nil disease plants usually occurred one week later compared to corresponding leaves on the disease plants.

3.2 The effect of disease on total green leaf area

Total green leaf area is a better measure of the effect of disease on plant growth potential than percent disease severity (Lim and Gaunt, 1981; Gaunt et al., 1985). The green leaf area reached a maximum in all treatments from G.S. 37 to G.S. 43, between late October and early November (Table 3.1). From G.S. 24 (4 September) to maturity, the full disease plants had consistently less total green leaf area than the nil disease plants. This reduction was attributable to a combination of

Table 3.1: The effect of four different disease treatments on total green leaf area on main stems of wheat cv. Kopara in 1979-1980 field trial.

Sampling date	Growth stage[1]	Disease treatments			
		Nil disease[2]	Late disease[3]	Early disease[4]	Full disease
8/8	22	13.53[5]	-	-	11.87
15/8		14.10	-	-	13.16
21/8	23	16.75	-	-	16.23
28/8		21.57	-	-	20.42
4/9	24	24.51	-	-	21.55
11/9		32.62	-	-	30.79
18/9		44.64	-	-	35.61
24/9	31	46.03	-	-	38.23
1/10		69.84	-	-	47.20
8/10	33	85.94	-	-	73.91
15/10		89.87	-	-	81.33
22/10	34	99.73	-	-	84.78
29/10	37	92.20	83.25	67.46	71.10
7/11	43	100.66	76.78	81.67	81.42
13/11		91.28	79.53	93.25	70.34
19/11		89.09	85.36	80.29	77.56
26/11	61	86.08	84.54	85.95	63.73
5/12	71	70.52	66.55	64.56	56.28
12/12		60.04	55.91	67.47	38.19
18/12	81	40.27	31.41	32.44	10.26
24/12		11.05	6.39	8.92	0.00
31/12	90	0.00	0.00	0.00	0.00

(1) Decimal growth scale (Zadoks et al., 1974).

(2) Benomyl (250g a.i. ha^{-1}) and mancozeb (1600g a.i. ha^{-1}) were applied at the following growth stages : 22, 23, 24, 25, 33, 41, 51, 61, 75.

(3) Benomyl (250g a.i. ha^{-1}) and mancozeb (1600g a.i. ha^{-1}) were applied at the following growth stages : 22, 23, 24, 25, 33. Plot samples were combined with the nil disease treatment until G.S. 34.

(4) Benomyl (250g a.i. ha^{-1}) and mancozeb (1600g a.i. ha^{-1}) were applied at the following growth stages : 41, 51, 61, 75. Plot samples were combined with the full disease treatment until G.S. 34.

(5) Total green leaf area (cm^2).

lower percent green leaf area and total leaf area (Gaunt and Thomson, 1985; Gaunt et al., 1985). Disease increased in the late disease plants (i.e., discontinuation of fungicide from G.S. 33) but was not sustained, and from G.S. 43 (mid November) onwards, the total green leaf area was similar to the nil disease plants. In those plants where disease was controlled from G.S. 33 onwards (early disease), the total green leaf area per shoot increased markedly relative to the full disease plants, and from G.S. 43, was the same as for the nil disease plants. The increase was attributed to reduced disease severity and, therefore, increased green leaf area.

3.3 The effect of disease on root development

Root lengths from cores obtained at G.S. 61 within and between rows were similar, and there was no difference attributable to disease (Table 3.2). Detailed analysis of soil zones from cores obtained at G.S. 91 showed that root length was consistently greater in the 0-10 cm and 10-20 cm zones compared to those below 20 cm (Table 3.3). Root lengths of the nil disease plants were significantly greater in the entire core (0-50 cm) compared to the full disease plants (Table 3.3). This was attributed to greater root length in all the zones. Total root length, and root length per unit volume of soil of the nil disease plants were similar to those of the early and late disease plants. Total root length in the late disease plants was significantly greater compared to the full disease plants, attributable to a greater root length in the 0-10 cm and 10-20 cm zones (Table 3.3). Plants exposed to the early disease epidemic had significantly greater total root length than the full disease plants. However, root length per unit volume in the various zones of the early disease plants was similar to that of the full disease plants.

Table 3.2: The effect of disease (speckled leaf blotch) on root length per unit area of soil at growth stage 61 of wheat cv. Kopara, in cores extracted within and between rows in 1979-1980 field trial.

Variable	Disease treatments		F test $P \leqslant 0.05$
	Nil disease[1]	Full disease	
Within rows	184.0[2]	173.4	NS
Between rows	185.9	141.9	NS
F test $P \leqslant 0.05$	NS	NS	

(1) Benomyl (250g a.i. ha^{-1}) and mancozeb (1600g a.i. ha^{-1}) were applied at the following growth stages : 22, 23, 24, 25, 33, 41, 51, 61, 75.

(2) Root length per unit area (L_A) of soil (cm cm^{-2}) in a 65 cm core length.

Table 3.3: The effect of disease (speckled leaf blotch) on root length per unit area and per volume of soil at growth stage 91 of wheat cv. Kopara, in cores extracted between rows in 1979-1980 field trial.

Soil profile	Disease treatments				LSD $P \leqslant 0.05$
Zone (cm)	Nil disease (1)	Late disease (2)	Early disease (3)	Full disease	
0-10 (4)	11.13	11.98	10.97	7.05	4.03
10-20 (4)	9.09	9.33	7.51	6.00	2.87
20-35 (4)	3.12	2.92	2.63	2.11	0.85
35-50 (4)	1.90	1.75	1.62	1.28	0.56
0-50 (5)	277.5	283.2	248.5	181.4	65.31

(1) Benomyl (250g a.i. ha^{-1}) and mancozeb (1600g a.i. ha^{-1}) were applied at the following growth stages : 22, 23, 24, 25, 33, 41, 51, 61, 75.

(2) Benomyl (250g a.i. ha^{-1}) and mancozeb (1600g a.i. ha^{-1}) were applied at the following growth stages : 22, 23, 24, 25, 33.

(3) Benomyl (250g a.i. ha^{-1}) and mancozeb (1600g a.i. ha^{-1}) were applied at the following growth stages : 41, 51, 61, 75.

(4) Root length per unit volume (L_V) of soil ($cm\ cm^{-3}$).

(5) Root length per unit area (L_A) of soil ($cm\ cm^{-2}$).

3.4 The effects of disease on yield and yield components

The header yields were adjusted to an equal moisture content (14%). With respect to each treatment, these yields were consistent with those of the quadrat samples (Table 3.4). Since the latter had a low variance compared to the header samples, the interpretation of results was based on the quadrat samples. Yield of the nil disease plants was higher than that for all other treatments. The yield of the late disease plants was less affected by disease than that of the early disease plants. Plants exposed to the full disease epidemic, however, yielded less than the late and early disease plants.

The yield reduction associated with the full disease epidemic was attributed to a lower number of grains per ear and individual grain weight. In the early disease plants a lower grain number per ear contributed towards the reduction in yield. An increase in individual grain weight compared with the full and the nil disease plants partly compensated for the observed reduction in grain number in the early disease plants.

The number of grains per ear was analysed in more detail to determine the effect on the mean number of spikelets per ear and grains per spikelet (Gaunt and Thomson, 1985). There was a lower number of grains per spikelet on both main stem and tillers in the early and full disease plants compared to the nil disease plants.

4 Discussion

Compared with plants fully protected from disease by a series of fungicide sprays during the crop cycle, yield in the full disease plants was reduced by 13%. Yield reductions by speckled leaf blotch were similar to losses reported by Brown and Paddick (1980) for wheat in Victoria, Australia. In a similar trial reported here (Chapter 2), when no disease developed because of unfavourable climatic conditions, there were no changes in green leaf area under a similar spray programme. It is therefore concluded that the effects reported here were owing to

Table 3.4: The effects of four different disease treatments on yield and yield components of wheat cv. Kopara in 1979-1980 field trial.

Variable	Disease treatments				LSD $P \leq 0.05$
	(1) Nil disease	(2) Late disease	(3) Early disease	Full disease	
Header yield ($t\ ha^{-1}$) at 14% moisture content	7.15	7.14	7.00	6.00	0.12
Quadrat yield ($gDM\ m^{-2}$)	734	726	705	636	25.2
Ear number m^{-2}	648	644	638	641	38.5
Grain number ear^{-1}	35.8	35.4	33.4	34.1	0.92
Individual grain weight (mg)	31.7	31.8	33.1	29.1	1.13

(1) Benomyl (250g a.i. ha^{-1}) and mancozeb (1600g a.i. ha^{-1}) were applied at the following growth stages : 22, 23, 24, 25, 33, 41, 51, 61, 75.

(2) Benomyl (250g a.i. ha^{-1}) and mancozeb (1600g a.i. ha^{-1}) were applied at the following growth stages : 22, 23, 24, 25, 33.

(3) Benomyl (250g ai.i ha^{-1}) and mancozeb (1600g a.i. ha^{-1}) were applied at the following growth stages : 41, 51, 61, 75.

disease, and not to phytotonic effects of the fungicides.

Detailed studies on plant development and growth were conducted in the same trial (Gaunt and Thomson, 1985). They found that the development of the speckled leaf blotch epidemic in field-grown wheat was similar to that reported by Sanderson (1978) for wheat grown in Canterbury in the 1975/1976 season. The presence of primary inoculum, and conducive climatic conditions during early crop growth, favoured the primary epidemic. There was a second epidemic during heading (G.S. 51-59), after the disease spread to the newly formed leaves. Speckled leaf blotch affected the plants from the onset of the epidemic to maturity, including the period of apparent decline of the disease during the rapid growth period in spring (Gaunt et al., 1985). The effects of disease on plant growth were determined as total green leaf area, and effects on yield as the number of plants and the number of grain bearing ears per unit area, spikelet number per ear, number of grains per spikelet, and individual grain weight. Green leaf area was reduced by disease from G.S. 24. This effect may be compared with stripe rust of wheat (Doodson et al., 1964a), where disease decreased translocation to developing leaves, thus reducing the rate of development and ultimate size of leaves (Doodson, 1976). However, disease did not affect those components determined before G.S. 31, i.e., the number of plants per unit area, the number of grain bearing ears, and the number of spikelets per ear produced. Although disease did not constrain yield when it was first present in significant amounts, the epidemiological potential (Teng and Gaunt, 1980) at this time may be important for later development of the disease epidemic. Disease which affected the plants before anthesis (G.S. 60), reduced the grain number per ear. There was no preferential loss of grains in the tillers for the maintenance of grains in the main stem. Grain number in all spikelets on the ear was reduced, and there was no selective effect on basal or apical spikelets. The constraint to grain number may have occurred over a period from floret initiation to a stage immediately following anthesis. The reduction was attributed to reduced spikelet and floret number (Gaunt and Thomson, 1985). This reduction may be related to mildew on barley and its effect on green leaf area (Carver and Griffiths, 1981, 1982), where the reduction in grain number was correlated with green leaf area. Total leaf area which was reduced by disease may have limited the amount

of photosynthates required for the development of the maximum potential number of spikelets and florets. It is postulated that disease may also have caused imbalances in plant growth regulators, as reported by Misaghi (1982), which may have affected the development of spikelets and florets. Disease between anthesis and grain filling affected grain weight. In addition to genetic potentials, grain weight may be affected for two reasons. The maximum potential grain size may not be attained because of reduced endosperm cell division (Brocklehurst, 1977, 1979), and second, the grain may not be completely filled. Nuclear division in the endosperm is related to the amount of photosynthates present, and may also be influenced by plant growth regulators (Brocklehurst, 1977). Therefore, a limitation to any one of these factors may reduce endosperm cell number. Grain filling may be constrained by the lack of photosynthates for conversion to starch within the endosperm (Bremner and Rawson, 1978). However, the photosynthetic capacity of the plant may not influence the yield through limiting the size of the grain, as the growth of the grain is limited by the rate of starch synthesis within the grain (Jenner and Rathjen, 1975).

In plants exposed to the early disease epidemic, disease severities were similar to those in the nil disease plants after G.S. 37. Grain number was not significantly different from the full disease plants (Table 3.4). Thus, it may be suggested that the total constraint to grain number occurred before the late disease control was effective. These plants also responded to late disease control by an increase in grain weight relative to the full disease plants. This suggests that the full disease plants may have also been constrained, either when grain size was determined (i.e., during endosperm nuclear division), or during grain filling. Carver and Griffiths (1981, 1982) and Lim (1982) did not obtain an increase in grain weight in compensation for reduced grain number caused by an early disease epidemic in barley. In contrast, compensation in grain weight was recorded in this trial. This may be because mean rainfall during the grain filling period (33.3 mm in December and 134.9 mm in January) was 49% higher than average. Therefore, the plants may have been able to realise a capacity for compensation which is normally prevented by low water availability usually experienced in Canterbury wheat crops.

Carver and Griffiths (1981) stressed the importance of the effect of disease on percent green leaf area, and hence on photosynthesis. It may be concluded, therefore, that yield reduction caused by disease was related to the reduction in total green leaf area during crop growth. A reduction in photosynthesis caused by a reduction in total green leaf area, may also result in a reduced amount of photosynthates available for root growth. In this trial, root length of the full disease plants measured at the beginning of flowering (G.S. 61) was not affected by disease. The lack of possible significant differences may be attributed to an insufficient number of samples obtained per plot (one). To minimise errors in root studies, Welbank and Williams (1968) suggested that more than one sample per plot is required, as root growth is more variable than shoot growth (Russell, 1977). Therefore, in the subsequent sampling, the sample number was increased to three per plot. An increase in total root length between anthesis and the fully ripe stage of the grain indicated that root growth was active. This is contrary to studies by Welbank and Williams (1968) and Mengel and Barber (1974), who suggested that linear growth ceased at about heading and was constant (reached a plateau) thereafter. McClure and Harvey (1962) and Hurd (1968), however, reported that root growth can continue in the grain development period under favourable nutrient and moisture conditions. Suitable nutrient and moisture conditions may favour increased shoot growth but not necessarily root growth. Although root growth may continue during the grain filling period, the amount of growth may not be greater than the amount of root death, resulting in no net increase in root growth. The presence of inadequate soil moisture may, however, favour increased root growth. The increase in root length from G.S. 61 to G.S. 91 observed in this trial was greater in the nil disease plants compared to the full disease plants. It is possible that this increase may have been favoured by soil water deficits.

Disease throughout the crop cycle was found to reduce total root length, when measured at G.S. 91, as reported by Martin and Hendrix (1974). Although reductions in root length per unit area were observed in the entire 0-50 cm zone, the greatest reduction was in the 0-20 cm zone. This may be expected from previous studies (Gregory et al., 1978b) which showed that the 0-30 cm zone is the region of greatest root activity (i.e., branching and uptake), and therefore may be affected

more by disease than the deeper zones. Total root length of plants exposed to the early disease epidemic was between that of the nil disease and the full disease plants. This indicates that disease in the early phase of plant growth may have reduced root growth, while the control of disease at later growth stages may have enhanced root growth. This suggests that the roots adjust quickly to the disturbance in root:shoot ratio brought about by disturbances to either shoot or root. As a greater proportion of photosynthates is partitioned to roots during the early growth stages (Mann, 1957; Bruinsma and Schuurman, 1966), it may be assumed that early disease reduces the amount of photosynthates available for root growth. Therefore, disease may have a greater effect on early root growth compared to disease late in the crop cycle, as shown by the trend in this trial (Table 3.3). From these results, it is suggested that early disease in crop growth is more detrimental to root growth than late disease.

Reduced root growth may also affect shoot growth, as they are interdependent (Evans and Wardlaw, 1976). In the presence of soil water deficits, the effect of a reduced root system may be compounded, as a plant may not be able to explore the soil for water and nutrients as much as a plant with a well-developed root system. Husain and Aspinall (1970) demonstrated that primordia development was sensitive to water deficits. Langer and Ampong (1970) reported that spikelet production was affected by a prolonged period of water deficit. Roots are synthesisers of cytokinin (Itai and Vaadia, 1965) which enhances cell division (Skoog et al., 1967) and influences the retention of chlorophyll (Staskawicz et al., 1978). Therefore, it is suggested that reduced root length in early crop growth may affect primordia development and green leaf area. Wardlaw (1974) suggested that water deficits reduce fertilization, and increase sterility. Water deficits were also reported to affect the number of grains set in wheat (Asana, 1962) and barley (Aspinall et al., 1964). Kirkham and Kanemasu (1983) reported that grain yield was positively related to the amount of water available after anthesis. It is possible that a reduced root system caused by disease would therefore affect yield components and yield by reducing water and nutrient uptake in a soil with limited water. This may explain the compensating effect of increased grain weight in the presence of adequate soil water, even though roots were reduced by

disease in early crop growth.

The combined effects of disease (powdery mildew) and soil water deficits on pot-grown barley were examined by Ayres and Zadoks (1979). Powdery mildew infection decreased the root:shoot ratio in soils where water was not limiting. There was no effect on the ratio, however, in soils with water deficits, where a decrease in the ratio would have been more detrimental to crop growth. They also observed that powdery mildew neither induced water stress in well-watered plants nor exacerbated the stress already existing in plants in dry soil. There are no comparable studies for powdery mildew in either glasshouse or field studies, but cereal rusts under glasshouse conditions have been reported to produce similar effects (Johnston and Miller, 1934, 1940; Bever, 1937). The experiment of Ayres and Zadoks (1979) was conducted in controlled environments where humidity was regulated to constant levels during the light and dark periods, and air movement was restricted. Thus, the transpiration rate in these studies would have been less than that for field conditions, where atmospheric demand is usually greater. Rust fungi, unlike powdery mildew which show a reduction in stomatal water loss, increase the transpiration rate per unit area of leaf by rupturing the host epidermis (Duniway and Durbin, 1971a). In the field, more complex relationships between disease, root development and soil water deficits may be expected. Disease could exacerbate the effects of soil water deficits. Therefore, the hypothesis that impairment of the root system caused by disease may contribute to reduced grain yield through reduction in the ability of the plant to cope with water deficits, requires investigation. The water relations of foliar-diseased wheat were, therefore, the subject of further investigation in this research programme.

CHAPTER FOUR

THE EFFECTS OF DISEASE AND IRRIGATION ON ROOT DEVELOPMENT, YIELD AND YIELD COMPONENTS OF WHEAT

1 Introduction

Plant growth is influenced indirectly by soil water deficits and directly by plant water deficits. Soil water deficits are the differences between evapotranspiration and rainfall, while plant water deficits are influenced by the rate of water uptake and transpiration as well as soil water supply. In a soil with water deficits, a plant may reduce transpiration to prevent an increase in plant water deficits. Thus it may not be assumed that a given soil water deficit will be accompanied by an equal amount of plant water deficit (Kramer, 1963). However, prolonged soil water deficits, even if they are small, usually result in plant water deficits. The water relations of rust-infected cereals have been investigated since the beginning of this century. Johnston and Miller (1934) reported that transpiration of rust-infected wheat leaves was increased compared to non-infected leaves. This increase was attributed to non-stomatal water loss after the rust pustules had erupted through the leaf epidermis. The loss was found to be greater in the dark compared to the light. In rust-infected bean plants, transpiration was reduced in comparison to healthy leaves before sporulation, and increased with the onset of sporulation (Duniway and Durbin, 1971a, 1971b). The initial reduction in transpiration was attributed to the closure of stomata, and stomatal closure was attributed to low leaf water potential (Duniway, 1971). Thus the water relations of foliar-infected plants are altered by changes in transpiration (Ayres, 1978); accordingly, plant water deficits may be increased indirectly by disease (Ayres and Zadoks, 1979; Ayres, 1982; Misaghi, 1982).

Plants infected by foliar diseases may have reduced root growth compared to healthy plants (see Chapter 1, Section 5.7). There is little information available on altered water relations of foliar-infected plants in relation to root growth, and their subsequent effects on plant growth and yield. It has been shown from studies reported in Chapter three that foliar disease affects root growth. The effect of foliar disease on root growth depends on the time at which initial infection occurs, and also on the duration and severity of the disease (Hendrix and Lloyd, 1970; Martin and Hendrix, 1974). Infection occurring during the early stages of crop growth (between seedling stage and tillering) has a greater effect on the ultimate size of the root system than infection at later growth stages (Martin and Hendrix, 1974). Diseased plants which have a reduced root system and an increased transpiration rate may become water stressed at an earlier stage than healthy plants.

The effect of soil water deficits on cereal growth has been documented for wheat (Day and Intalap, 1970; Wardlaw, 1971; Fischer, 1973; Innes and Blackwell, 1981) and barley (Aspinall, 1965; Hussain and Aspinall, 1970; Day et al., 1978, Brooks et al., 1982), and it has been shown that prolonged water deficits reduced yield. Short periods of water deficits affected processes such as tillering, stem elongation (Aspinall et al., 1964) and the growth of the meristematic regions (Williams and Shapter, 1955). After these deficits were alleviated, growth at the meristematic regions occurred at the same rate as for plants which had not been water stressed. However, the plants used in the above experiments were grown in pots, and thus the roots would have been confined to a limited area compared to field-grown plants. The water relations of pot-grown plants may not be similar to field-grown plants, as the volume of soil available for root exploration in the field is generally greater. From field experiments, Day et al. (1978) reported that there was little evidence to suggest that any developmental stage was particularly sensitive to short periods of soil water deficits. However, under prolonged water deficits, the growth and development of organs initiated during the stress period was affected. For example, the number of grains per ear was closely related to the soil water deficit during spikelet and floret initiation. Soil water deficits at the following growth stages have been reported to affect

final grain yield: tillering (Salter and Goode, 1967), ear emergence and anthesis (Aspinall et al., 1964; Bingham, 1966; Wells and Dubetz, 1966; Fischer, 1973), and early grain development (Asana et al., 1958; Asana and Saini, 1962; Aspinall, 1965; Wardlaw, 1971; Brocklehurst et al, 1978; Davidson and Birch, 1978; Brooks et al., 1982). The events that occur during the period between ear emergence and anthesis (microsporogenesis, the expansion of the lodicules, and the growth of pollen filaments) are sensitive to short periods of water deficits. When soil water deficits occur at later growth stages, a reduced root system resulting from early disease may not be able to exploit the soil as much as the root system of a healthy plant. Thus the effects of disease and soil water deficits may be compounded, resulting in reduced plant growth and yield.

The combined effects of disease (powdery mildew) and soil water status on the water relations and growth of pot-grown barley were investigated by Ayres and Zadoks (1979). Both powdery mildew and soil water deficits restricted growth, and the effect was additive. Although powdery mildew reduced root growth, this did not induce plant water stress in soils even under severe water deficits. This was attributed to inhibited stomatal opening and a consequent reduction in evapotranspiration. Subsequently, there was a reduction in water demand caused by reduced shoot growth. However, shoot growth was not reduced in diseased plants where there were no soil water deficits, even though they had a reduced root system. In contrast to powdery mildew, rusts increased plant transpiration (Duniway and Durbin, 1971a). Thus a combination of rust and water deficits may act synergistically on wheat, causing yield reductions (Hendrix and Lloyd, 1970).

It is hypothesised that the reduced amount of roots in plants affected by early disease may induce plant water stress during later growth stages, when soil water deficits are likely to occur. This may reduce the ability of the plant to compensate for the reduction in those yield components affected by disease at early growth stages. The effect of different disease epidemics (nil, early and full) on shoot and root growth, and water use of wheat, was studied. Irrigation was applied at later growth stages to alleviate plant water stress caused by a reduced root system, to test whether compensation occurred in later determined yield components.

2 Materials and Methods

The trial was carried out on a Templeton silt loam soil near Lincoln College on land that was previously used for growing peas and oats. On 4th June 1981 Kopara wheat seed, treated with captan (100 g a.i. per 100 kg seed), was sown at 100 kg ha^{-1}. Single superphosphate (8% P) was applied at sowing at 250 kg ha^{-1}.

On 13th August (G.S. 12), 12 x 10 m plots were marked out with an access path (0.5 m) bordering each plot. There were four randomised blocks, each block and plot being separated by a 5 m buffer zone which was untreated. Each replicate block consisted of six randomised treatments: nil, early and full disease, with and without irrigation.

On 17th August (G.S. 23), plant population was assessed in five randomly placed 0.1 m^2 quadrats in each plot. On 15th September (G.S. 24) and 9th October (G.S. 30), plots were sprayed with MCPA (900 g a.i. ha^{-1}) and dicamba (150 g a.i. ha^{-1}) using a tractor mounted boom with type 1/4 T Teejet nozzles, to control broad leaf weeds.

Fungicide sprays were applied as described in Chapter 2, Section 2, at seven growth stages during crop growth (Table 4.1), using triadimefon (250 g a.i. ha^{-1}), chosen for its efficacy against speckled leaf blotch (Thomson *et al.*, 1981), stripe rust (Moore, pers. comm.) and minimal phytotonic effects.

Ten plants from each plot were sampled weekly at random (Table 4.1), and growth and disease severity were measured as described previously (see Chapter 2, Section 2).

At four growth stages (14 24, 18 31, 80, 92), three soil cores were extracted per plot, as described in Chapter 2, Section 2. The cores were extracted to a depth of 50, 50, 70 and 75 cm at each of the four samplings respectively. The cores were cut into several lengths (10-25 cm), pooled for each plot, and the roots extracted and measured (see Chapter 2, Section 2).

Volumetric soil water content was measured at 10 cm intervals by neutron moderation, using a Troxler depth moisture gauge (Model 127 7SN 478, Troxler Electronics Laboratories Incorporated, North Carolina,

Table 4.1: Time of plant and root samplings, and fungicide applications in 1981-1982 field trial on wheat cv. Kopara.

Date	Growth stage (1)	Assessment of plant development and disease severity (3)	Fungicide applications (2)		Root sampling
			Full	Late	
24/8	21	x	-	-	-
31/8	22	x	-	-	-
7/9	23	x	-	-	-
10/9	23	-	x	-	-
14/9	24	x	-	-	-
18/9	24	-	-	-	x
20/9	24	-	x	-	-
21/9	24	x	-	-	-
6/10	30	x	-	-	-
9/10	30	-	x	-	-
13/10	31	x	-	-	-
19/10	32	-	-	-	x
20/10	32	x	x	x	-
27/10	33	x	-	-	-
2/11	37	x	-	-	-
3/11	37	-	x	x	-
9/11	42	x	-	-	-
16/11	55	x	-	-	-
24/11	60	x	-	-	-
26/11	65	-	x	x	-
4/12	70	-	-	-	x
14/12	83	x	-	-	-
21/12	85	x	-	-	-
23/12	85	-	x	x	-
29/12	86	x	-	-	-
7/1	87	x	-	-	-
14/1	90	x	-	-	-
15/1	92	-	-	-	x

(1) Decimal growth stage key (Zadoks et al., 1974).

(2) Triadimefon was applied at 250g a.i. ha^{-1}.

U.S.A.), to a depth of one metre. Two aluminium access tubes (1200 mm long, 40 mm internal diameter and 1.47 mm wall thickness), were installed randomly between rows at a minimum distance of two metres inside each plot at diagonally opposed ends. Soil water content was assessed weekly in all plots from G.S. 25 until harvest.

At G.S. 79, 230 mm of water was applied to those plots designated to be irrigated, by trickle irrigation over a period of 36 hours. The trickle system consisted of whiskers 150 mm long (0.018 mm internal diameter) set 240 mm apart on lateral pipes (25 mm internal diameter) laid between every third row. The lateral pipes were connected to a header pipe (50 mm internal diameter) which received water from a header tank.

At maturity, plants in 15 quadrats (0.1 m^2) were sampled randomly within the 10 x 5 m central strip of each plot, bulked, and the total numbers of main stems and tillers with grain bearing ears were determined. A twenty-five plant subsample was analysed in detail for spikelet number and grain number per ear on the main stem and each tiller. The main stems and tillers were threshed separately, the grain dried at 80^{o}C for 24 hours, and individual grain weight for main stem and each tiller determined. The remainder of the quadrat samples was mechanically threshed (Kurt Pelz Saatweister Allesdrescher K35, West Germany), cleaned, and the total dry weight determined. The central 10.0 x 1.5 m strip was harvested by header (Walter and Wintersteiger Universal Seed Master, Austria) and the yield determined. The individual grain weights of the quadrat and header harvest were obtained as described in Chapter 2, Section 2.

After harvest, water content in the soil around all the access tubes was determined gravimetrically to a depth of one metre for each 10 cm increment. The neutron probe was calibrated by regressing the volumetric water content against the gravimetric water content for each depth. The neutron probe readings for each access tube were converted to volumetric water content using the regression slopes and intercept for a Templeton silt loam soil (Reid, pers. comm.). As there was loss of radiation from the neutron source in the top 10 cm layer, a good estimate of volumetric water content in this layer was not obtained. Therefore the gravimetric water content in the 0-20 cm layer was

regressed against the neutron probe reading at 20 cm to obtain a good correlation. The regression slope and intercept thus obtained were used for calculating the volumetric water content in the 0-20 cm layer for all probe readings.

3 Results

3.1 Development of the disease epidemics

Diseases were first detected in the crop at G.S. 22 when the plants had three emerged leaves. The combined natural epidemic reached a maximum severity at G.S. 16 30 (Table 4.2). The epidemic declined with the senescence of diseased lower leaves and the emergence of new leaves in spring at G.S. 31. Symptoms were present, on the lower leaves only, until G.S. 75, when there was a second peak of disease. Although disease spread to the upper leaves, the subsequent severities did not exceed 8% (at G.S. 85). In the early disease plants a similar epidemic was observed until G.S. 32. Thereafter, disease was controlled successfully and disease severities were lower than those in the full disease plants, and similar to the nil disease plants. In the nil disease plants disease was maintained at low severities throughout the season. In these plants, no disease was detected between G.S. 31 and G.S. 60 and only low amounts at other growth stages (maximum 2.4%).

Brown rust, powdery mildew (caused by Erysiphe graminis f.sp. tritici), speckled leaf blotch and stripe rust were all present in the trial, with stripe rust being the major disease. Large amounts of stripe rust inoculum from volunteer plants in the adjoining fields contributed towards the build up of the epidemic, with the bottom three leaves being heavily infected by G.S. 30. Subsequently, stripe rust spread to the emerging upper leaves. The full disease and early disease plants had significantly greater amounts of stripe rust than the nil disease plants from G.S. 24 to G.S. 37 (Table 4.2a). At G.S. 42, the severity of stripe rust had declined in the early disease plants, thus the full disease plants had more disease than the nil and early disease plants at this growth stage. There was no apparent disease in any of

Table 4.2: The effect of three different disease treatments and irrigation on the mean percentage disease severity on main stems of wheat cv. Kopara in 1981-1982 field trial.

Sampling date	Growth stage[1]	Disease treatments						LSD P≤0.05
		Nil disease[2]		Early disease[3]		Full disease		
		UI[4]	I[5]	UI	I	UI	I	
24/8	13,22	0.02	0.39	0.36	0.04	0.03	0.05	0.58
31/8	22	0.26	0.18	0.13	0.02	0.12	0.06	0.25
7/9	14,23	0.03	0.14	0.45	0.08	0.18	0.24	0.56
14/9	24	1.37	2.41	1.99	2.58	4.21	4.06	3.65
21/9	15,24	1.54	1.28	9.36	7.47	11.58	7.31	3.85
28/9	30	0.76	2.36	11.37	12.99	16.53	15.02	9.37
6/10	16,30	0.90	1.35	25.61	19.37	18.26	22.79	10.11
13/10	17,31	0.00	0.00	6.37	3.97	4.10	6.81	3.08
20/10	18,32	0.00	0.00	2.31	1.97	2.73	2.88	1.78
27/10	19,33	0.00	0.00	2.38	2.91	2.94	2.13	1.05
2/11	37	0.00	0.00	0.88	1.31	2.27	2.40	1.62
9/11	42	0.00	0.00	0.20	0.20	2.30	2.24	1.46
16/11	55	0.00	0.00	1.17	0.28	1.05	3.80	3.64
24/11	60	0.00	0.00	0.01	0.06	0.36	0.56	2.44
7/12	75	0.06	0.17	0.10	0.18	1.15	1.46	0.33
14/12	83	0.59	1.12	0.83	1.73	2.57	3.06	1.24
21/12	85	2.08	1.85	2.48	1.38	7.99	7.60	2.89
29/12	86	0.00	1.81	0.00	0.00	0.88	6.25	4.79

(1) Decimal growth stage (Zadoks et al., 1974).

(2) Triadimefon (250g a.i. ha^{-1}) was appied at the following growth stages : 23, 24, 30, 32, 37, 65, 85.

(3) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 32, 37, 65, 85.

(4) Unirrigated.

(5) Irrigation (230 mm) was applied at growth stage 83.

Table 4.2a: The effect of three different disease treatments and irrigation on the mean percentage disease severity of stripe rust on main stems of wheat cv. Kopara in 1981-1982 field trial.

Growth stage[1]	Disease treatments						LSD P≤0.05
	Nil disease[2]		Early disease[3]		Full disease		
	UI[4]	I[5]	UI	I	UI	I	
13,22	0.00	0.33	0.25	0.00	0.00	0.01	0.53
22	0.19	0.06	0.00	0.00	0.00	0.00	0.24
14,23	0.00	0.03	0.40	0.03	0.04	0.11	0.46
24	1.20	2.27	1.90	2.36	4.05	3.75	3.64
15,24	1.33	1.08	8.77	6.65	10.78	6.94	4.04
30	0.42	1.73	10.13	9.74	12.91	11.47	7.80
16,30	0.61	1.22	24.91	18.02	16.61	21.46	9.67
17,31	0.00	0.00	6.37	3.97	4.10	6.81	3.08
18,32	0.00	0.00	2.31	1.97	2.73	2.88	1.78
19,33	0.00	0.00	2.41	2.91	2.94	1.71	1.05
37	0.00	0.00	0.88	1.31	2.27	2.40	1.62
42	0.00	0.00	0.20	0.20	2.30	2.24	1.46
55	0.00	0.00	1.07	0.28	1.05	1.30	3.64
60	0.00	0.00	0.01	0.06	0.36	0.56	2.44
75	0.00	0.00	0.00	0.04	0.58	1.09	0.24

(1) Decimal growth stage (Zadoks et al., 1974).

(2) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 23, 24, 30, 32, 37, 65, 85.

(3) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 32, 37, 65, 85.

(4) Unirrigated.

(5) Irrigation (230 mm) was applied at growth stage 83.

the treatments at G.S. 55 and G.S. 60. At G.S. 75, stripe rust severity increased in the full disease plants. Speckled leaf blotch was confined to the lower leaves and was present from G.S. 23 to G.S. 30 (Table 4.2b). Pycnidia were produced sparsely on lesions, but secondary spread was very low and the epidemic did not develop at later growth stages, although traces of speckled leaf blotch were observed at G.S. 75. Powdery mildew was observed from G.S. 22 to G.S. 30 in all treatments and was confined mainly to the lower leaves. The mean severity was not greater than 1% at any growth stage (Table 4.2b). Pustules of brown rust were observed from G.S. 75 to G.S. 85 in all treatments, and disease severity was significantly higher in the full disease plants at G.S. 85 compared to all other treatments (Table 4.2b). The development of disease was not influenced by irrigation until G.S. 86 when brown rust was observed to reach 6.2% mean severity in the full disease irrigated plants compared to 0.88% in the full disease unirrigated plants. The increase in leaf area duration in the irrigated plants may have prolonged the disease.

3.2 The effect of disease and irrigation on total green leaf area

The total green leaf area of the early and the full disease plants was the same as the nil disease plants up to G.S. 24. At G.S. 15 30 there was a reduction in total green leaf area in the diseased plants compared to the nil disease plants (Table 4.3). The reduction was attributable to a lower percentage green leaf area (Table 4.5) caused by an increase in percentage disease severity, and disease induced and natural senescence. Disease induced senescence was that proportion of the non green leaf area not directly occupied by disease lesions nor attributable to natural senescence. This was calculated by subtracting the percentage area occupied by disease from the non green leaf area and comparing the remaining senesced area. If this was found to be greater in the disease plants than the nil disease plants, the difference was considered as disease induced senescence. For example, at G.S. 15 30, the maximum percentage non green leaf area in the full disease unirrigated plants was 65.07 (100 - 34.93) and the percentage area occupied by disease symptoms for the corresponding plants was 16.53 (Table 4.2). The total percentage senesced area therefore was 48.54

Table 4.2b: The effect of three different disease treatments and irrigation on the mean percentage disease severity of speckled leaf blotch, powdery mildew, and brown rust on main stems of wheat cv. Kopara, in 1981-1982 field trial.

Disease	Growth stage[1]	Disease treatments						LSD P≤0.05
		Nil disease[2]		Early disease[3]		Full disease		
		UI[3]	I[4]	UI	I	UI	I	
Speckled leaf blotch	13,22	0.02	0.04	0.00	0.00	0.00	0.00	0.06
	22	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	14,23	0.00	0.09	0.02	0.00	0.05	0.11	0.10
	24	0.13	0.03	0.06	0.16	0.05	0.19	0.17
	15,24	0.13	0.10	0.30	0.67	0.67	0.28	0.50
	30	0.11	0.21	0.79	0.72	0.60	0.63	0.81
	16,30	0.25	0.14	0.73	1.30	1.63	1.22	1.13
	75	0.00	0.00	0.03	0.00	0.03	0.02	0.06
	83	0.00	0.00	0.00	0.02	0.00	0.01	0.02
	85	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Powdery mildew	13,22	0.00	0.02	0.11	0.04	0.03	0.04	0.11
	22	0.01	0.10	0.10	0.01	0.01	0.05	0.10
	14,23	0.03	0.03	0.03	0.05	0.10	0.03	0.14
	24	0.08	0.02	0.08	0.07	0.12	0.18	0.11
	15,24	0.09	0.10	0.21	0.15	0.20	0.09	0.25
	30	0.12	0.09	0.23	0.36	0.16	0.17	0.33
	16,30	0.05	0.00	0.01	0.06	0.03	0.11	0.13
Brown rust	75	0.06	0.17	0.08	0.14	0.58	0.36	0.28
	83	0.59	1.12	0.83	1.73	2.57	3.05	1.24
	85	2.08	1.85	2.48	1.35	7.99	7.60	2.87
	86	0.00	1.81	0.00	0.00	0.88	6.25	4.79

(1) Decimal growth stage (Zadoks et al., 1974).

(2) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 23, 24, 30, 32, 37, 65, 85.

(3) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 32, 37, 65, 85.

(4) Unirrigated.

(5) Irrigation (230 mm) was applied at growth stage 83.

(65.07 - 16.53). The area senesced in the nil disease unirrigated plants was 32.28% (100 - 67.72), and the area occupied by disease was 1.05%. The total percentage senesced area was 31.23 (32.28 - 1.05). Therefore the disease induced senescence was 17.31% (48.54 - 31.23) in the disease plants. One week later, after the emergence of the sixth leaf (G.S. 16 30) total green leaf area in the full disease plants had increased by only 2.15 cm^2 compared to an increase of 5.75 cm^2 in the nil disease plants. The lower increase in total green leaf area, despite the expansion of the sixth leaf, was attributable to high disease severity (20.5%) in the full disease plants which reached a peak at this growth stage (Table 4.2). At G.S. 17 31, total green leaf area increased in all plants with the rapid emergence of new leaves in spring (Table 4.3). However, the disease plants had lower total green leaf areas, attributable to decreased total leaf area (Table 4.4) and percentage green leaf area (Table 4.5). Reduction in the latter was contributed to by areas occupied by disease lesions and disease induced senescence on lower leaves.

Controlling early disease by the application of fungicides at G.S. 32 in the early disease plants produced an increase in total green leaf area by G.S. 60, compared to the full disease plants. This was attributable to a decreased disease severity and a lower disease induced senescence on lower leaves. In turn, these both contributed to a higher percentage green leaf area. At G.S. 42, after emergence of the flag leaf, the full disease, early disease and nil disease plants had mean total green leaf areas of 46.6 cm^2, 54.0 cm^2 and 64.1 cm^2 respectively, the greatest area attained in all plants for the growth cycle. Mean percentage green leaf area was also at a maximum at this growth stage; the full disease, early disease and nil disease plants had 57.4%, 61.7% and 75.5% respectively. By G.S. 60, the total green leaf area in the early disease plants was the same as that in the nil disease unirrigated plants. In the full disease plants, disease induced senescence contributed to the reduction in percentage green leaf area to a greater extent than percentage disease severity, thus reducing the total green leaf area from G.S. 60 to G.S. 83. The decrease in total green leaf area in all plants at G.S. 85 and 86 compared to the previous growth stages was attributed solely to natural senescence.

Table 4.3: The effect of three different disease treatments and irrigation on total green leaf area on main stems of wheat cv. Kopara in 1981-1982 field trial.

Sampling date	Growth stage[1]	Disease treatments						LSD P≤0.05
		Nil disease[2]		Early disease[3]		Full disease		
		UI[4]	I[5]	UI	I	UI	I	
24/8	13,22	5.98[6]	5.91	5.30	5.70	5.39	5.49	1.04
31/8	22	5.88	6.35	5.55	7.00	7.44	7.17	1.75
7/9	14,23	10.57	9.56	9.17	10.88	10.42	9.99	1.62
14/9	24	11.87	12.31	11.90	12.13	11.87	11.40	1.55
21/9	15,24	17.75	18.93	15.12	17.51	16.80	15.01	1.39
28/9	30	18.17	17.18	12.27	11.07	11.06	10.01	3.73
6/10	16,30	23.68	23.13	10.74	11.65	13.12	12.25	5.15
13/10	17,31	24.32	26.03	19.18	20.76	20.70	20.22	3.76
20/10	18,32	33.46	34.58	26.52	28.17	27.85	25.13	6.67
27/10	19,33	41.63	48.98	32.91	33.31	37.17	31.98	6.12
2/11	37	50.05	58.78	41.77	34.43	35.73	36.57	4.84
9/11	42	60.00	68.16	56.13	51.85	45.97	47.20	9.51
16/11	55	54.67	62.45	52.06	46.96	43.18	39.69	4.18
24/11	60	48.19	58.47	48.61	44.42	33.20	35.50	5.18
7/12	75	35.08	47.14	39.43	34.15	23.56	26.06	5.04
14/12	83	25.36	32.42	27.73	26.84	13.55	17.15	3.87
21/12	85	7.98	18.53	12.85	13.67	3.38	8.48	5.54
29/12	86	0.15	2.01	0.10	1.11	0.01	0.43	1.07

(1) Decimal growth stage (Zadoks et al., 1974).

(2) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 23, 24, 30, 32, 37, 65, 85.

(3) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 32, 37, 65, 85.

(4) Unirrigated.

(5) Irrigation (230 mm) was applied at growth stage 83.

(6) Total green leaf area (cm^2).

Table 4.4: The effect of three different disease treatments and irrigation on total leaf area on main stems of wheat cv. Kopara in 1981-1982 field trial.

Sampling date	Growth stage[1]	Disease treatments						LSD P≤0.05
		Nil disease[2]		Early disease[3]		Full disease		
		UI[4]	I[5]	UI	I	UI	I	
24/8	13,22	6.02[6]	5.96	5.35	5.75	5.43	5.54	0.93
31/8	22	5.98	6.43	5.62	7.06	7.46	7.23	1.74
7/9	14,23	10.71	9.69	9.37	11.04	10.63	10.15	1.66
14/9	24	12.27	12.83	12.40	12.56	12.57	12.04	1.49
21/9	15,24	19.91	20.70	18.43	20.80	20.33	18.31	2.25
28/9	30	22.08	22.14	21.45	21.72	22.25	21.81	1.34
6/10	16,30	36.13	35.56	32.91	34.31	35.36	33.89	2.46
13/10	17,31	44.15	44.43	41.18	43.05	44.87	42.65	2.97
20/10	18,32	49.71	51.45	47.25	48.58	49.02	45.82	8.06
27/10	19,33	60.40	67.63	59.76	61.20	65.11	58.89	8.24
2/11	37	64.53	70.70	65.41	64.01	64.90	63.21	4.63
9/11	42	78.88	86.00	84.28	79.76	78.04	78.05	4.52
16/11	55	78.88	86.00	84.28	79.91	79.32	78.37	4.25
24/11	60	78.88	86.00	84.28	79.91	79.32	78.37	4.27
7/12	75	65.19	72.58	71.79	67.59	66.20	66.38	4.27
14/12	83	48.69	55.92	56.47	51.41	49.94	50.42	2.96
21/12	85	48.69	55.92	56.47	51.41	49.94	50.42	2.92
29/12	86	32.18	37.10	37.91	33.58	32.76	32.90	2.24

(1) Decimal growth stage (Zadoks et al., 1974).

(2) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 23, 24, 30, 32, 37, 65, 85.

(3) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 32, 37, 65, 85.

(4) Unirrigated.

(5) Irrigation (230 mm) was applied at growth stage 83.

(6) Total leaf area (cm^2).

Table 4.5: The effect of three different disease treatments and irrigation on mean percentage green leaf area on main stems of wheat cv. Kopara in 1981-1982 field trial.

Sampling date	Growth stage[1]	Disease treatments						LSD P≤0.05
		Nil disease[2]		Early disease[3]		Full disease		
		UI[4]	I[5]	UI	I	UI	I	
24/8	13,22	98.93	98.84	98.62	98.69	99.23	98.99	0.61
31/8	22	98.17	98.74	98.79	99.03	99.06	99.01	1.15
7/9	14,23	97.86	98.12	97.03	97.85	97.09	97.74	0.98
14/9	24	95.14	93.59	93.74	94.36	91.55	91.09	4.53
21/9	15,24	80.96	81.21	71.84	75.07	70.34	72.21	11.84
28/9	30	67.72	58.50	42.03	32.29	34.93	32.10	19.06
6/10	16,30	44.19	38.17	18.43	18.98	17.98	22.41	10.88
13/10	17,31	43.62	47.11	35.38	36.23	33.94	34.30	5.24
20/10	18,32	62.72	62.32	51.94	53.50	53.80	50.41	5.4
27/10	19,33	66.52	67.38	53.22	50.27	54.41	53.09	5.09
2/11	37	78.17	80.00	56.75	55.42	50.80	53.11	7.02
9/11	42	74.54	76.42	61.87	61.43	57.61	57.11	3.95
16/11	55	67.80	69.17	56.07	55.43	52.52	48.07	4.88
24/11	60	59.74	64.35	52.10	52.26	40.60	42.96	5.66
7/12	75	54.41	64.55	52.72	50.57	36.65	40.00	7.07
14/12	83	53.38	59.13	49.97	53.53	29.61	36.04	6.46
21/12	85	17.35	32.43	24.19	28.57	7.64	18.57	10.29
29/12	86	0.50	6.13	0.31	3.63	0.04	1.73	3.75

(1) Decimal growth stage (Zadoks et al., 1974).

(2) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 23, 24, 30, 32, 37, 65, 85.

(3) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 32, 37, 65, 85.

(4) Unirrigated.

(5) Irrigation (230 mm) was applied at growth stage 83.

In the nil disease plants, total green leaf areas between the unirrigated plants and those designated to be irrigated were the same until G.S. 32. From G.S. 33 to G.S. 86, total green leaf areas in the irrigated plants were significantly higher than in the unirrigated plants. These differences were attributed to a reduced total leaf area in the unirrigated plants from G.S. 37 to G.S. 60, and at G.S. 83, and a combined reduction in total leaf area and percentage green leaf area at G.S. 75, 85, and 86. These differences cannot be explained as the treatments were identical until the application of water at G.S. 83. There was a non-significant response to irrigation amongst the full disease and the early disease plants, and the total green leaf areas in the plants from these treatments were similar to those for the unirrigated plants. Among the nil disease plants, those that received irrigation did show significant increases in total green leaf areas at G.S. 85 and G.S. 86. It is not certain whether the response at G.S. 85 was owing to irrigation, as total green leaf areas of the nil disease irrigated and the nil disease unirrigated plants were starting to show a difference before irrigation was applied.

3.3 The effect of disease and irrigation on root development

Root lengths extracted from soil cores to a maximum depth of 75 cm were measured at different depths and presented as root length per unit area of soil (Table 4.6). Root length of the nil disease plants increased from G.S. 24 to 31, and from G.S. 80 to 92, and maximum length was recorded in the final sampling (G.S. 92). There was an increase (121%) in total root length of the nil disease unirrigated plants at G.S. 31 compared to the previous sampling, attributable to significant increases in the 0-10 cm (100%), 10-20 cm (187%), 20-30 cm (113%), and 35-50 cm (45%) zones (Table 4.7). By the grain ripening stage (G.S. 92), the total root length had increased 51% from the previous sampling, associated with increases of 79%, 30%, 102%, and 79% in the 0-10 cm, 10-20 cm, 35-50 cm, and 50-75 cm zones respectively (Table 4.7).

There were no significant differences in root length at G.S. 24 associated with disease in the early and full disease plants. By G.S. 31, reduced root length was observed in the early and full disease unirrigated plants in comparison with the nil disease unirrigated plants (27% and 39% respectively). This was associated with significant reductions of 30% and 40% in the 0-10 cm, and 35% and 49% in the 10-20 cm zones respectively. In the early disease unirrigated plants, there was an increase (37%) in root length by G.S. 80 from the previous sampling, after the control of disease by fungicide sprays which commenced at G.S. 31. At G.S. 80, there were no significant differences in root length between the early disease unirrigated plants and the nil disease unirrigated plants. The increase in total root length occurred in the 0-10 cm (41%) and 10-20 cm (51%) zones. However, in the full disease unirrigated plants, there was no increase between G.S. 31 and 80, and the total root length was less (37%) than the nil disease unirrigated plants. Disease affected root development in the full disease unirrigated plants, resulting in significantly lower root lengths (32%, 40%, 43%, and 48% in the 0-10 cm, 10-20 cm, 20-30 cm and 50-70 cm zones respectively) compared with the corresponding zones of the nil disease unirrigated plants. At G.S. 92, there was no increase in root length from G.S. 80 in the early disease unirrigated plants, and there was significantly less (34%) root length than the nil disease unirrigated plants. Reduced root growth in the 0-10 cm (41%) and 10-20 cm (31%) zones contributed to the difference in total root length in the early disease unirrigated plants, compared to the nil disease unirrigated plants. In the full disease unirrigated plants, total root length increased significantly (44%) at G.S. 92 from the previous sampling (G.S. 80), and was similar to total root length in the early disease unirrigated plants. However, there was still a 40% difference compared to the nil disease unirrigated plants, associated with differences in the 0-10 cm (46%) and 10-20 cm (45%) zones.

Irrigation was not applied until G.S. 83, and consequently there was no difference in root length in irrigated plants compared to the corresponding unirrigated plants in three samplings (G.S. 24, 31 and 80). After irrigation, there was no increase in root length in any of the irrigated plants. At G.S. 92, the nil and early disease irrigated plants had shorter root lengths (28% and 39%) compared to the

Table 4.6: The effect of three different disease treatments and irrigation on root length per unit area in the soil profile of wheat cv. Kopara in 1981-1982 field trial.

Growth stage[1]	Disease treatments						LSD P≤0.05
	Nil disease[2]		Early disease[3]		Full disease		
	UI[4]	I[5]	UI	I	UI	I	
24	93.0[6]	89.6	91.0	89.7	87.2	87.0	17.0
31	205.7	169.2	150.0	145.3	126.3	139.6	36.8
80	210.4	225.1	204.9	167.4	133.1	120.0	38.5
92	317.5	229.6	208.7	127.8	191.2	133.2	61.0
LSD (0.05)	65.8	33.4	24.5	48.4	26.7	38.1	

(1) Decimal growth stage (Zadoks et al., 1974).

(2) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 23, 24, 30, 32, 37, 65, 85.

(3) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 32, 37, 65, 85.

(4) Unirrigated.

(5) Irrigation (230 mm) was applied at growth stage 83.

(6) Root length per unit area (L_A) of soil ($cm\ cm^{-2}$) extracted from a core length of 50 cm (G.S.'s 24 and 31), 70 cm (G.S. 80), and 75 cm (G.S. 92).

Table 4.7: The effect of three different disease treatments and irrigation on root length per unit volume of soil in different zones of the soil profile, of wheat cv. Kopara in 1981-1982 field trial.

Soil profile Zone (cm)	Growth stage (1)	Disease treatments						LSD P≤0.05
		Nil disease (2)		Early disease (3)		Full disease		
		UI (4)	I (5)	UI	I	UI	I	
0-10	24	3.94 (6)	3.93	3.90	3.86	3.75	3.74	0.66
	31	7.86	6.74	5.51	5.11	4.75	4.90	1.30
	80	8.41	8.84	7.79	7.42	5.71	6.20	2.25
	92	15.07	10.57	8.85	5.26	8.19	5.35	3.07
LSD P≤0.05		2.88	2.19	1.88	1.72	1.49	1.18	
10-20	24	2.62	2.79	2.77	2.81	2.80	2.56	0.50
	31	7.52	6.26	4.91	5.07	3.87	4.78	1.30
	80	7.52	8.25	7.42	6.08	4.48	2.94	1.76
	92	9.75	6.31	6.76	3.62	5.35	2.94	1.74
LSD P≤0.05		2.21	1.09	0.99	2.12	1.31	1.06	
20-35	24	1.19	1.16	1.29	1.18	0.96	1.16	0.54
	31	2.53	1.72	2.03	1.97	1.69	2.09	0.83
	80	2.05	1.92	2.10	1.31	1.17	1.20	0.41
	92	1.73	1.68	1.91	0.97	1.37	1.62	1.10
LSD P≤0.05		0.93	1.04	1.05	0.85	0.67	1.04	
35-50	24	0.64	0.34	0.34	0.36	0.49	0.44	0.21
	31	0.93	0.90	1.02	0.93	0.98	0.84	0.55
	80	0.66	0.92	0.86	0.57	0.55	0.52	0.39
	92	1.33	1.28	0.88	0.82	1.19	0.95	0.74
LSD P≤0.05		0.93	0.58	0.57	0.57	0.46	0.62	
50-70	80	0.52	0.58	0.42	0.21	0.27	0.14	0.38
50-75	92	0.93	0.66	0.43	0.48	0.70	0.47	0.64

(1) Decimal growth stage (Zadoks et al., 1974).

(2) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 23, 24, 30, 32, 37, 65, 85.

(3) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 32, 37, 65, 85.

(4) Unirrigated.

corresponding unirrigated plants (Table 4.6). The root lengths of early and full disease irrigated plants were similar at G.S. 92, but significantly lower (44% and 42%) compared to the nil disease unirrigated plants. These reductions were associated with less root length in the 0-10 cm (50% and 49%), and 10-20 cm (43% and 53%) zones.

3.4 The effects of disease and irrigation on soil water content and water use

3.4.1 Total soil water content

In all treatments, total water content (Table 4.8) was below field capacity at all times of measurement (field capacity of a Templeton silt loam soil ranges between 290 - 311 mm). The irrigated plots may have reached field capacity after irrigation. The days between irrigation and the next soil water measurement may have allowed for loss by evaporation and/or drainage, thus reducing the water content to below field capacity.

The total water content in the nil disease unirrigated plots fluctuated during the growing season with water input from rainfall and crop water use (Table 4.8). The soil water content rapidly decreased between G.S. 55 (half inflorescence emerged) and G.S. 86 (hard-dough stage). Thereafter, there was no change in the soil water content in the unirrigated plots because of the loss of transpiring areas in the maturing crop. A similar trend in the changes in water content during crop growth was observed in the early disease unirrigated and full disease unirrigated plots. Total water content in the full disease unirrigated plots did not differ significantly from that in the nil disease unirrigated plots at all growth stages. The full disease unirrigated plots were not significantly different, except at G.S. 70 and 82, compared to the early disease unirrigated plots. The total water content in the early disease unirrigated plots was similar to that in the nil disease unirrigated plots for all growth stages. In all the plots, soil water content decreased rapidly between G.S. 55 and G.S. 82. After irrigation at G.S. 83, the soil water content in the irrigated plots increased by an average of 75% compared to the

Table 4.8: The effect of three different disease treatments and irrigation on the total water content (mm) in the soil profile (0-100 cm) of wheat cv. Kopara in 1981-1982 field trial.

Sampling date	Growth stage[1]	Disease treatments						LSD P≤0.05
		Nil disease[2]		Early disease[3]		Full disease		
		UI[4]	I[5]	UI	I	UI	I	
17/9	24	262.7	270.5	270.0	263.9	264.4	270.7	10.0
24/9	24	265.8	272.4	265.8	265.4	266.2	268.9	12.2
1/10	30	257.0	266.9	256.4	255.9	252.0	261.0	10.1
8/10	30	268.9	280.1	273.7	271.7	270.1	275.5	11.9
15/10	31	250.9	262.5	260.0	254.1	254.1	261.8	13.2
23/10	32	257.6	269.2	262.4	258.4	260.6	267.9	12.4
29/10	33	258.0	270.0	262.8	263.6	261.3	272.4	13.2
5/11	38	242.7	256.6	250.4	248.4	243.2	258.4	13.7
12/11	42	231.8	246.5	240.9	237.3	231.8	246.8	23.9
19/11	55	220.0	239.2	230.8	226.8	215.0	231.2	16.2
26/11	65	200.7	215.3	211.1	203.9	195.7	215.7	17.3
3/12	70	175.8	200.4	190.9	184.5	171.4	196.4	16.8
10/12	82	144.4	164.2	163.2	154.3	141.0	167.9	19.9
24/12	85	114.4	286.6	126.2	281.9	118.3	282.2	11.5
31/12	86	103.6	273.5	114.6	264.8	107.3	267.4	12.4
7/1	87	120.8	274.8	133.3	267.5	124.3	271.0	13.2
14/1	90	121.0	264.1	131.5	253.1	125.6	262.5	13.4
28/1[6]		121.9	247.6	131.0	238.9	123.7	236.3	16.0

(1) Decimal growth stage (Zadoks et al., 1974).

(2) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 23, 24, 30, 32, 37, 65, 85.

(3) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 32, 37, 65, 85.

(4) Unirrigated.

(5) Irrigation (230 mm) was applied at growth stage 83.

(6) Measurements taken after harvest.

unirrigated plots. The total water content among the plots designated to be irrigated was similar at all growth stages. Within the full disease plots, the unirrigated plots had less water at G.S. 38, 65, 70, and 82 compared to the plots designated to be irrigated. Total soil water content in the early disease plots was similar for both unirrigated plots and for those designated to be irrigated until irrigation was applied. Within the nil disease plots, those plots designated to be irrigated had similar soil water contents up to G.S. 55 compared to the unirrigated plots. At G.S. 55 and 70, the plots designated to be irrigated had a greater total water content, as the water use in these plots was low (Table 4.9).

The volumetric water content in the 0-20 cm layer of soil, and at 10 cm increments up to 100 cm depth was calculated from soil water measurements obtained weekly. The volumetric water content in the upper layers of soil (0-50 cm) fluctuated with extraction by roots, and replenishment by rainfall (Table 4.9). This fluctuation was greater in the 0-20 cm layer (Figure 4.1), and at 20 cm (Figure 4.2) and 30 cm (Figure 4.3) depths compared to the lower depths. At all depths, the irrigated plots had significantly greater amounts of water after irrigation at G.S. 83, until harvest.

The water content in the 0-20 cm layer of soil was consistently less than that for lower depths throughout the season. This may be associated with the greater quantity of root material in the upper layer (Table 4.7), loss by evaporation from the soil surface, and possible errors in soil moisture measurement associated with the use of the neutron probe. Water extraction increased after G.S. 38, although there was an increase in volumetric water content at G.S. 55. Between G.S. 55 and 86 there was a large amount of water extracted from this layer. There were no differences between disease treatments at all stages of growth (Figure 4.1).

At a depth of 20 cm, the volumetric water content in all plots was greater than that in the surface layers (0-20 cm). There was a rapid decline in water content from G.S. 55 to G.S. 86, as observed in the 0-20 cm layer. The amount of water extracted by the different disease plants did not differ at this depth (Figure 4.2).

Figure 4.1: The effect of three different disease treatments and irrigation on water content in the soil profile (0–20 cm) in 1981–1982 field trial on wheat cv. Kopara.

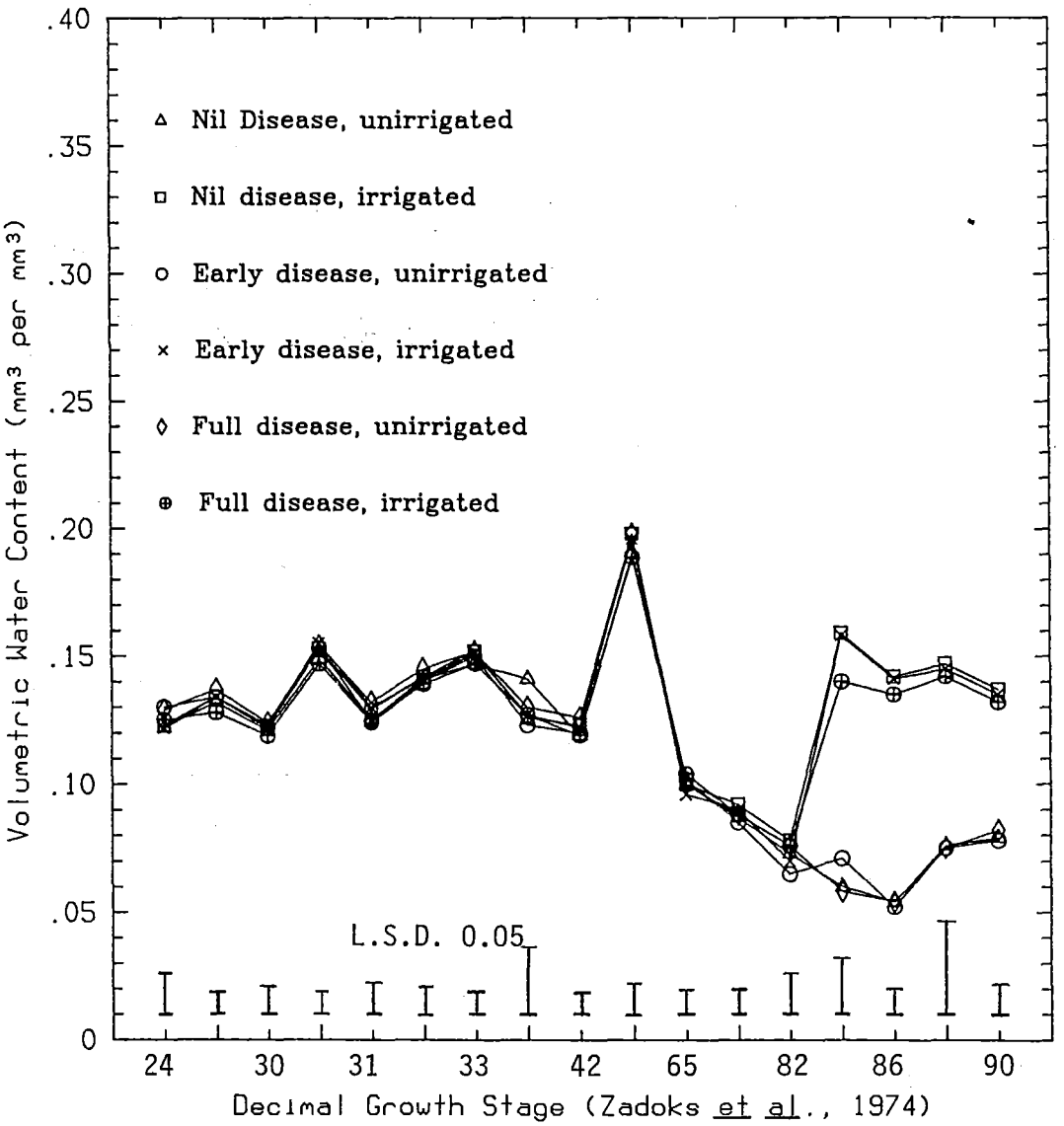
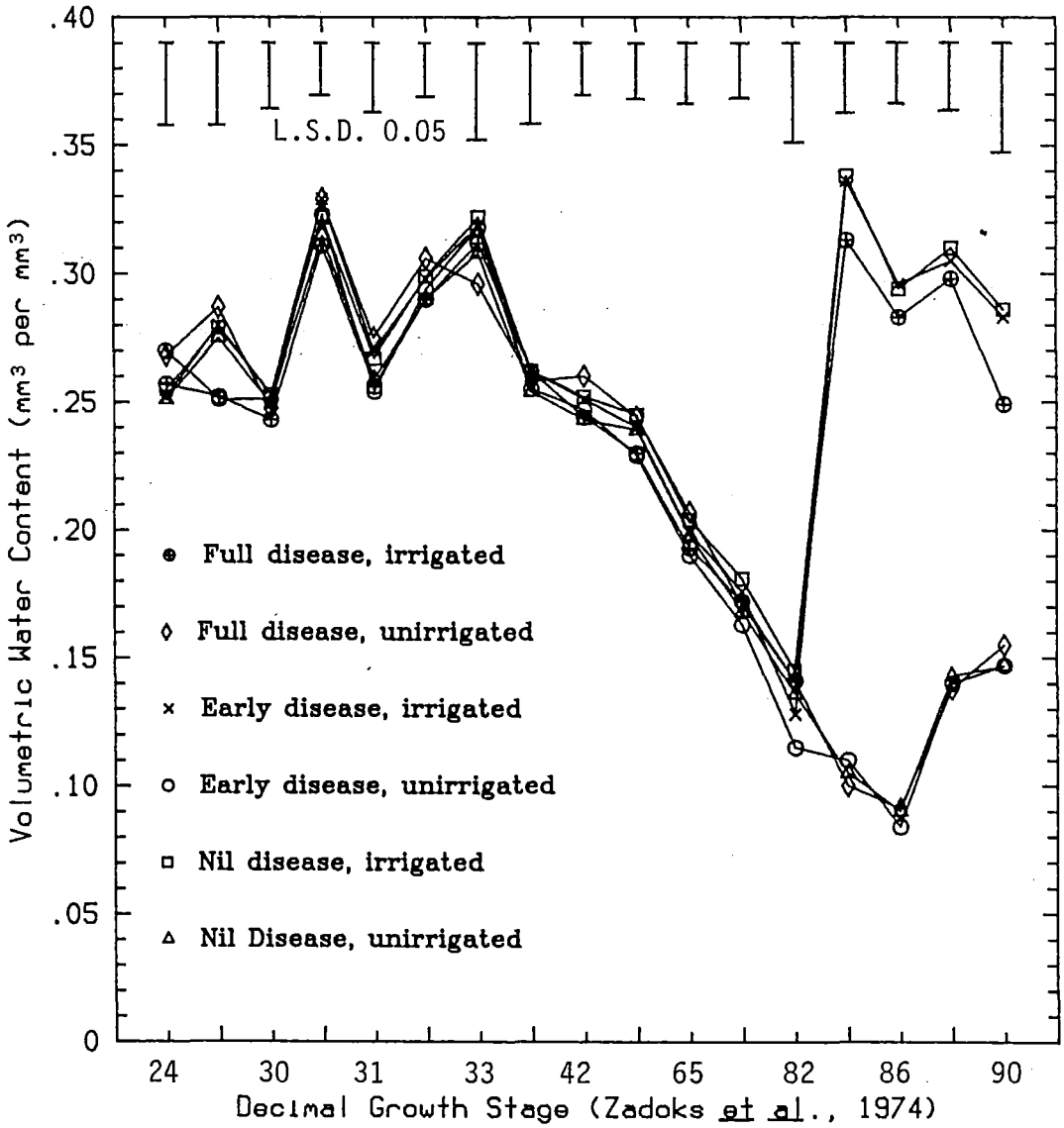


Figure 4.2: The effect of three different disease treatments and irrigation on water content in the soil profile (20 cm) in 1981–1982 field trial on wheat cv. Kopara.



The water content in all plots at 30 cm was slightly higher than that at 20 cm, but by G.S. 55, it had declined to the same level (Figure 4.3). Water content at 30 cm fluctuated up to G.S. 33, but with greater extraction from then on, declined gradually until G.S. 86. At G.S. 15 24, 33 and 38, there was a lower water content in the early disease plots designated to be irrigated compared to the early disease unirrigated plots. In comparison to the full disease unirrigated plots, the early disease plots designated to be irrigated had a significantly lower water content at G.S. 15 24, 31, 33, 38, and 42. These differences did not follow any particular trend in terms of treatment effects and thus are difficult to explain biologically.

At the time water measurements began, the water content at 40 cm was similar to that at 30 cm. Soil water content decreased gradually from G.S. 33 to G.S. 55, and then rapidly up to G.S. 85 (Figure 4.4). At G.S. 15 30 the water content of the early disease unirrigated plots was lower than that for those early disease plots designated to be irrigated. It was also lower at G.S. 31 and 33, than that for all the full disease plots, and at G.S. 70, for the full disease irrigated plots.

At 50 cm depth, the water content was slightly less than at 40 cm. However the pattern of water extraction was similar. As observed in the 30 and 40 cm depths, the early disease plots designated to be irrigated had lower water contents compared to the early disease unirrigated plots at G.S. 16 30 and 31, and full disease irrigated plots at G.S. 16 30, 33, and 82 (Figure 4.5). However, no consistent pattern in water extraction was observed at this depth.

The water content at 60 cm declined gradually from G.S. 33, and rapidly from G.S. 55 to G.S. 85 (Figure 4.6). Variations in water content were observed between treatments at this depth. Between G.S. 24 and 82 (except for measurements taken at G.S. 16 30 and 32), the full disease plots designated to be irrigated contained more water than the full disease unirrigated plots. Conversely, in the early disease plots, the unirrigated plots had a higher water content at G.S. 24 and between G.S. 42 and 82, compared to the plots designated to be irrigated. From G.S. 85 to harvest, the nil disease and full disease unirrigated plants extracted more water than the early disease

Figure 4.3: The effect of three different disease treatments and irrigation on water content in the soil profile (30 cm) in 1981–1982 field trial on wheat cv. Kopara.

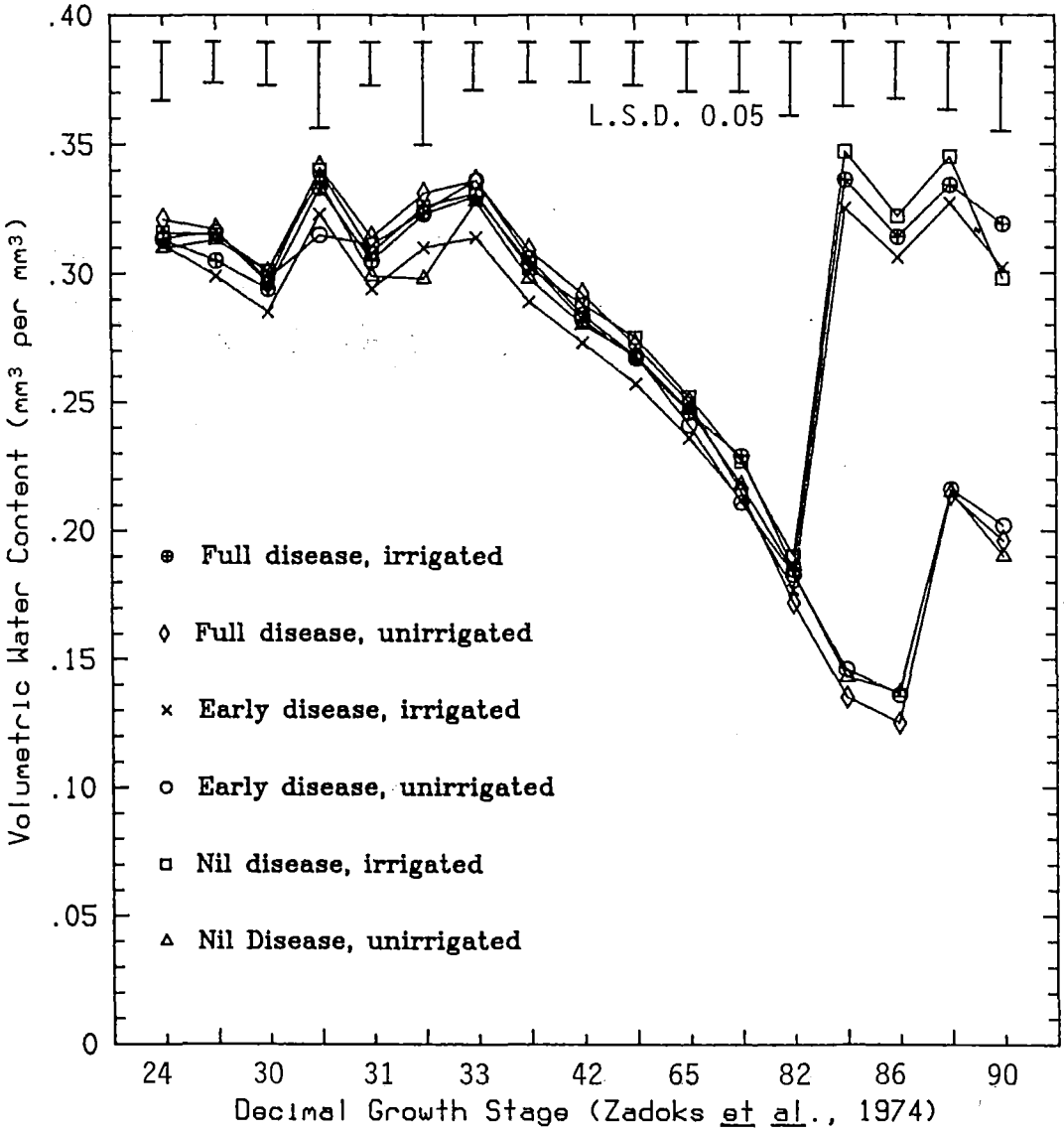


Figure 4.4: The effect of three different disease treatments and irrigation on water content in the soil profile (40 cm) in 1981–1982 field trial on wheat cv. Kopara.

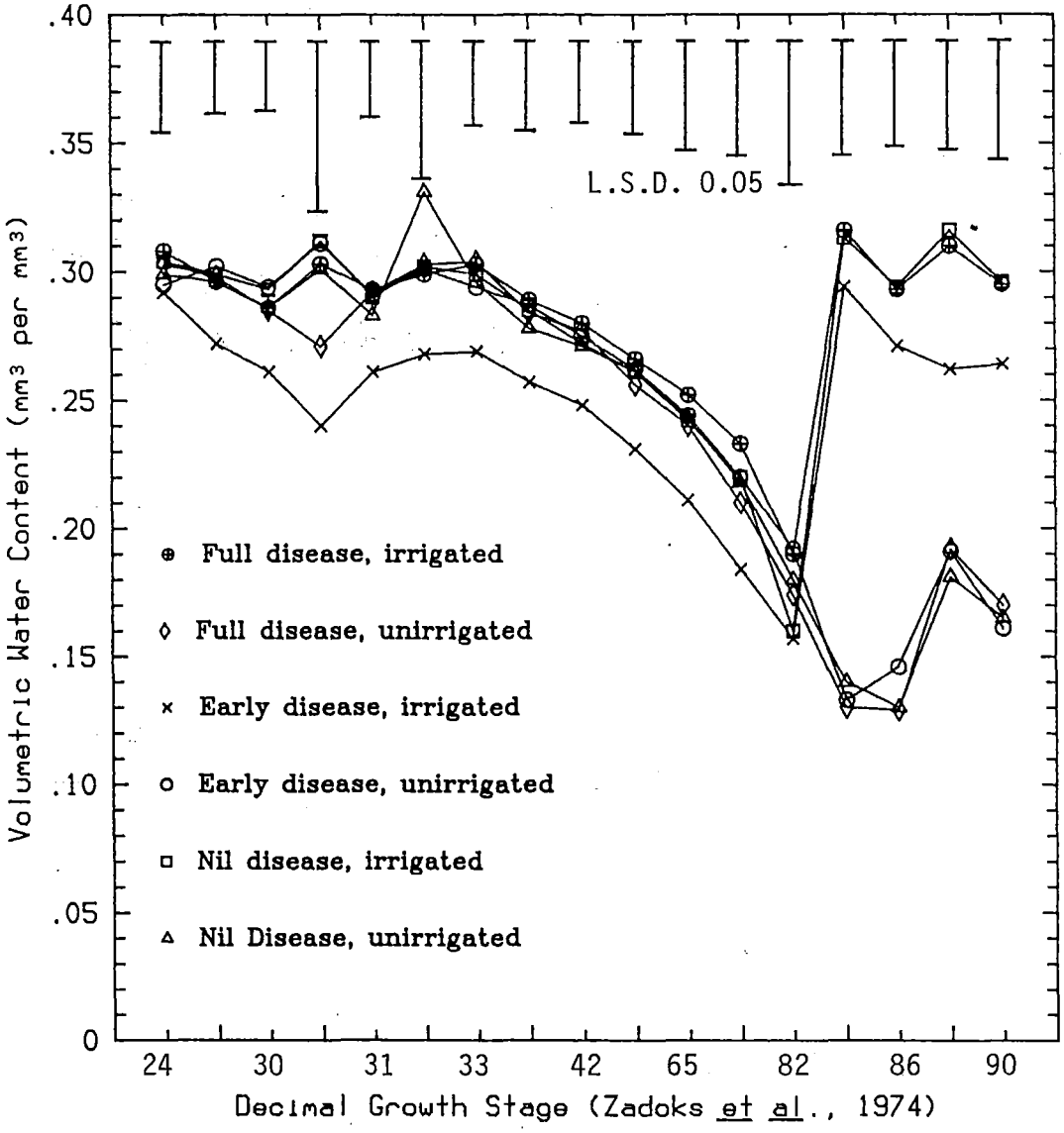


Figure 4.5: The effect of three different disease treatments and irrigation on water content in the soil profile (50 cm) in 1981–1982 field trial on wheat cv. Kopara.

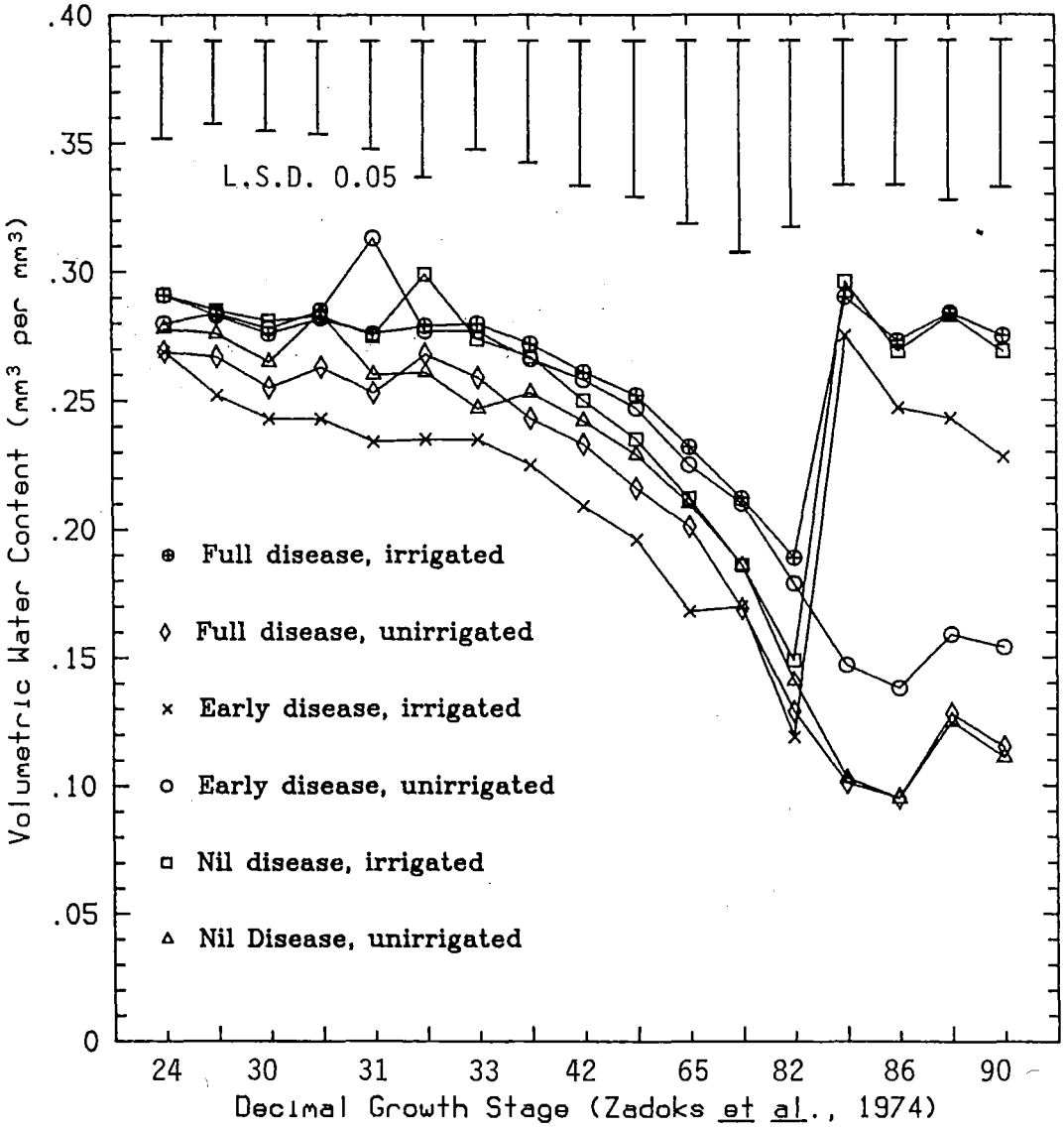
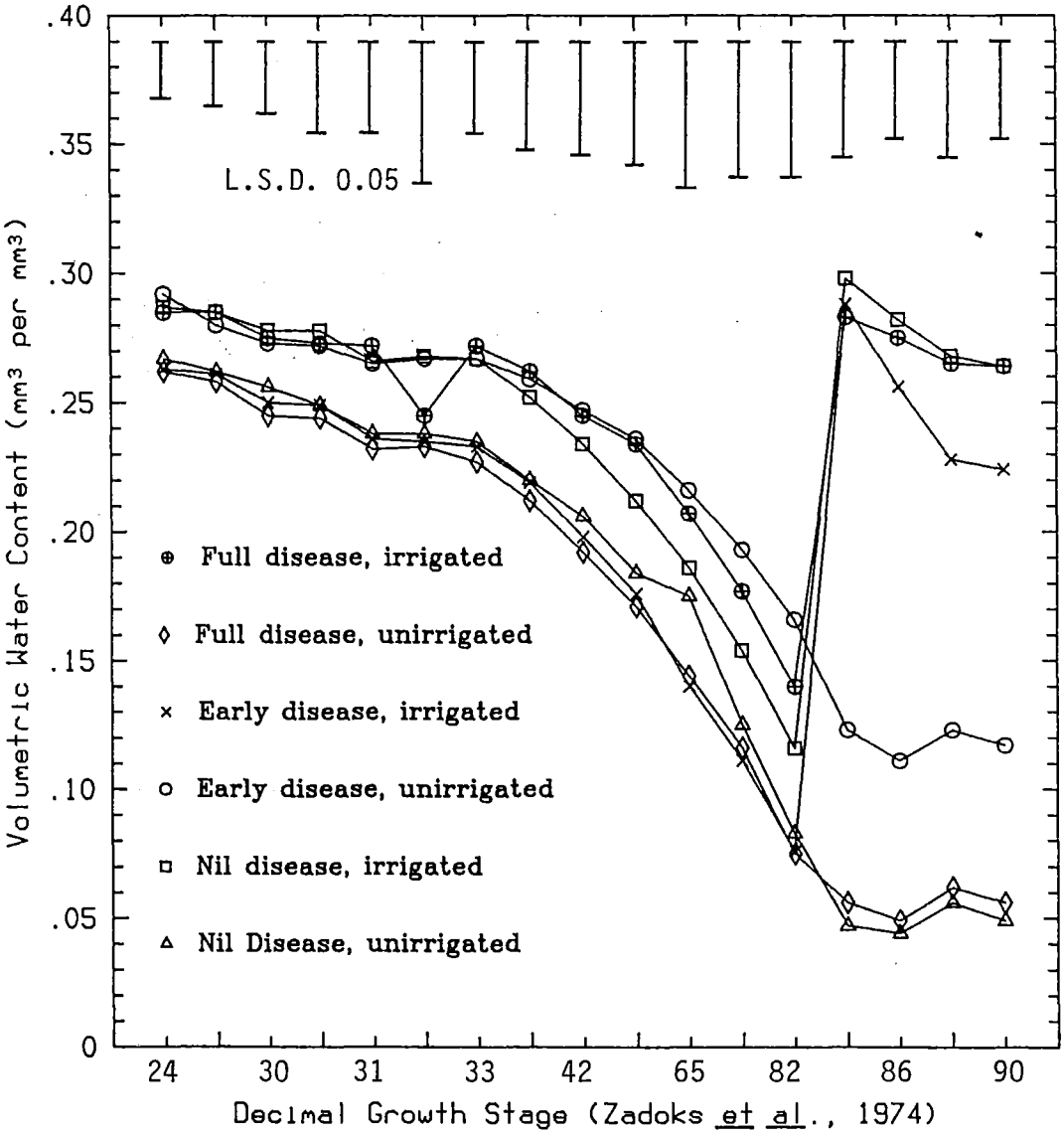


Figure 4.6: The effect of three different disease treatments and irrigation on water content in the soil profile (60 cm) in 1981–1982 field trial on wheat cv. Kopara.



unirrigated plants.

In all plots, the water content at 70 cm declined in a pattern similar to that at 60 cm. Differences in water extraction were observed among the unirrigated plots between G.S. 55 and G.S. 70. At G.S. 55 the early disease plots had a higher water content than the full disease plots. At G.S. 65 and G.S. 70 the nil disease plants used more water than the early disease plants (Figure 4.7). A similar pattern of water extraction to that at 60 cm was observed among the unirrigated plants between G.S. 85 and harvest.

At 80 cm depth, there was little change in the water content of all plots up to G.S. 38. Between G.S. 55 and 87 there was a rapid decline in water content (Figure 4.8). There were no apparent differences in water content between disease treatments at this depth, except for the early disease plots designated to be irrigated and the full disease unirrigated plots. Between G.S. 55 and 70, these plots had a higher water content than the full disease plots designated to be irrigated.

At a depth of 90 cm, there was no change in total water content in all plots up to G.S. 65. A rapid decline in water content was observed after G.S. 82 (Figure 4.9), suggesting that water extraction at this depth was greater at later stages of growth. There were no differences in water content between all treatments, except the early disease plots designated to be irrigated, which had a higher water content than the early disease unirrigated plots at G.S. 15 24, 16 30, 42, and 70. After irrigation, in contrast to the 60 cm and 70 cm depths, the early disease unirrigated plots had a lower water content than the nil and full disease unirrigated plots.

At 100 cm depth, total water content remained constant in all plots up to G.S. 70, but declined up to G.S. 85 (Figure 4.10). No differences in water content were observed between the disease treatments, except at G.S. 82, when the early disease unirrigated plots had a lower water content than the early and full disease plots designated to be irrigated. After irrigation, the early disease unirrigated plots had a lower water content than the nil and full disease unirrigated plots.

Figure 4.7: The effect of three different disease treatments and irrigation on water content in the soil profile (70 cm) in 1981–1982 field trial on wheat cv. Kopara.

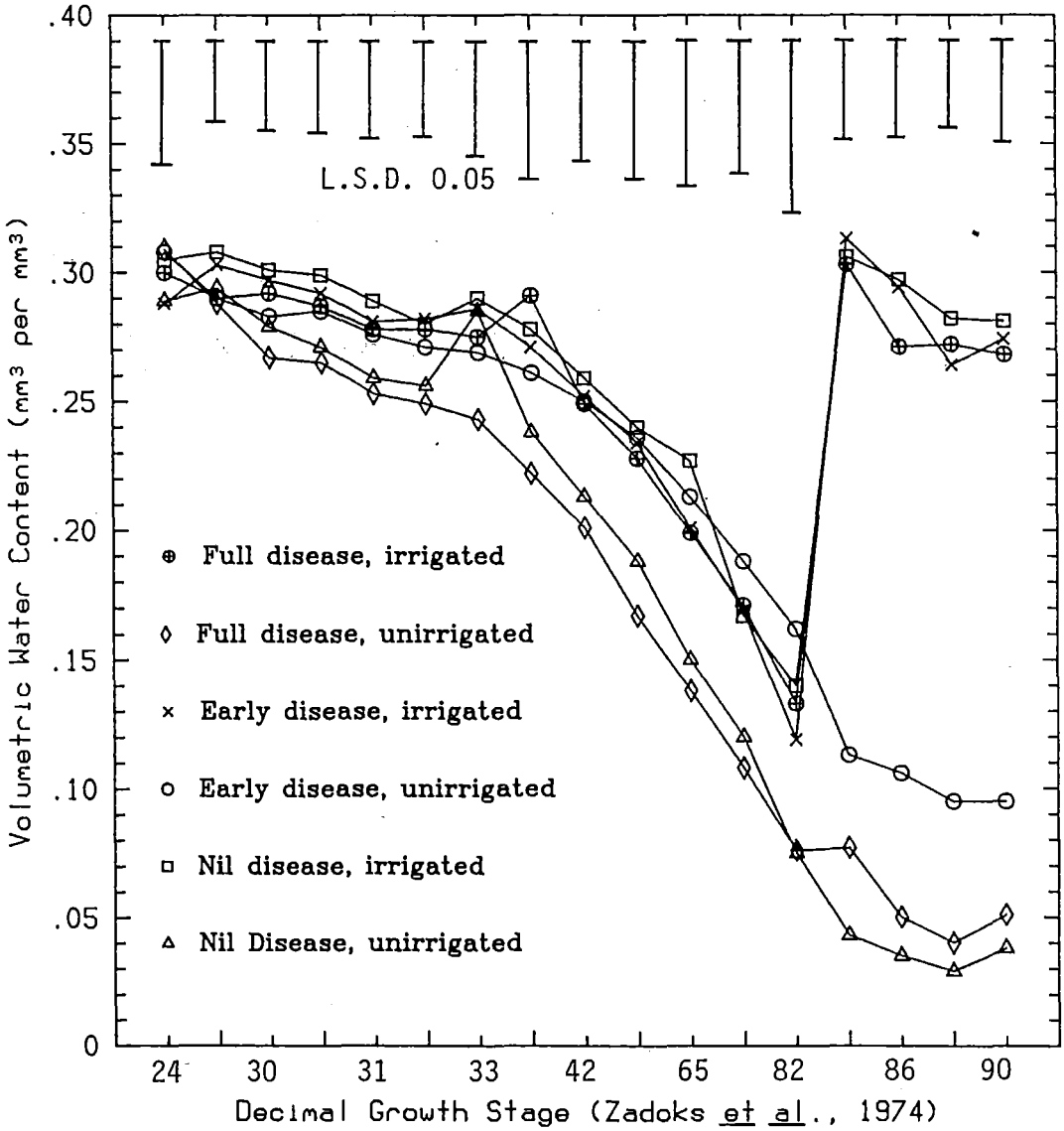


Figure 4.8: The effect of three different disease treatments and irrigation on water content in the soil profile (80 cm) in 1981–1982 field trial on wheat cv. Kopara.

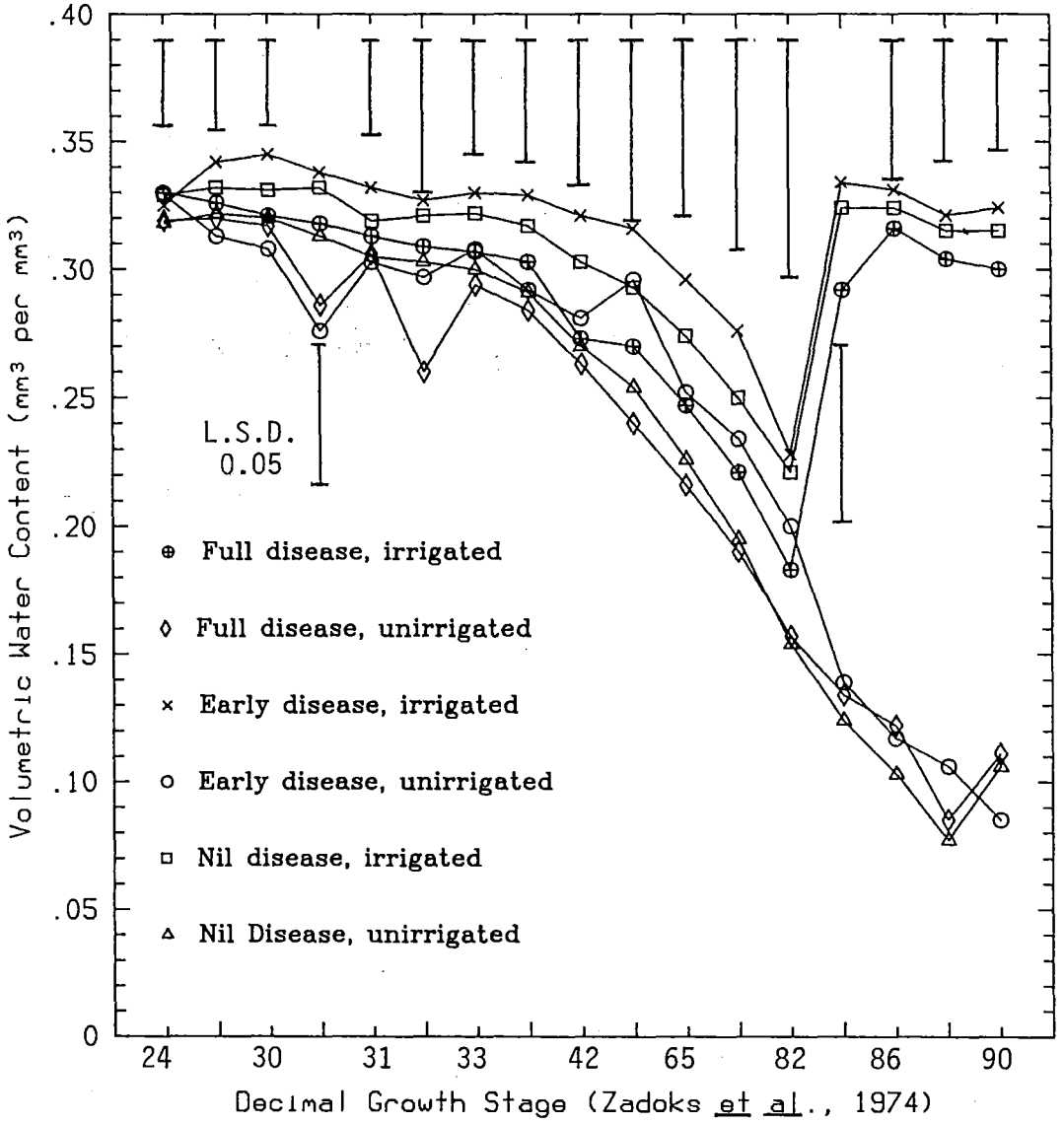


Figure 4.9: The effect of three different disease treatments and irrigation on water content in the soil profile (90 cm) in 1981–1982 field trial on wheat cv. Kopara.

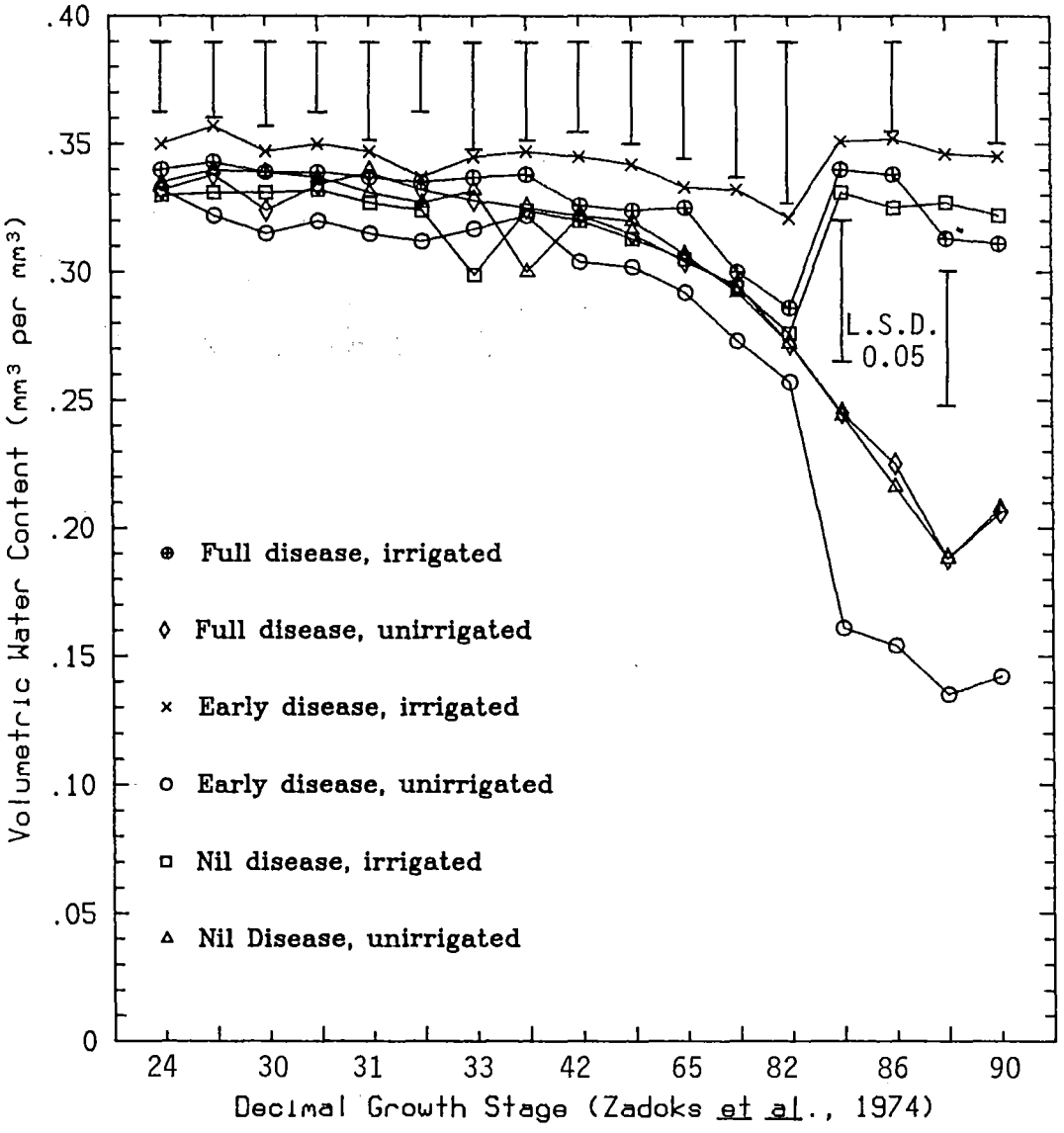
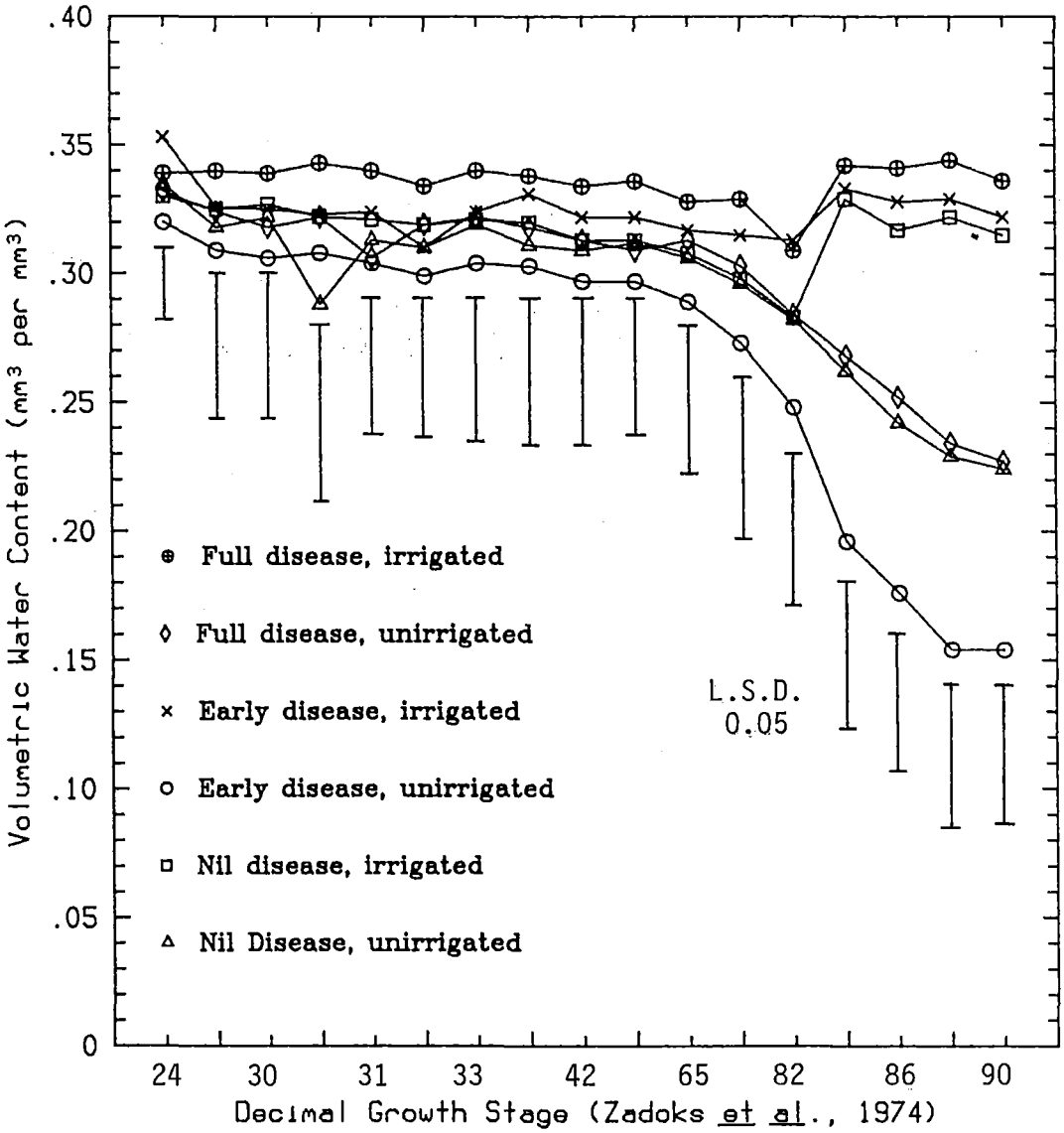


Figure 4.10: The effect of three different disease treatments and irrigation on water content in the soil profile (100 cm) in 1981–1982 field trial on wheat cv. Kopara.



In general, there was no consistent pattern in water extraction at all depths up to G.S. 82. After G.S. 82, the unirrigated plants extracted more water from depths below 80 cm, but this was not observed in the irrigated plants. However, after irrigation, the irrigated plants did extract more water from layers between 20 cm and 70 cm compared to unirrigated plants.

3.4.2 Water use

Water use was calculated by subtracting the total water content in one week from the total water content in the previous week, and by adding water input from rainfall and irrigation for that week. Water use (Table 4.9) generally increased with the growth of the plants and production of leaves, and also increased after replenishment of available water by rainfall (Table 4.9). The amount of water use in the individual layers of the soil profile may have been influenced by the volume of roots present in a particular layer.

Water use by the nil disease plants increased from G.S. 24 up to G.S. 85, except at G.S. 33, when there was a decline. Water use declined after G.S. 85, because of the loss of leaves (transpiring areas) in the maturing crop. Water use by the nil disease unirrigated plants was similar to the early disease unirrigated plants at all growth stages, except at G.S. 24, 16 30, and 85. At G.S. 24 and 85, the early disease unirrigated plants used 7.3 mm and 7.0 mm more water compared to the nil disease unirrigated plants, but at G.S. 16 30, there was a 5.4 mm reduction in water use. As there is no definite trend, it is difficult to explain the differences in water use. Water use by the full disease unirrigated plants was similar to the nil disease unirrigated plants at all growth stages, except at G.S. 16 30 and 85. At these two growth stages, plants in the former treatment used 6.2 mm and 7.3 mm less water compared to plants in the latter treatment. The nil disease unirrigated and irrigated plants had similar water use until irrigation was applied. After irrigation (at G.S. 83), water use by the irrigated plants increased compared to the unirrigated plants by 15.9 mm and 10.9 mm at G.S. 87 and 90 respectively. This indicates that the nil disease plants were able to use large amounts of water even

during the caryopsis development stage. It is not certain whether this amount of water was used entirely by the plants, or includes that lost by surface evaporation and/or drainage. A similar trend in water use by plants of the unirrigated and irrigated treatments was observed when compared within the early and the full disease treatments.

Cumulative water use was calculated for the crop growth period, and it was found that there were no differences among the unirrigated treatments. There was a 9% increase in cumulative water use by the nil disease irrigated plants compared to the nil disease unirrigated plants. In the early disease irrigated plants, the cumulative water use was 15% more than the corresponding unirrigated plants. A 12% increase in cumulative water use was observed in the full disease irrigated plants compared to the full disease unirrigated plants. The pattern of water use indicates that disease did not affect water use in this trial.

3.5 Water use efficiency

The water use efficiency of wheat cv. Kopara (amount of water used (mm) per unit of grain dry matter (kg ha^{-1}) produced) was determined. However, to determine water use efficiency, grain yield is not the most useful determinant of crop yield compared to total crop dry matter. Nonetheless, it provided an indication of water use in terms of economic yield. The unirrigated plants were found to have a greater (31%) water use efficiency compared to the irrigated plants (Table 4.10). (15%) Among the unirrigated plants, the nil disease plants had a higher efficiency of water use than the full disease plants.

3.6 The effects of disease and irrigation on yield and yield components

In the absence of disease and irrigation, the grain yield was 492 g m^{-2} (Table 4.11a). A potential ear number of 602 was reduced at harvest to 372 ears m^{-2}. There were 36 grains ear^{-1} and an individual grain weight of 36.7 mg. Compared to the nil disease unirrigated

Table 4.9: The effect of three different disease treatments and irrigation on actual water use during the growth period of wheat cv. Kopara in 1981-1982 field trial.

Sampling date	Growth stage[1]	Disease treatments						Rainfall (mm)
		Nil disease		Early disease		Full disease		
		UI[4]	I[5]	UI	I	UI	I	
24/9	24	3.6[6]	4.8	10.9	5.2	4.9	8.5	6.7
1/10	30	9.0	5.7	9.6	9.7	14.4	8.1	0.2
8/10	30	26.3	25.0	20.9	22.4	20.1	23.7	38.2
15/10	31	18.0	17.6	13.7	17.6	16.0	13.7	0.0
23/10	32	38.5	38.5	42.8	40.9	38.7	39.1	45.2
29/10	33	9.4	9.0	9.4	4.6	9.1	5.3	9.8
5/11	38	16.5	14.6	13.6	16.4	19.3	15.2	1.2
12/11	42	22.3	21.5	20.9	22.5	22.8	23.0	11.4
19/11	55	30.6	26.1	28.9	29.3	35.6	34.4	18.8
26/11	65	20.7	23.9	19.7	22.9	19.3	15.5	0.0
3/12	70	30.0	20.0	25.3	24.5	29.4	24.4	5.1
10/12	82	36.6	41.4	32.9	35.4	35.6	33.5	5.2
24/12	85	37.6	44.4	44.6	49.1	30.3	48.8	7.6
31/12	86	12.4	14.7	13.2	18.7	12.6	16.4	1.6
7/1	87	6.3	22.2	4.8	20.8	6.5	19.9	23.5
14/1	90	0.3	11.2	2.3	14.9	-0.8	9.0	0.5
22/1[7]	92	1.9	10.6	2.6	9.5	3.3	15.5	2.4
Cumulative water use		320.0	351.2	316.1	364.4	317.1	354.0	

(1) Decimal growth stage (Zadoks et al., 1974).

(2) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 23, 24, 30, 32, 37, 65, 85.

(3) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 32, 37, 65, 85.

(4) Unirrigated.

(5) Irrigation (230 mm) was applied at growth stage 83.

(6) Actual water use (mm) per week.

(7) Water use from 14/1 - 22/1 was estimated from the water content in the soil between 14/1 - 28/1.

Table 4.10: The effect of three different disease treatments and irrigation on water use efficiency of wheat cv. Kopara in 1981-1982 field trial, calculated with and without adjustments for drainage.

Disease treatments						LSD $P \leq 0.05$
Nil disease[1]		Early disease[2]		Full disease		
UI[3]	I[4]	UI	I	UI	I	
Water use efficiency not adjusted for drainage :						
15.41[5]	11.43	14.37	11.27	13.33	10.14	1.86
Water use efficiency adjusted for drainage :						
15.41	13.45	14.37	12.88	13.33	12.02	2.0

(1) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 23, 24, 30, 32, 37, 65, 85.

(2) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 32, 37, 65, 85.

(3) Unirrigated.

(4) Irrigation (230 mm) was applied at growth stage 83.

(5) Water use efficiency = grain yield (kg ha^{-1}) per mm water used.

plants, the yield of the early disease unirrigated plants was 7.4% lower, but this was not significant. A full disease epidemic in the unirrigated plants, however, significantly reduced yield (13.8%) compared to the nil disease unirrigated plants. The yield reduction in the full disease unirrigated plants was attributable to a reduction in grain number (Table 4.11a). There was no yield response to irrigation in the nil disease plants. Compared to the nil disease unirrigated plants, yield in the early disease irrigated plants was reduced by only 2.4%. This suggests that when irrigation is applied, the yield loss caused by early disease may be reduced. In the full disease irrigated plants, yield was 11.5% lower than the nil disease unirrigated plants, but was not significant on the basis of a test of least significant differences at the 5% level of significance. As the yield of the full disease unirrigated plants was significantly reduced compared to the nil disease unirrigated plants, this indicates that there was a positive response to irrigation by the full disease plants.

Although a large number of stems were produced, 602 m^{-2} in the nil disease unirrigated, and 566 m^{-2} in the irrigated plants, the potential ear number was reduced by 38.2% and 34.8% respectively at maturity. Disease reduced the total stem number m^{-2} in the early and full disease plants, which in turn contributed to a lower ear number m^{-2} at harvest, although fewer potential ears were lost compared to the nil disease plants. The full disease epidemic reduced the number of grains per spikelets in the first tiller compared to the nil and early disease unirrigated plants (Table 4.11b). There was also a reduction in the number of grain bearing spikelets in the second tiller of the full disease unirrigated plants compared to the nil disease irrigated plants. Disease affected the number of grains per spikelet on the main stem and tillers (Table 4.11b). Early and full disease reduced grains per spikelet on the main stem compared to the nil disease irrigated plants. On the first tiller of the full disease plants, there were less grains per spikelet compared to the nil disease plants. In the early disease plants, the grain number per spikelet of the first tiller was reduced when compared to the nil disease irrigated plants, but not with respect to the plants from other treatments.

Table 4.11a: The effects of three different disease treatments and irrigation on yield and yield components of wheat cv. Kopara in 1981-1982 field trial.

Variable	Disease treatments						LSD $P \leqslant 0.05$
	Nil disease[1]		Early disease[2]		Full disease		
	UI[3]	I[4]	UI	I	UI	I	
Quadrat yield ($gDM\ m^{-2}$)	491.7	483.1	455.0	479.7	423.7	435.4	67.9
Plant number m^{-2}	192	204	181	198	189	194	-
Total stem number m^{-2}	602	566	499	513	505	497	66
Ear number m^{-2}	372	368	341	363	352	354	47
Ear number $plant^{-1}$	1.94	1.81	1.89	1.84	1.87	1.82	0.43
Grain number ear^{-1}	36.0	35.6	36.5	35.4	33.8	34.2	1.9
Individual grain weight (mg)	36.7	36.8	36.4	37.3	35.5	36.1	1.6
Header yield ($t\ ha^{-1}$) at 14% moisture content	4.29	4.43	4.69	4.13	4.33	4.28	0.92

(1) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 23, 24, 30, 32, 37, 65, 85.

(2) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 32, 37, 65, 85.

(3) Unirrigated.

(4) Irrigation (230 mm) was applied at growth stage 83.

Table 4.11b: The effect of three different disease treatments and irrigation on number of spikelets per ear and number of grains per spikelet of wheat cv. Kopara in 1981-1982 field trial.

Variable	Disease treatments						LSD $P \leq 0.05$
	Nil disease[1]		Early disease[2]		Full disease		
	UI[3]	I[4]	UI	I	UI	I	
Mean number of spikelets per ear :							
Main stem :							
Filled	17.1	17.2	16.8	16.7	16.7	16.7	0.5
Unfilled	2.0	2.0	2.2	2.3	2.2	2.1	0.5
First tiller :							
Filled	14.6	15.7	14.6	14.3	12.7	13.4	1.6
Unfilled	2.8	2.6	2.8	3.0	2.6	2.7	0.4
Second tiller :							
Filled	9.0	10.1	7.0	6.9	5.6	6.0	3.3
Unfilled	1.7	2.0	1.7	1.5	1.2	1.3	0.6
Third tiller :							
Filled	1.0	2.1	1.3	0.9	0.8	0.3	1.4
Unfilled	0.2	0.3	0.3	0.2	0.2	0.1	0.3
Mean number of grains per spikelet :							
Main stem	2.6	2.7	2.5	2.5	2.5	2.5	0.1
First tiller	2.4	2.5	2.3	2.3	2.2	2.2	0.1
Second tiller	2.3	2.3	2.2	2.2	2.0	2.2	0.3
Third tiller	2.4	2.3	2.1	1.6	1.9	0.6	1.1

(1) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 23, 24, 30, 32, 37, 65, 85.

(2) Triadimefon (250g a.i. ha^{-1}) was applied at the following growth stages : 32, 37, 65, 85.

(3) Unirrigated.

(4) Irrigation (230 mm) was applied at growth stage 83.

The header yield at 14% moisture was lower than the quadrat yields (Table 4.11a). The difference in yield was attributed to losses during the harvesting operations. Header yields of the early and full disease plants did not differ significantly from that of the nil disease plants.

4 Discussion

Speckled leaf blotch severity was very low in 1981-1982 compared to the 1979-1980 season. This may be attributed to the small amount of primary inoculum from stubble from the 1980-1981 crops, in which disease severities were also low. Less than average rainfall meant that the season was not conducive to the development of speckled leaf blotch (the mean monthly rainfall from May 1980 to January 1981 was 34.1 mm compared to 55.5 mm for the long term average).

There was a severe infection by stripe rust in the trial plots. The source of primary inoculum was volunteer plants. This disease occurred for the first time in autumn sown wheat in Canterbury in 1980. The strain of the pathogen was reported to be 104 E137 (Harvey, 1981). In the trial plots, primary infection occurred on wheat grown from treated seed at early growth stages (G.S. 22). Initially, the stripe rust epidemic progressed slowly, possibly because of the length of time required to produce secondary inoculum through several monocycles. In spring, after the appearance of new leaves and rapid plant growth, the epidemic did not spread further up the plant, and although the disease spread to the upper leaves later in the season, the disease severity was less than on the lower leaves. The reduced severities in the upper leaves may be related either to adult plant resistance on Kopara wheat (Harvey, 1981), or unfavourable climatic conditions. High temperatures, long light duration and low humidity are not favourable to urediniospore germination (Rapilly, 1979) and these conditions may have been responsible for the decline in increase in infection.

Powdery mildew was present in small amounts during early crop growth. This may be related to the moderate resistance of the cultivar Kopara (Smith and Smith, 1970) to powdery mildew. It is also possible that the severity of stripe rust restricted the development of powdery mildew and speckled leaf blotch. Brown rust, which was present after anthesis on the upper three leaves of the main stem, was more severe on plants which were irrigated. This was associated with an increased green leaf area duration, which occurred in the irrigated treatments.

The effect of disease on plant growth was recorded at several growth stages as the root length per unit area, root length per unit volume of soil, and the total green leaf area present on the main stem. The maximum root length per unit surface area in the nil disease unirrigated plots before harvest (317.5 cm cm^{-2}) was greater than that for two winter wheats (187.5 and 183.4 cm cm^{-2}) and spring wheat (111.0 cm cm^{-2}) reported by Welbank et al. (1974).

Up to G.S. 80, root development was similar to that recorded for barley (Welbank and Williams, 1968; Welbank et al., 1974) and for wheat (Welbank et al., 1974; Gregory et al., 1978b). The length of roots was reported to be constant after anthesis (Welbank et al., 1974). Gregory et al. (1978b) found that although root growth continued after anthesis, root death was greater than before anthesis, resulting in no net gain in root weight. In this trial, however, there was a net increase in root length from G.S. 80 to harvest in the nil disease unirrigated plants. This increase was not observed in the nil disease irrigated plants, which had a root length per unit surface area similar to that observed by Welbank et al. (1974) for winter wheat. This indicates that soil water deficits during later growth stages may have necessitated further root growth to meet the water requirements of the plants. Increases in root length at later growth stages were greater in the 50-75 cm layer compared to the upper layers. Similar increases were observed at lower depths at later growth stages by Gregory et al. (1978b), although there was no net gain in the total weight of roots present in the entire profile. From water extraction data (Figures 4.8-4.10), it may be assumed that roots were present beyond 75 cm, and therefore, it is possible that root growth also occurred at these depths in later growth stages. The greater increase in roots at lower depths indicated that seminal roots are capable of growth at later stages of

the crop cycle. This increased root growth may have occurred through a reallocation of resources, as discussed later in this section (p.135).

Stripe rust on wheat plants grown in mist chambers reduced root growth, as observed by Martin and Hendrix (1974). The effect of disease was similar to the effect of shading on root growth (e.g., Welbank et al., 1974). These two effects may be related to a reduction in net photosynthesis. The reduction in net photosynthesis usually reduces the amount of photosynthate translocated to the roots (Doodson et al., 1964b). The reduced root development caused by disease in this trial was greater between G.S. 24 and 31 than at later growth stages. This may be because root growth is greater before the stem elongation phase compared to later growth stages (Gregory et al., 1978b).

After the alleviation of disease constraints at G.S. 32 in the early disease plants, there was a 26% increase in root length up to G.S. 80 (Table 4.6), but root length of the full disease plants did not increase over the same period. This indicated that plants were able to produce more roots after disease constraints were removed, to offset the previous reduction in root growth. After the alleviation of disease, root growth may depend on either stored reserves or current photosynthesis. Root growth will also depend on those parts of the plant from which the resources are able to be drawn, e.g., the main stem and tillers. Gallagher et al. (1975, 1976a) and Bidinger et al. (1977) reported that some photosynthates required for grain filling were translocated from stored stem reserves. It is possible that photosynthates required for root growth after the alleviation of disease may also be translocated from stem reserves, or current photosynthates may be used. The utilisation of such resources for root growth may limit the amount of photosynthate available for further root growth or grain filling at later growth stages, as observed in the early disease unirrigated plants. Although these plants produced more roots compared to the full disease plants after disease was controlled at later growth stages, the increase in root growth did not result in a root system equal to that of the nil disease unirrigated plants.

The effect of disease on leaf growth was recorded throughout crop growth as the total green leaf area present on the main stem. Disease reduced the total green leaf area of the main stem by affecting both the

percent green leaf area (Table 4.4) and total leaf area (Table 4.5). The reduction in leaf growth may be associated with the reduced movement of photosynthates to developing leaves as a result of stripe rust infection (Doodson et al., 1964b), which delays the rate of development and ultimate leaf size (Doodson, 1976). The effect of disease in the early disease plants continued after the epidemic had declined, and was expressed through reduced leaf expansion and final leaf size. This may be attributed to a reduction in effective green leaf area of emerged leaves, contributing less photosynthate compared to the nil disease plants. An additional factor may be that a greater proportion of current photosynthates were partitioned to the roots after disease constraints were alleviated. This increased root growth may have competed with the shoots for photosynthates. By anthesis, however, total green leaf area of the early disease plants was similar to the nil disease plants. The increased root growth in the early disease plants after alleviation of disease constraints may have enabled the plants to obtain more water and nutrients required for increased shoot growth within predetermined limits, at later growth stages, compared to the full disease plants.

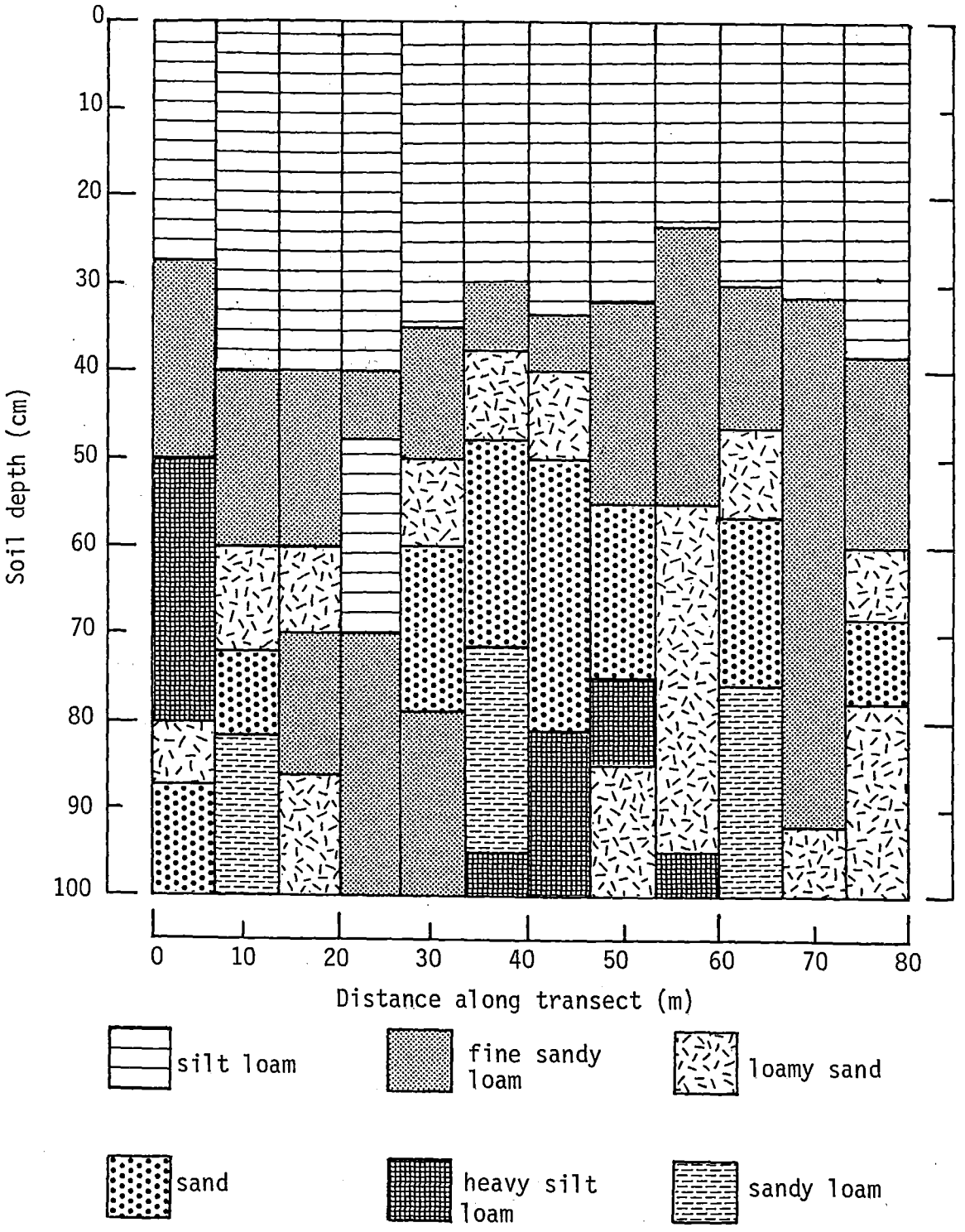
Soil water use at different depths in the profile indicates the effective rooting depth of the crop. However, in winter wheat, the actual rooting depth may be greater than the effective rooting depth, as roots may be found down to 200 cm (Gregory et al., 1978b). The change in water content at a given depth does not necessarily indicate the activity of roots, as it is possible for water to move upwards with changes in hydraulic potential (Gregory et al., 1978a). It is also possible for roots to be present in a soil layer without extracting water. Soil water measurements were determined to a depth of 100 cm, and the results indicated (Figures 4.1-4.10) that water was extracted at all depths. In this trial roots were not measured beyond 75 cm as the presence of shingle and sand made core extraction difficult. Although root measurements were not made beyond 75 cm, it is evident from the pattern of water extraction, and from studies by Weaver (1926), Sallans (1942), and Gregory et al., (1978b) that roots were probably present at 100 cm and possibly deeper. There was a lower soil water content in the 0-30 cm layer throughout the season compared to deeper layers. This is indicative of greater soil water extraction in the 0-30 cm layer of the

soil profile, and is related to the greater volume of roots present in this layer. When surface layers were depleted of soil moisture, however, there was greater extraction from lower depths. Similar results were obtained by Gregory et al., (1978a), who found that when water was available in the upper layers of the soil, plants preferentially extracted water from these regions, whereas they extracted water from lower depths when the upper layers were depleted. Extraction from the lower depths was greater after anthesis, when surface layers had a low water content. This indicated that seminal roots played an important role in extraction of water, as observed by Sallans (1942).

Differences in soil water content between treatments were observed at all depths below 20 cm. These differences were caused by water extraction and/or influenced by soil texture. In the 30-50 cm depths, the differences were observed before G.S. 55, while at the lower depths (70-100 cm) the differences were generally observed after G.S. 55. The differences from 30-50 cm were present at G.S. 24, before differences in disease severity were observed. These differences were not consistent, as variations were found in sub-treatments (unirrigated plots and plots designated to be irrigated) within a main (disease) treatment. Therefore, the differences in water content were not related to disease treatments. The inconsistent differences in water content may by attributed to varying soil textures. These ranged from coarse (sandy loam) to fine (silt loam). From 0-30 cm, soil textures were silt loams, but beyond 30 cm the textural layers varied, and were also undulating with different textures found within a 10 cm layer of soil (Figure 4.11). As the water holding capacity of these textures vary widely, it is possible that any differences in soil water content would be attributed to texture. In future experiments where soil texture varies over short distances, it may be necessary to increase the number of access tubes per plot to reduce intra-plot variation.

The amount of evapotranspired water was calculated from the rainfall and water content of the soil, and presented as water use (Table 4.9). In this trial disease did not affect water use, contrary to the findings that disease reduced evapotranspiration because of stomatal closure (Misaghi, 1982). However, this effect would depend on

Figure 4.11: Textural variability within the soil profile in a transect of replicate four in 1981-1982 field trial on wheat cv. Kopara.



the type of pathogen. Although rusts initially reduce evapotranspiration through stomatal closure, this may subsequently be offset by water loss through pustules which burst through the leaf epidermis (Duniway and Durbin, 1971a). The loss of water through the pustules may account for the lack of differences in water use among the nil disease and the disease plants, despite the differences in green leaf area. Water use alone, however, does not indicate how efficiently water is used by crops. This is better expressed by water use efficiency. The water use efficency of wheat (expressed as kg grain per hectare per mm evapotranspiration) was low in this trial compared to wheat and barley in other trials (e.g., Singh and Kumar, 1981). Soil water content in this trial was determined only to 100 cm depth. Wheat plants may have an effective rooting depth (i.e., the maximum depth at which water extraction occurs) greater than 100 cm (Weaver, 1926), and thus more water may have been extracted than was measured. For a more accurate estimate of water use efficiency, soil water contents should be determined down to the effective rooting depth. McGowan (1974) defined the effective rooting depth as the maximum depth at which the hydraulic potential gradient is zero. In this trial, the effective rooting depth is referred to as the maximum depth at which water was extracted. Neutron measurements could not be taken below 100 cm, as a shingle layer below that depth prevented the installation of longer access tubes.

There was a reduction in water use efficiency of irrigated plants (24%) compared to unirrigated plants (Table 4.10). This may be attributed to the application of irrigation very late in crop growth (during the grain filling stage). Before irrigation commenced, there was an average of 162 mm of water in the plots designated to be irrigated. The estimated field capacity of the trial plots was 331 mm to a depth of 100 cm (Reid, pers. comm.). Two hundred and thirty millimetres of irrigation was applied. Therefore, drainage may have occurred, as the total water content exceeded field capacity by 61 mm (230 + 162 - 331). Thus, water use efficiency of the irrigated plants was reduced in comparison to unirrigated plants because of water loss through drainage. No significant differences between disease treatments in the irrigated plants were observed, but in the unirrigated plants, water use efficiency was significantly reduced by disease. Drainage may have produced anomalies in the calculated values for water use

efficiency, as the amount of water lost by drainage was not taken into account. When drainage was accounted for, water use efficiency of the full disease unirrigated plants was reduced (14%) compared to the nil disease unirrigated plants (Table 4.10), as observed when water use efficiency was calculated on the total amount of water applied. The water use efficiency of the nil disease irrigated plants was similar to the nil disease unirrigated plants. However, the water use efficiency of the early and full disease irrigated plants was lower than that of the nil disease unirrigated plants.

A disease severity index for the entire season was determined as the mean disease severity over all samplings. A total green leaf area index was determined in the same way. The disease severity index derived in this way showed a poor regression with water use efficiency (R-squared = 8.1%). A poor, but better relationship than that with disease severity was obtained between total green leaf area index and water use efficiency (R-squared = 15.9%). The poor relationship obtained with the disease severity index may be attributed to the large range in disease severities at different growth stages. An overall mean would mask the effects of disease at individual growth stages, as the crop may be more sensitive to disease at certain growth stages than others (Lim, 1982; Gaunt and Thomson, 1985). In a similar manner, growth and development are influenced by the amount of green leaf area at different growth stages (Lim, 1982). Therefore, an overall mean would not represent the effect of disease or total green leaf area on water use efficiency at any given growth stage. A stepwise regression analysis of percentage disease severity and total green leaf area with water use efficiency was carried out to determine whether any particular growth stage was closely related to water use efficiency. Significant 'F' values were obtained for regressions between percentage disease severity and water use efficiency at G.S. 75 and 85, but at all other growth stages there were no significant 'F' values. The significant regressions for G.S. 75 (milk stage) and 85 (dough stage), had R-squared values of 56.9% and 44.7% respectively. The R-squared values at all other growth stages ranged from 0.2% to 31.6%. For the stepwise regressions between total green leaf area and water use efficiency, significant 'F' values were obtained at G.S. 32 and 37, with R-squared values of 36% and 34.6% respectively. At all other growth stages, the

'F' values were non-significant, the R-squared values ranging from 0.2% to 13.0%. The growth stages at which significant regressions were obtained were stem elongation (during which spikelet growth and development occur) and grain filling. Therefore, it is possible that water use efficiency (which is influenced by both yield and water use) may have been affected by grain number and grain weight, or water use during these growth stages. Grain number was reduced by a full disease epidemic in the unirrigated plants (Table 4.11a), indicating that the relationship shown at G.S. 32 and 37 was the result of an effect on grain number. The grain weight, however, was not affected by the disease treatments (Table 4.11a). Water use efficiency may have been influenced by water use at G.S. 85 and 75, as the nil and early disease unirrigated plants used 24.1% and 47.2% respectively more water at G.S. 85 compared to the full disease unirrigated plants. A similar pattern may have occurred at G.S. 75, but soil measurements were not taken at this growth stage. It may be concluded that disease affects water use efficiency through complex physiological interactions. When early disease constraints were alleviated, water use efficiency was increased to a level similar to that of the nil disease plants.

One of the main objectives of this research was to determine the effect of disease on root growth and its subsequent effect on yield and yield components, and to explain the lack of compensation for yield reductions caused by early disease constraints. This involved the manipulation of the disease epidemic to create full, early and nil disease epidemics, and their interaction with irrigation. Although the disease complex consisted of stripe rust, speckled leaf blotch, powdery mildew and brown rust, the major disease was stripe rust. A full disease epidemic without irrigation reduced yield by 13%. With irrigation, the yield of the full disease plants was not reduced. This indicates that there was a significant disease/irrigation interaction which may be explained by the effect of disease on root growth. In the full disease irrigated plants, root lengths between G.S. 80 and 92 were similar, indicating that irrigation may have reduced the need for further increases in root length. On the other hand, in the full disease unirrigated plants, root lengths increased significantly (44%) between G.S. 80 and 92 (Table 4.6). This suggests that the lack of soil moisture may have induced further increases in root length. The

increase in root length at later growth stages may have occurred through a reallocation of photosynthates, which may otherwise have contributed towards grain yield. It is concluded that a full disease epidemic and prolonged soil water deficits during grain filling reduced yield through a reduction in photosynthates available for grain filling. While an early disease epidemic reduced yield, this was to a lesser extent than a full disease epidemic, and the yields of the early disease plants were not significantly different to either the nil or full disease plants. This suggests that one of two possible mechanisms operated. First, the plants that were constrained by early disease were able to compensate at later growth stages, as observed by Gaunt and Thomson (1985), and contrary to the findings of Lim (1982) and Lim and Gaunt (1985). Second, the nil disease plants were not able to reach their potential yield because of physiological limitations caused by stresses such as water deficits. While there was no response to irrigation in the nil disease plants, it may be that it was applied too late in the season for any notable responses to occur. The soil was below field capacity at all growth stages until irrigation was applied, thus water deficits did occur during crop growth.

Grain number was the only yield component affected by disease in this trial. In a similar trial with Kopara wheat in Canterbury, Chan and Gaunt (1982) reported that grain number was reduced by a full disease epidemic of stripe rust. However, in Southland, Risk and Beresford (1982) observed no reduction in grain number in Kopara wheat, but grain number in cultivar Takahe was reduced by disease. In the Northern Hemisphere, stripe rust epidemics occur late in the growth season, and therefore do not affect the early determined yield components. However, glasshouse studies demonstrated that stripe rust infections in early plant growth can reduce the final number of tillers per plant, and the grain number per ear, and late infection reduces grain weight (Doodson et al., 1964a). Infection during the early growth stages caused by speckled leaf blotch on wheat (Gaunt and Thomson, 1985) and powdery mildew on barley (Lim, 1982) reduced grain number, in relation to other diseases in the field. The timing and severity of disease is important (Lim, 1982; Gaunt and Thomson, 1985), as this reflects the different yield responses of the plant which depend on the constraints to developing yield components. The effect of disease on

the number of spikelets per ear and grain number per spikelet were determined at harvest, and both these yield components were reduced by disease. The number of spikelets per ear was reduced on the tillers, but not on the main stem. The grain number per spikelet, however, was reduced in the main stem and first tiller. As apical development was not studied in this trial, it is not certain whether disease affected the number of spikelets produced. From previous studies on wheat (Gaunt and Thomson, 1985) and barley (Lim, 1982), it may be suggested that disease affects spikelet development rather than spikelet primordium production, resulting in a lower number of spikelets and florets (e.g., Lim, 1982; Gaunt and Thomson, 1985). Reduction in spikelet number may also be linked to the effects of reduced root growth, which reduces the absorptive capacity of the roots, and in soils with water deficits, causes a reduction in photosynthesis through stomatal closure (Ayres and Zadoks, 1979). Furthermore, roots are sites of synthesis of growth regulators such as cytokinins, which regulate photosynthetic activity (Michael and Seiler-Kelbitsch, 1972) and cell division (Skoog et al., 1967; Leopold and Kriedemann, 1975). Disease, through reduced root growth and/or through interference with the process of plant growth regulator production, may reduce the activity of plant growth regulators, and therefore affect the meristematic apical region, thus reducing spikelet development. The reduction in grain number per spikelet may be caused by various contributing factors, e.g., reduction in floret production or abortion, reduction in floret fertility, and/or grain abortion. As the ear was not analysed in detail during the growing period, the exact reason is not known.

The total stem number (main stem and tillers) was reduced by disease. As this total count included productive as well as non-productive tillers, it is assumed that disease affected the development of tiller buds that were initiated. This is possible through disease acting as an alternative sink, thus reducing the amount of photosynthate available to the meristematic tiller buds for growth and differentiation. The total number of grain bearing ears per unit area was similar in all treatments at harvest. This indicates that although a greater number of tillers were produced by the nil disease plants, not all tillers produced grain bearing ears. A similar trend was observed by Day et al. (1978) in barley affected by water deficits.

Therefore, it is concluded that the effects of disease and of water deficits on cereal growth were similar. The reduction in productive ear number in the nil disease plants may be associated with root growth. Soil water deficits may be considered the causal factor in increased root growth in the nil disease unirrigated plants after anthesis. At this growth stage, the lower leaves have senesced and some of the photosynthate required for grain filling is supplied by the flag and penultimate leaves. Therefore, it is possible that the resources required for root growth may be supplied by the secondary tillers of the plant. Day et al. (1978) reported that stem straw weights were reduced in barley plants which were water stressed during the early stages of growth compared to non-stressed plants. This reduction may have been caused by remobilisation of stored photosynthate to roots to alleviate water stress by enabling the roots to reach lower depths where stored water may be found. This assumption, however, needs to be verified experimentally. It is concluded that increased root growth may occur at the expense of tillers when soil water deficits occur at the grain filling stage.

Grain weight was not affected by disease in this trial, contrary to studies reported by other workers on wheat (Doodson et al., 1964a; Gaunt et al., 1982) and barley (Melville et al., 1976; Teng, 1978; Lim, 1982). In the nil disease plants, constraints to grain size may have been caused by water stress (e.g., Brocklehurst et al., 1978), therefore the plants may not have been able to realise their full potential for grain weight. On the other hand, as disease severities were low during the grain filling stage, grain weight may not have been affected significantly. Furthermore, as soil water deficits were similar in all treatments during the early grain filling stages, the reduced amount of roots caused by disease may not have affected grain weight. There was no significant response to irrigation in any of the treatments. As irrigation was applied at the mid-milk stage of grain filling, it may have been too late to significantly influence grain filling.

CHAPTER FIVE

CONCLUSIONS

1 The importance of studies on the effects of foliar diseases on shoot and root development

Studies of diseases and their effects on crop growth have led to an increased knowledge of pathogen, host and environment interactions. Nearly all field studies of foliar disease-yield loss relationships have dealt with above ground parts of the crop, and neglected the effects on roots. Roots are an integral part of a plant, and the growth of either root or shoot is dependent on the activites of the other. There is a complex interrelationship between shoot and root systems and no analysis of growth is complete unless the relationship is considered.

To study the effect of disease on plant growth, fungicide treated healthy plants are often grown and compared with non-treated diseased plants. Fungicides may cause phytotonic effects (Griffiths and Scott, 1977; Peat and Shipp, 1981) so that yield is increased in the absence of disease. In this study benomyl and triadimefon had no phytotonic effects on shoot, root growth, or yield under the prevailing conditions (Chapter 2).

The effect of disease on total green leaf area was demonstrated by Lim (1982) and Gaunt and Thomson (1985). Lim (1982) suggested that there were causal links between green leaf area at several growth stages and yield components, and that green leaf area was an important variable in yield loss studies. This study indicated that root growth and water use are also important variables to investigate.

2 Disease and its effect on root development

The field trials described here were carried out under specific agroclimatic conditions, and the results may not be reproduced under different conditions. However, the interactions described provide a basis for general guidelines for further research. A full disease epidemic in the field affected root development to a greater extent than early and nil disease (Chapter 4). On alleviation of disease constraints in the early disease treatment, root development increased to offset earlier reductions. Disease on plants after growth stage 33 (in the late disease treatments) also reduced root development (Chapter 3), indicating that disease may affect root development at all growth stages. With alleviation of disease constraints at any stage of growth, the plants may have the capacity to allocate resources for root development. However, such allocations may cause constraints to growth and development in other plant parts.

3 The effect of disease on water use

Water use by plants is dependent on the amount of water available and the atmospheric demand (evapotranspiration). Water use increased after irrigation, indicating that plants were capable of greater water use even during the later stages of crop growth (between G.S. 83 and 92). In soils where water was limiting in the upper layers at later growth stages, the roots at lower depths played an important role in extracting water. Thus, small amounts of roots at lower depths may contribute significantly towards water uptake. Based on changes in soil water content, disease had no effect on water use, even though the transpiring surfaces (leaf area) of the diseased plants were reduced. Increased water use per unit of leaf area in the diseased plants may be related to water loss through pustules and possibly stomata. Although most water relation studies on diseased plants indicated reduced water loss through stomata, those studies were conducted in pots (e.g., Duniway and Durbin, 1971b). It is possible that, in the field where roots are able to explore a greater volume of soil, stomatal resistance may be less and water may be lost at the same rate as that for healthy

plants.

4 Relationship between root development and yield

A full disease epidemic without irrigation caused significant yield loss through reductions in grain number. The reduction in grain number may be related to the effect of disease on total green leaf area (e.g., Lim, 1982) and root development. Reduced root development may have affected the ability of the roots to supply the shoot with water, nutrients and plant growth regulators. These constraints may have a greater effect on yield in situations where prolonged soil water deficits occur. Increased root growth during the grain filling stages in the full disease unirrigated plants, because of soil water deficits, through utilisation of current and/or stored photosynthates, may have occurred at the expense of the grains. The presence of adequate soil water in the irrigated plots reduced the need for further root growth and grain weight was not reduced. When disease constraints were alleviated, as in the early disease plants, increased amounts of photosynthate may be used for root and shoot development to offset earlier reductions. This may reduce the amount of photosynthate available for the developing apex, thus prolonging the effect of early disease. In this study (Chapter 4) the supply of water at later stages of growth increased grain weight, suggesting that reduced root development caused by early disease and soil water deficits may have prevented adequate compensation by grain weight. Reduced root development and its subsequent effect on plant growth may, therefore, be minimised by providing adequate soil moisture.

5 The effect of disease and irrigation on yield

In the 1981-1982 trial the yield of the nil disease plants was not increased by irrigation at G.S. 83. Therefore, the growth and development of the nil disease plants was not constrained by water at the late grain filling stage. The yield of the full disease unirrigated plants was, however, reduced. The full disease plants responded to irrigation and the yield was not significantly different to the nil

disease plants. Therefore, the full disease plants were water constrained during the grain filling stage. This water constraint may have been caused by a combination of a reduced root system and soil water deficits. The yield of the early disease plants was between that of the nil and full disease plants. Irrigation of the early disease plants reduced the effect of disease on yield. The response to irrigation indicates that the early disease plants may have been water constrained. Although there were soil water deficits in the early disease unirrigated plots during grain filling, the increased root development after alleviation of disease constraints may have enabled the deeper roots to take up stored soil water to alleviate possible plant water stresses.

The hypothesis that early disease reduces root development and causes the plants to be water constrained at later stages (after anthesis) was tested. In these studies, disease reduced root development in the field. The reduction in roots may prevent the plant developed root system from a given volume of soil. from extracting water as efficiently as a well ^ The plants are, therefore, water constrained during the later growth stages when soil water deficits occur. These studies have partially proved the hypothesis that the lack of compensation for the effects of early disease (e.g., Lim, 1982) by later determined components is caused by water constraints.

6 Disease management

The results reported here may provide guidelines for the economic application of fungicides and/or water to wheat production. Fungicides and pesticides are important constituents of the green revolution. They are mainly derived from depleting and non-renewable petroleum products (Morrison and Boyd, 1973). The unpredictable and often escalating nature of fossil fuel prices create problems for countries whose long term development programmes are based on agriculture and crop productivity. Environmentalists oppose the use of agrochemicals because of the impact on the environment, fauna and flora. The cost and environmental impact factors point to the importance of planned and sensible use of agrochemicals. Water, which is one of the basic

requirements for plant growth, is a limited and expensive resource in certain agricultural areas of the world (e.g., semi-arid tropics, California, Israel, and Australia). In Canterbury, New Zealand, the amount of rainfall during the summer months (mean 50mm ± 16mm per month) is similar to the winter months (mean 67mm ± 18mm per month), but there is a high evaporative demand in summer (November to January). Most cereal cropping systems in Canterbury depend solely on rainfall, thus crops experience severe water deficits in most years during summer.

There was a disease:irrigation interaction in the 1981-1982 trial (Chapter 4). The interaction is best expressed in terms of water use efficiency, as it indicates the efficiency of yield production. The investigation of disease and water use provides information on the rational use of fungicides and/or water. In areas where water is a limiting resource, crop diseases may be controlled by the efficient use of fungicides. However, in areas where irrigation is available, the effect of foliar diseases may be counteracted by a minimum of one or two applications of fungicides at the early growth stages; and the effects of late disease on root development at later growth stages may be counteracted by the application of water. This would reduce the effect of reduced root development at later growth stages, and increase the water use efficiency of the crop. In this context, the efficient use of fungicides and water would ensure optimum production levels in agriculture. However, it should not be assumed that the combined use of fungicides and irrigation would give an economic yield return.

7 The need for a multidisciplinary approach in disease-yield loss studies

In the field of plant pathology, the effect of disease on yield loss cannot be related solely to the plant organ affected, as pathogens affect not only the infected organ, but all other parts of the plant, through complex physiological processes. Therefore, to understand disease-yield loss relationships it is not sufficient to relate the amount of disease present on plants to the amount of yield lost. It is important to explain the intricate relationship between the amount of disease present and the resulting yield loss of a particular crop through physiological processes. In this study, although disease did

not affect total water use, disease did determine how efficiently the water was used. Therefore, water use efficiency is an important factor in disease-yield loss relationships. As water use efficiency of diseased plants may be altered through complex physiological processes, it is not certain how this occurs. There is a need for a multidiscplinary approach in understanding how disease affects water use efficiency. Such investigations may provide the basis for more precise crop, disease and water management, and the optimal use of agrochemicals in cropping systems. Information on the effect of disease constraint factors on water use may ultimately be used in whole crop models to understand, predict and control disease-yield losses.

The continued effects of early disease constraints, even after the control of disease, prevented compensation by later determined components (Lim, 1982). In these studies, it has been shown that one of the reasons for this lack of compensation was caused by water constraints. Disease can also influence the water use efficiency of the crop. The disease-irrigation interaction suggests that irrigation can be used as a tool to study the effects of disease on crops for a better understanding of how disease influences yield.

8 Guidelines for further research on disease-yield loss relationships

There is a need for studies on disease-yield loss relationships to include the entire plant, i.e., both shoots and roots. There are many unexplained functions of the roots in relation to changes in the shoot system caused by disease. However, root studies in the field are labour intensive and time-consuming, and new methods of measuring roots must be developed, e.g., the use of computers and video images as a tool for rapid measurement. It is difficult to study the entire root system of a plant in the field, as roots occupy large areas of the soil both vertically and horizontally. However, representative core samples may be taken, as used in this study. Where intra-plot differences occur, many core samples must be taken to reduce variation within plots. Although there may be only a few roots at lower depths in the soil profile, it is evident from this study that they play an important role in water extraction when the upper soil layers are depleted of water.

Therefore, it is important to determine the actual rooting depth of the plants in root studies, and determine the contribution by these roots towards plant productivity.

From these studies, it is suggested that plants have the ability to reallocate resources to roots at any stage of growth to offset any earlier reductions in root growth. The root development at later growth stages occurs by the remobilisation of current or stored photosynthates. In the 1981-1982 trial, many potentially productive tillers ceased to develop further towards the later stages of growth in the nil disease plants. It is possible that root growth at later growth stages utilised the resources from these tillers. In the early disease plants, the continued effects of disease after alleviation of disease constraints may be caused by remobilisation of photosynthates. Studies on remobilisation of photosynthates and dry matter changes may provide information on the amount and source of photosynthates supplied to the roots by the different parts of the shoot. In disease-root development studies, it is important to sample roots at frequent intervals throughout crop growth to closely monitor root development in relation to changes in the shoot caused by disease.

In disease-yield loss studies, it is important to determine water use, as the water balance of plants may be affected by disease. Water use data are also required for estimates of water use efficiency, which indicates whether the water used by the plants has contributed towards grain yield. To measure the amount of water use by plants, soil water measurements should be taken down to the actual rooting depth. In cereals, the actual rooting depth may be up to two metres (Weaver, 1926). It is also important to account for upward fluxes and drainage, as they influence soil water measurements.

This research programme provides guidelines for further disease-root development and disease-irrigation experiments. Studies need to be conducted to identify growth stages at which irrigation may be applied to diseased plants to obtain optimum responses. For example, an important growth stage for irrigation may be immediately after early disease constraints are alleviated, which may reduce the requirement for further root growth. Thus, more photosynthates which would otherwise have been allocated to the roots may be available for the developing

apex.

In future field trials on the effect of disease on root development and water relations, the nature of the investigation and the methods used would be influenced by the specific objectives of the research. It is important to determine whether resources should be concentrated on root studies, water use, or plant water deficits. There is sufficient information from glasshouse studies, and from the studies described here, to show that root development is affected by foliar diseases. It may be more beneficial therefore, to monitor water use rather than root development. Water use studies may be further simplified by measuring plant water deficits. Hence, it may be possible to monitor the effects of disease on soil water status and the amount of roots from measurements of plant water deficits. Infra-red thermometers may provide a quick method of determining the plant water status of a crop, and also identify disease induced water stress, and/or the need for irrigation.

Roots are the major sites for synthesis of plant growth regulators. The possible effect on plant growth regulators in disease-root development studies requires investigation, as they may be indirectly affected by foliar disease. Possible changes to the production and activity of plant growth regulators may affect the physiological and growth processes of plants.

In the long term, field experiments should be carried out in different sites and varying climatic conditions, using the guidelines proposed. The results from further research may be incorporated into disease-yield loss simulation models. This would enable prediction of root development and water requirements of cereal crops affected by foliar diseases in varying soils and climatic regions. The interdisciplinary approach adopted in these studies provided information on the role of roots in a foliar disease complex. This information may be used as inputs for plant growth mechanistic models, in a similar way to physical variables such as radiation, temperature, and water availability. Such models would provide a better understanding of the ways in which disease affects plant growth and productivity.

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