A BIOMETRICAL STUDY

OF THE EFFECT OF NONSPECIFIC PATHOGENICITY GENES

ON HOST AND PATHOGEN FITNESS RELATED CHARACTERS

IN THE USTILAGO HORDEI-HORDEUM VULGARE SYSTEM.

By

DAVID D. POPE

B.Sc., North Carolina State University, Raleigh, 1975
M.Sc., The University Of British Columbia, Vancouver, 1982

A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF
THE REQUIREMENTS FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY
in
THE FACULTY OF GRADUATE STUDIES
(Department of Botany)

We accept this thesis as conforming to the
required standard

THE UNIVERSITY OF BRITISH COLUMBIA
April 1986

In presenting this thesis in partial fulfilment of the
requirements for an advanced degree at the University
of British Columbia, I agree that the Library shall make
it freely available for reference and study. I further
agree that permission for extensive copying of this thesis
for scholarly purposes may be granted by the head of my
department or by his or her representatives. It is
understood that copying or publication of this thesis
for financial gain shall not be allowed without my written
permission.

Department of _Botany (Genetics)_

The University of British Columbia
2075 Wesbrook Place
Vancouver, Canada
V6T 1W5

Date _April 28/86_

ABSTRACT

Nine Ustilago hordei sporidia that produced 20 dikaryons were isolated at random from an F2 teliospore (18D1+ x 20C1-) descended from race 7 and race 11. The 20 dikaryons were homozygous for a dominant gene conferring virulence on the barley variety Trebi and were suspected of segregating for nonspecific pathogenicity genes on this variety. Varieties Odessa (the universal suscept, with no known specific resistance genes) and Trebi were inoculated with each dikaryon and 58 host and pathogen fitness related variables were measured.

Yield reduction occurred both in diseased and healthy plants as a result of the dikaryon treatments. A statistically significant negative correlation between host and pathogen reproductivity was found (r=-0.466, P=0.0481) on Trebi but not on Odessa.

Statistically significant differences among dikaryons were found for some fitness related variables. The segregation of nonspecific pathogenicity genes with pleiotropic effects was believed to cause these differences. One of the genes was found to be tightly linked to the mating locus, coupled with the "-" mating allele. Analysis of variance revealed significant dominance and/or epistatic interaction effects on fitness related variables.

The two varieties reacted differently to the dikaryons. Pathogen isolates exhibited specific adaptation to Trebi but not to Odessa. The presence of the nonspecific pathogenicity genes was readily measured statistically on Trebi, in the background

of a matched specific resistance gene but not on Odessa.

The traditional method of measuring disease damage level (percent smutted plants) was determined to be a reliable estimator of pathogen fitness on Trebi ($R^2=0.84$) and pathogen reproductivity on both varieties (r=0.902, P=0.0001 on Trebi and r=0.815, P=0.0001 on Odessa). Due to weak correlation, prediction of host fitness should not be attempted using values calculated with either of the two traditional methods of measuring disease damage level (percent smutted plants and percent smutted heads).

Stepwise regression of various combinations of variables indicated that Trebi, Odessa or smut dikaryon fitness can be accurately estimated with certain predictor variables.

Spearman rank correlation tests suggested that "constant (concordant) ranking" of dikaryons for percent smutted plants and for pathogen fitness was evident on Odessa and on Trebi (r=0.871, P=0.0001 and r=0.713, P=0.0004, respectively).

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ACKNOWLEDGEMENTS

1 INTRODUCTION

Fully one half of all living species of plants and animals are parasitic for at least a portion of their life cycle (Price, 1980). Plant parasites are particularly important because of the impact they can have on the quality of human life. Plants provide 95% of the world's food (Walsh, 1984) and of the 350,000 known plant species identified, only about 24 crop plants "stand between people and starvation" (Wittwer, 1980).

Plant parasites can appear suddenly, reach epidemic proportions quickly and reduce host yield potentials by diverting host resources for their own reproductive needs. The FAO (1981) estimated that approximately 1/3 of all crops are lost to parasites and pests each year. Much research is targeted at methods of reducing these losses. The most promising results come from the field of genetics.

Interactions between plants and their parasites are known to be mediated by their respective genetic systems. Shortly after the rediscovery of Mendel's work, Biffen (1905, 1907) showed that two recessive resistance genes controlled wheat resistance to the fungal pathogen Puccinia glumarum. Following the discovery of sex in the smut, Ustilago violacea, by Kniep (1919), genetic studies of pathogenicity became more comprehensive. Flor's novel series of experiments (1942, 1947, 1955, 1956) and Person's subsequent theoretical expansion thereof (1959), were important contributions toward understanding fundamental principles governing these inter-organism interactions. Their work revealed how discrete

autonomous genetic systems could be integrated to regulate disease expression. These interactions (described in more detail in a later section) provided the basis for the first wave of disease resistance breeding.

As a consequence of the knowledge gained from this work, new discoveries have been made, and innovative theories, and host management strategies have been devised. It is with some of the these that this work is concerned.

2 GENETICS OF HOST-PARASITE INTERACTIONS

Disease expression is a complex character influenced by genetically controlled resistance in the host and by genetically controlled pathogenicity in the pathogen. Resistance is shown by a host when the pathogen is hindered and disease is reduced (Robinson, 1969). Pathogenicity is shown when a pathogen can attack a host and disease is promoted (Robinson, 1969).

Breeders have concentrated their efforts on bolstering host resistance levels without regard for the ramifications of the accompanying genetic changes induced in the pathogen population. Critical forces involving "...feedbacks between population genetics and population dynamics over space and time..." (Fleming, 1982) and physiologic mechanisms involved in complex interactions are overlooked or ignored. Future crops are placed at risk because breeders have not adopted a holistic approach for managing pathosystems.

A pathosystem is a subsystem of an ecosystem (Robinson, 1976) which involves interactions between plants and their parasites and may be natural (wild pathosystem) or artificial (crop pathosystem). The important role of pathogen genotype in crop pathosystems is now being recognized and investigations of plant diseases are now incorporating simultaneous genetic studies of both organisms.

2.1 SPECIES COMPATIBILITY

There are three recognized types, levels, or subsystems within a pathosystem, of genetically controlled interactions between a host and its pathogen. The first subsystem is one of species compatibility. Before any individuals of a pathogen species can attack any individuals of a host species, compatibility between species must exist. For instance, potato is a nonhost of wheat stem rust because of the absence of compatibility between them (Heath, 1985). It is considered impossible for any nonpathogen to be capable of overcoming this type of resistance. Researchers hypothesize that genes blocking species compatibility might be transferable between host species to effect permanent protection from some diseases (Heath, 1985). As yet, little is known about the genetics of species compatibility.

2.2 SPECIFIC GENES

The second subsystem, the vertical subsystem (Robinson, 1986), involves specific resistance genes and specific pathogenicity genes that interact according to the gene-for-gene theory (Flor, 1971; Person, 1959).

Specific resistance and pathogenicity can be recognized only under certain conditions. Alleles at a specific resistance locus in the host interact in a unique and predictable way with alleles at a specific complementary pathogenicity (virulence) locus in the pathogen. The presence or absence of certain

alleles at either interacting locus can be detected by virtue of the discrete segregation ratios they produce. Once detected and identified, specific genes can be manipulated using classic Mendelian techniques.

Typically, in gene-for-gene interactions, host resistance alleles are dominant and host susceptibility alleles are recessive, although recessive and incomplete resistance have been recorded. Also, pathogen avirulence alleles are dominant and pathogen virulence alleles are recessive. Here too, exceptions have been found (Day, 1974; Vanderplank, 1982; Barrett 1985; Person, Christ and Pope, 1986). Barrett (1985) believes that there are more documented examples of systems with dominant resistance than those with recessive resistance because of breeders selection techniques.

In a classic gene-for-gene interaction the combination of a resistant host genotype with an avirulent pathogen genotype triggers a "stop signal" (Person and Mayo, 1976) and does not result in a disease phenotype. Any other genotypic combination will result in disease.

The effect of specific resistance is to reduce the initial pathogen inoculum (Vanderplank, 1968). Specific resistance genes are used in disease resistance breeding programs and offer temporary resistance against specific virulence genes in the pathogen population. Newly introduced specific resistance genes bring intense selection pressures to bear on the pathogen population (Person, 1968). The matching specific pathogenicity allele increases in frequency in the pathogen population to

epidemic proportions (Person, 1959, 1965). Unfortunately, specific resistance genes involved in gene-for-gene interactions provide short lived protection. Most researchers agree that for some crops, gene-for-gene resistance is inadequate and that new breeding tactics should be used. In response to the failure of specific resistance in some crops, new theories and host management strategies have been devised.

Specific resistance is known by several other names: vertical (Vanderplank, 1963, 1968, 1975, 1978, 1984), race-specific, R-gene, qualitative, oligogenic, major gene, hypersensitive and inoculum reducing resistance. Each of these names has a corresponding specific pathogenicity or virulence counterpart.

2.3 NONSPECIFIC GENES

Nonspecific resistance and nonspecific pathogenicity genes comprise the third subsystem, the horizontal subsystem (Robinson, 1973, 1986). Effects of nonspecific genes are observable only on susceptible hosts (ie. in gene-for-gene interactions where specific resistance is unmatched by specific virulence). Identification of nonspecific genes is precluded by the presence of unmatched specific resistance. The action of each nonspecific allele is not contingent upon the presence of any allele in the other organism. Each allele contributes a small additive increment to the continuously varying disease phenotype (Knutson and Eide, 1961; Habgood, 1973; Clifford and Clothier, 1974; Schwarzbach and Wolfe, 1976). Most nonspecific

resistance and pathogenicity genes do not display gene-for-gene characteristics (Person, 1966).

Wolfe (1972) contends that there is no clear cut distinction to be made between specific and nonspecific genes. He believes that they represent extremes of a continuum and that all genes are of the gene-for-gene type. Genes thought to be nonspecific have not yet been shown to be involved in gene-for-gene interactions.

Other researchers agree that there are no nonspecific genes (Riley, 1973; Ellingboe, 1975, 1981; Clifford, 1975; Nass et al., 1981). They believed that the quantitative effects of so called nonspecific genes are simply the ghost or residual effects of specific resistance genes, of the gene-for-gene type, that have been matched and defeated by specific virulence genes. Anderson (1982) criticized the findings of Nass et al. Anderson attributed the putative residual effects to assumed linkage and genetic drift of quantitative resistance genes during breeding of the near isolines.

Single nonspecific genes generally do not produce discrete segregation ratios, consequently, statistical and quantitative genetic techniques must be used when studying nonspecific genes (Kulkarni and Chopra, 1982). Methods of studying quantitative characters such as those controlled by nonspecific resistance and pathogenicity genes were developed in higher organisms (Mather and Jinks, 1971; Falconer, 1981) and can be applied to most hosts and many pathogens, including fungi (Caten, 1979).

It is rare for a pathosystem to lend itself readily to a

comprehensive genetic study of nonspecific genes, usually because of common biological constraints (ie. the inability to grow the pathogen in culture, isolation and breeding problems, etc.) and preexisting specific resistance. Despite these problems, nonspecificity has been suggested to be involved in several pathosystems: Cercosporella in wheat (Bruehl et al., 1968), Trichometosphaeria turcica in cereals (Nelson et al., 1970), Ustilago hordei in barley (Emara, 1972; Emara and Sidhu, 1974), Phytopthora infestans in potatoes (Caten, 1974; Shattock, 1976), Ceratocystis ulmi in elm (Bassi and Burnett, 1979), and, Gaeumannomyces graminis var tritici in wheat (Blanch et al., 1981).

Despite the epidemiologic significance of nonspecific pathogenicity, little is known about how nonspecific resistance would affect pathogenicity at the population level. The value of using nonspecific resistance in disease management programs can be ascertained only after the dynamics of the interplay of nonspecific genes are more thoroughly investigated both experimentally and theoretically.

Nonspecific pathogenicity also is known by other names: horizontal, nonspecific, polygenic, quantitative, minor gene, rate increasing and nonhypersensitive inducing pathogenicity, as well as aggressiveness. Each of these names has a matching nonspecific resistance counterpart.

2.3.1 CONSTANT RANKING

According to Vanderplank (1963), pathogenic isolates causing quantitatively different smut disease levels on a variety (because of nonspecific pathogenicity gene differences), can be ranked in order of disease severity, provided that gene-for-gene interactions are not involved. This rank order is considered indicative of the cumulative effects of all nonspecific pathogenicity genes combined with the cumulative effects of all nonspecific resistance genes. Since nonspecific gene effects are considered to be additive (Fleming and Person, 1982), rank order is supposedly maintained on different varieties. Similarly, host varieties can be ranked in order of their level of resistance against a series of pathogen isolates (Driver, 1962). Simultaneous ranking of both organisms is known as "constant ranking" (Vanderplank, 1963; Robinson, 1976) and is based exclusively on the level of disease damage, assessed by measuring variables thought to be correlated to pathogen reproductivity, or by directly measuring pathogen reproductivity.

2.4 QUEST FOR DURABLE RESISTANCE

Ephemeral disease resistance in economically important crops has sparked a search for the genetic elucidation of durable resistance. Durable resistance is defined as resistance that remains effective in a cultivar over a wide geographic area in an environment favorable to the disease (Johnson and Law, 1973, 1975). Durable resistance, considered to be both temporally and spatially stable, is now one of the most highly sought after breeding characters in crop plants (Person et al., 1983). Much attention has been focussed on the genetic causes of durable resistance in plants in attempts to avoid recurring boom-and-bust cycles (Johnson, 1961).

Several host management alternatives for combating disease losses have been proposed. Some of these alternatives are thought to provide durable crop resistance through genetic homogeneity and others through spatial or temporal genetic heterogeneity (thought to closely parallel natural pathosystems):

1. Multilines (Borlaug, 1958, 1965; Browning and Frey, 1969; Frey et al., 1973; Groth and Person, 1977; Marshall and Weir, 1985);

2. Pyramiding of specific resistance genes (Luig and Watson, 1970; Abdalla and Hermsen, 1971; Nelson, 1978);

3. Allele cycling (Person, 1966);

4. Nonspecific resistance (Vanderplank, 1968; Main and Gallegly, 1964; Umaerus, 1969; Eide and Lauer, 1967; Simons and Murphy, 1967; Person et al., 1983);

5. Combinations of these methods (Graham and Hodgson, 1965; Raymundo and Hooker, 1982).

For each alternative listed above (from Pope, 1982) there are associated positive and negative aspects. Nonspecific resistance promises great efficacy in reducing disease loss. Studies indicate that nonspecific resistance is potentially durable (Lewellen et al., 1967; Caten, 1974; Vanderplank, 1975; Parlevliet and Zadoks, 1977; Fleming and Person, 1982; Raymundo and Hooker, 1982; Person et al., 1983; Robinson, 1986).

Under epidemic conditions, a high level of nonspecific pathogenicity would produce a rapid rate of spread. High levels of nonspecific resistance could retard the rate of spread of the epidemic. The large numbers of genes involved in nonspecific resistance could buffer against or dampen, increases in nonspecific pathogenicity (Fleming and Person, 1982). The consensus of opinion is that nonspecific resistance can help attain durable resistance (Walsh, 1984).

Durable resistance can never be conclusively shown to exist in a crop until it has persisted in many geographical locations. The threat that this resistance might break down is constantly present. There are a few examples of crops with suspected durable resistance. One such example is the almost complete protection from stem rust that the Sr6 and Sr9 resistance genes gave Canadian wheat for 20 years (Harlan, 1976). It is interesting to note that the resistance is not geographically stable. The same genes failed in Texas. Crops are considered to have potential durable resistance until such time that the

resistance loses its effectiveness and is declared, retrospectively, to have been ephemeral.

It is not yet possible to make factual statements about the precise genetic nature of durable resistance. One popular view is that the combined effects of specific and nonspecific resistance genes can produce durable resistance. Before we can fully understand all aspects of durable resistance we should direct more attention to the least studied components of host-parasite interactions, in particular, the horizontal subsystem.

3 QUANTITATIVE MEASUREMENT OF DISEASE LEVELS

3.1 DEFINITION OF FITNESS

Fitness is a measure of the ability of an individual to pass on alleles to its offspring. The absolute fitness of an individual is the final outcome of all its developmental and physiological processes (Falconer, 1981). Absolute fitness is greater than or equal to 0 and is the expected number of offspring that an individual will contribute to the next generation (Roughgarden, 1983). Individuals within a population differ in absolute fitness.

Relative fitness is the "relative ability of different genotypes to pass on their alleles to future generations" (Hedrick, 1983). The net result of the effects of a number of variable characters, which may be influenced by genetic variation in combination with environmental components, is a measure of relative fitness (Hedrick, 1983). Variation in metric characters can reflect variation in fitness to different degrees (Falconer, 1981).

Relative fitness of an individual is the absolute fitness of that individual divided by the highest absolute fitness in the population. The fundamental theorem of natural selection states that the average relative fitness increases each generation to a peak value (ie. it is maximized). This is not considered realistic because an individual's relative fitness does not remain constant through time (because of frequency-dependent and density-dependent selection effects).

For a trait to be selected it must increase the relative fitness of the bearer and not just the absolute fitness (Wilson, 1980). Fitness is a function of the trait under selection and the size of the population.

Another measure of genotypic fitness is derived from r- and K-selection (Andrews, 1984). Selection for high r- traits, is associated with populations in the exponential phase of growth and promote increased growth rate for a population under conditions of low density. Selection for high K- traits is associated with populations that are near or at the carrying capacity of the environment and promote high equilibrium population size for a population under conditions of high density. Density-dependent selection causes the evolution of high K- traits while density-independent selection causes evolution of high r- traits which occur in a low density population when it is expanding (Dobzhansky, 1950).

In pathosystems, parasitic fitness can concern the ability of isolates or genotypes within a pathogen population to compete successfully and to persist over time (Nelson, 1979). Host attributes conferring nonspecific resistance influence certain components of parasitic fitness. A reduction in one or more components of parasitic fitness can be caused by nonspecific resistance.

Istock (1982)indicated that primary fitness characters include survival probabilities, development times and fertilities associated with particular genotypes in certain environments. "Natural populations may store large reservoirs

of variation, in the polygenic form, which is manifest only with environmental change. At this point, such speculations serve mostly to emphasize our need to know much more about the nature of polygenic variation" (Istock, 1982).

Biometrical analysis is an important and useful tool for the study of fitness characters because these characters usually show continuous variation as a result of the underlying polygenic determination. Mendelian alleles make additive, dominance and epistatic contributions to the phenotypic values of individuals and of the population. These contributions can be neutral, positive or negative (Mather, 1971; Falconer, 1981).

There appears to be a decline in the additive genetic variance and heritability as one studies characters closer and closer to the primary fitness characters. This phenomenon may not be generalizable to natural populations, because most supporting information comes from studies of domesticated animals which live in fairly stable environments. Developmental characters typically have heritabilities of 0.1 to 0.4. Fertility measures typically have lower heritabilities of 0.05 to 0.25 (Istock, 1982).

MacKenzie (1978) stated that one obvious measure of parasitic fitness is the apparent infection rate, r, as defined by Vanderplank (1963,1968). Differences in r, among populations, isolates, biotypes, strains or races, reflect differences in parasitic fitness, when tested on the same host genotype under identical environmental conditions (MacKenzie, 1978). Fleming (1982) concurred that fitness is linked to the

rate of disease progress in exponential growth models. It is interesting to note that some individuals continue to use these fitness differences to distinguish pathogenic biotypes or races (Nelson, 1979).

Quantitative measurements of disease phenotype can be made in different ways depending on the system involved and may indicate the type of resistance operating (Kranz, 1983). The measure of the disease phenotype represents the outcome of the interaction between the host and the pathogen and is indicative of the fitness of each. The current belief is that host fitness is expected to be negatively correlated with pathogen fitness (Pimentel, 1961). Data supporting this belief is furnished by Hoy et al. (1985) for the smut-sugarcane system. A high disease reading translates to a high pathogen reproductivity and low host reproductivity. Conversely, a low disease reading indicates high host reproductivity and low pathogen reproductivity.

Durable resistance is a characteristic of a pathosystem and not just of the host population as the expression implies. Durable resistance is measured in terms of the disease level or quantity and is influenced by the resistance of the host and by the pathogenicity of the pathogen (Johnson, 1981). Therefore, pathogen fitness is an important component in studies of durable resistance.

3.2 FITNESS IN RUST PATHOSYSTEMS

Attempts to measure fitness can be found in some epidemiologically related papers. The largest body of knowledge concerning epidemiology in plant pathosystems involves studies of the cereal rust fungi. Rusts are basidiomycetes with complex life cycles that can show great variability (Ingold, 1973). The asexual repeating uredial stage of these specialized obligate parasites allows them to reproduce and spread rapidly. Urediospores increase in numbers exponentially on healthy tissue (Vanderplank, 1963). They cause host yield depression and can spread up to 300 miles in a few days. Urediospores are wind dispersed. They contact host tissue, germinate, penetrate and colonize. All this occurs in 7-14 days (Katsuya and Green, 1967; Leonard, 1969).

Disease level or severity is assessed in terms of infection types which are routinely measured on a relative scale of 0-4. A reading of IT0 (infection type 0) corresponds to a host resistant reaction with necrotic or chlorotic flecks and no sporulation (hypersensitive or specific resistance and specific avirulence). Infection type 4 is a fully susceptible reaction with a sporulating pustule without chlorosis or necrosis. Infection type is affected by temperature, light, host genotype, pathogen genotype, humidity, infection density, plant age and differences in experimental methods (Luig and Rajaram, 1972).

Infection type as a measure of disease was developed by Stakman and co-workers around 1919 (Hoerner, 1919). Stakman's system has undergone minor modifications and is used extensively

for most of the cereal rusts (Roelfs, 1984).

Disease level or severity is the cumulative result of the effects of several factors or components. These components are: infection frequency, latent period, spore production and infectious period. Variations in all four of these components have been recorded and are purported to affect host and pathogen fitnesses. Few studies have directly measured total spore and seed production at the end of a growing season (ie. pathogen and host fitnesses) and compared these totals with component measurements taken at various periods during the season.

Controlled inoculum experiments provide the best approach for measuring components of resistance. The critical components of quantitative resistance can be thought of as resistance that reduces infection efficiency, extends the latent period from inoculation to sporulation, and reduces sporulation. (Parlevliet, 1979).

Rouse et al. (1980) noted that direct measurement of components of nonspecific resistance were tedious, time consuming and prohibitive to the plant breeder. They suggested using alternate approaches for rapid, precise sampling of individual selections.

Infection efficiency, latent period and spore production per lesion parameters can be measured accurately for cultivars with differing levels of nonspecific resistance. Measurements of components of resistance, no matter how accurate, are not sufficient in themselves for reliable assessment of their combined effects on resistance in a variety (Leonard and Mundt,

1984).

Statistically significant interactions between components of rate reducing resistance and epidemiologic fitness have been demonstrated in some host-parasite systems (Johnson and Taylor, 1976; Parlevliet, 1979; Rouse et al, 1980).

3.2.1 INFECTION FREQUENCY

Infection frequency is defined as the proportion of spores that result in sporulation lesions. Resistance to first contact and to colonization will decrease the infection frequency. Differences in infection frequency reflect differences accumulated over various development stages (Parlevliet, 1979). These developmental stages start from the time of the initial establishment phase and end just prior to spore formation. Infection frequency varies with host genotype and developmental stage of the host.

When infections occur in low frequency there is an approximate linear relationship between the number of sporulating infections and the total number of spores produced. When the density increases, the number of spores produced per infection decreases. Relative fitness changes from generation to generation and its average over several generations might differ considerably from relative fitness defined above.

Increasing infection density, by applying higher doses of inoculum, shortens the latent period from infection to sporulation (Yarwood, 1961; Lapwood and McKee, 1966; Katsuya and Green, 1967; Leonard, 1969; Mehta and Zadoks, 1970; Parlevliet,

1975).

3.2.2 LATENT PERIOD

Latent period is the time from infection to spore production and is sometimes confused with the incubation period. Incubation period is the time between inoculation and the first visible symptoms. Latent period increases from the primary leaf to the young flag leaf stage for all cultivars after which it decreases again. Differences among cultivars are small in the seedling stage and large at the adult stage. Latent period is thought to reflect the growth rate of the pathogen.

Latent period is the crucial component determining the apparent infection rate when a large number of reproductive cycles (macrocyclic) are required to complete the epidemic. Parlevliet (1979) stated that for pathogens with fewer reproductive cycles, the effect of the other components becomes more important in the interaction. Ultimately, when only one reproductive cycle occurs per reproductive cycle of the plant (monocyclic), the infection frequency and spore production are the most important fitness determining factors. Examples of monocyclic diseases include the smuts and bunts in cereals.

Latent period is governed by polygenes and it is likely that infection frequency and sporulation capacity are similarly determined (Parlevliet, 1981). Shaner and Finney (1980) noted that latent period was the component that could be measured with least error and was significantly correlated with disease increase in the field.

3.2.3 SPORE PRODUCTION

Spore production is the number of spores produced per lesion or per unit area of infected tissue. Spore production can be measured in specific intervals of time or over the entire infectious period. Spore production is usually measured in spores produced per leaf area, per lesion or pustule, per unit area of lesion or per unit area of sporulating surface (Johnson and Taylor, 1976).

Spore production represents the total effect of all the components of resistance and may be the most useful criterion upon which to base selection (Johnson and Taylor, 1976).

A count of propagules is considered an alternative for, or a complement to, disease assessment. Zadoks (1972) stated that the measurement of the total spore production of pathogens provides an accurate measure of the resistance of the host. Johnson and Taylor (1976) agreed with Zadoks that cumulative spore counts, in quantitative analyses, are analogous to disease resistance and went on to say that spore counts also provide a measure of pathogenicity of the pathogen. They conclude that obtaining cumulative spore counts is too laborious to be used as a routine, efficient selection method.

Lesions are formed when uredia break the plant surface and sporulate during infectious periods of 2-3 weeks (Chester, 1946; Leonard, 1969). Lesion size is the area of host tissue showing disease symptoms. The colony size is the area actually showing signs of the presence of the pathogen. Area, diameter and length are frequently measured and used as estimators of

pathogen fitness. Lesion size is thought to reflect the growth rate of the pathogen in the host and therefore it is thought to reflect its net spore production. Lesions eventually become senescent and lose the ability to become reinfected.

4.2.4 INFECTIOUS PERIOD

Infectious period is the period of time during which pustules produce and release spores. Since so few studies have concentrated on measuring the numbers of spores produced by the end of the growing season, there is little data available concerning aspects related to the infectious period.

3.2.5 RELATIONSHIPS AMONG COMPONENTS

Deshmukh and Howard (1956) and Lapwood (1963) found a close correlation between the resistance to growth of mycelium through host tissues and resistance to production of spores or sporangia. Lapwood (1961) determined that the rates of growth of Phytophthora infestans in potato leaf tissue was the same for some cultivars but that the number of spores per lesion varied. The amount of sporulation corresponded more closely with resistance of cultivars in the field than did growth rate of mycelium.

Heagle and Moore (1970) reported that Puccinia coronata produced fewer pustules, smaller pustules with fewer spores, and retarded hyphal growth and a longer latent period on a resistant oat variety than on a susceptible one. Clifford (1972) and

Parlevliet and Van Ommeren (1975) found that <u>Puccinia</u> <u>hordei</u> produced fewer pustules and had a longer latent period on a resistant barley cultivar than on a susceptible one. Similar results were described for potatoes and <u>Phytophthora</u> <u>infestans</u> (Lapwood, 1966). Lee and Shaner (1985) found a negative correlation between latent period and lesion size.

Rapilly (1979) determined that both latent period and sporulation greatly condition the rate of epidemic progression. He found that the total number of spores produced depends on the duration of their production and on the speed of spore formation and pustule enlargement. Both components were considered as contributors to aggressiveness.

Umaermus (1970) used increased latent period and reduced sporulation capacity of <u>Phytophthora</u> <u>infestans</u> to select for high levels of horizontal resistance in potatoes. Jones (1978) suggested using visual inspection for increased latent period of <u>Erysiphe</u> <u>graminis</u> f.sp. <u>avenae</u> was possible on the third or higher leaf of adult plants.

Gregory <u>et</u> <u>al</u>. (1982) and Gregory <u>et</u> <u>al</u>. (1984) studied the effect of corn genotype on the estimates of relative parasitic fitness among populations of <u>Helminthosporium</u> <u>carbonum</u> by measuring lesion size as an attribute of parasitic fitness to determine the variation in four populations of <u>H</u>. <u>carbonum</u>, race 3. They noticed that host genotype had a great influence on the evaluation of parasitic fitness. They found that less susceptible host genotypes were more effective in detecting differences among populations. They stated that parasitic

fitness in the pathogen may be analogous to rate-reducing resistance in the host. They also think that increased fitness corresponds to an increase in lesion size. Therefore, it may not be appropriate to base race designation on lesion size. Also, they suggested that parasitic fitness should be monitored to detect shifts towards increased fitness and to detect and avoid destructive epidemics.

As previously mentioned, some of the components associated with the macrocyclic rust pathogens are not found in all systems. In the monocyclic barley-smut system, there is only one latent period. The life cycle of the smut coincides with that of its host. Spore production has never been measured directly. The infectious period is irrelevant in this system because only 1 crop of spores is produced per growing season.

The large differences between macrocyclic and monocyclic diseases led Parlevliet (1979) to state that total spore count may provide an accurate measure of pathogenicity only for monocyclic diseases. For macrocyclic diseases, measurements of the individual components of the epidemic development were more important. Parlevliet showed that values obtained from measuring components at various stages during the growing season can vary significantly and that these differences could be caused by any number of contributing factors.

4 THE USTILAGO HORDEI-HORDEUM VULGARE SYSTEM

4.1 BIOLOGY OF U. HORDEI

Ustilago hordei (Pers.) Lagerh. is a bipolar smut fungus, obligately parasitic on barley. In the past, smuts have caused extensive damage to various cultivated plant species. U. hordei is well suited for biometric and population genetic studies of pathogenicity because it is easy to culture, store and harvest.

Teliospores of U. hordei are generally 5-11 u in diameter, smooth and light colored on one side (Fischer, 1953). Upon germination, the diploid nucleus moves out into the long slender promycelium, where it undergoes the first meiotic division (Fischer and Holton, 1957). A wall forms across the promycelium between the nuclei. The nuclei undergo the second meiotic division and two more dividing walls are formed. This results in four linearly arranged, uninucleate cells being produced, with the basal cell extending into the teliospore. Haploid sporidia, representing the products of meiosis, bud continuously from the four promycelial cells. Each bud, in turn, can divide to produce clones (Fischer and Holton, 1957).

Sporidia of opposite mating type can fuse to form dikaryotic hyphae which can penetrate and infect barley seedlings (Fischer and Holton, 1957). The dikaryotic mycelium grows intercellularly in association with the apical tip (Kozar, 1969) and forms sori in the spikelets, replacing the seeds with smut balls consisting of millions of spores. Mechanical harvesting techniques rupture the basal part of the glumes that

encase the smut balls (Stevens, 1913) and spread the spores to other seeds. Seeds can become infected when sown (Tapke and Bever, 1942; Groth and Person, 1976). See figure 1 for the life cycle of U. hordei.

The barley host, Hordeum vulgare L. is a prolific, self-fertilizing crop plant. Highly isogenic cultivars are readily available (Pope, 1982).

4.2 BACKGROUND INFORMATION

Current studies involving covered smut of barley use the "percent of plants smutted" as a measure of disease damage level. Tapke (1929, 1931) used both smutted head and smutted plant counts in his work. Clark et al. (1933) found a high correlation (r=0.741) between the percent of plants bunted and percent of heads bunted with Tilletia caries and concluded that either could be used, despite values based on head counts being consistently lower than values based on plant counts. Ruttle (1934) found a similar correlation in Ustilago hordei. Groth (1974, 1976) stated that it is not valid to establish a correlation between the percentage smutted heads and the percentage smutted plants because the two variables are not independent.

Gaines (1923) decided that head count accurately assessed the impact of smut on crops (ie. on crop yields). While Briggs (1926) and Churchward (1937-1938) maintained that smutted plants should be used for assessment of smut particularly for genetic reasons (ie. for the identification of particular genes).

A reduction in tillering was reported for nearly all cereal smuts (Welsh, 1932; Mather and Hansing, 1960; Gaunt and Manners, 1971). Ruttle (1934) found reduced tillering of barley in plants smutted with Ustilago hordei. No difference in tillering between inoculated smutted and inoculated nonsmutted plants was found in the barley-smut system (Groth, 1974; Groth and Person, 1978). Groth identified two distinct hurdles that the smut must overcome in order to produce spores. If the smut can overcome both hurdles then it can smut all or nearly all the culms. The first hurdle might equate to specific resistance and the second, to nonspecific resistance. When resistance was high, nearly all culms were healthy. When disease severity was high, most plants were usually either totally smutted or nonsmutted.

Groth inoculated 12 varieties of barley with 21 different dikaryons, some from Tapke's physiologic races (Tapke, 1937, 1945). Occasionally the levels of smutting differed from Tapke's. Groth discovered that an inverse relationship existed between the within-plant disease severity and the average number of culms produced. In other words the higher the percentage of smutted heads a plant had, the lower the number of tillers it had. Smutted heads occurred nonrandomly in smutted plants. Tiller families tended to be either all healthy or all smutted. Older culms remained healthy more frequently than did younger culms, both within and between tiller families. The pathway of smuts to parts of the crown is thought to be highly variable. There is probably a close connection between growth to and through the crown, and distribution of smutted culms. He

believed that the fungus could be present in all seedlings just after inoculation and postulated that yield loss may occur even in the absence of fungal sporulation.

Batts and Jeater (1958) stated that only a limited amount of mycelium was produced in any plant and that in high tillering plants only a limited amount of mycelium would find its way into a tiller. As a result only a few tillers would become diseased.

The seedling could outgrow the smut mycelium (Ohms and Bever, 1955). Mycelia might need to reach a critical point, grow at a particular rate or be in a specific location during a plant developmental stage before a tiller or tiller family will become smutted (Person, personal communication). Deep sowing and cool temperatures at germination might tend to slow plant growth and extend the critical period.

Tapke (1938, 1941) determined that smut levels on barley could be affected by the post germination environment. The deeper the seeds were sown, the greater the number of smutted plants and the greater the number of smutted tillers that were produced. Woodward and Tingle (1941) observed that less fertile soil produced high smutting in Ustilago hordei. Ebba (1975) found evidence of genotype by environment interactions. An isolate that produced a low level of smut in Vancouver produced high smutting on the same cultivar in California.

Multiple infections were demonstrated in oats infected with Ustilago kolleri (Person and Cherewick, 1964). Multiple infections also occur in Ustilago hordei (Megginson and Person, 1974; Mylyk and Person, unpublished).

4.3 QUANTITATIVE INVESTIGATIONS

Quantitative techniques were first employed by Emara (1972) to investigate aggressiveness in Ustilago hordei. Odessa seeds were inoculated with representatives from Tapke's 13 races (1937, 1945). The percent infected spikes was measured for rows of 30 plants each (an unusually small number). A considerable amount of genetic variability was detected. Most of it was additive with a small contribution by dominance and epistatic effects. Narrow sense and broad sense heritabilities were calculated (table 1).

Emara and Sidhu (1974) studied the polygenic inheritance of aggressiveness in Ustilago hordei by selfing and crossing two teliospores and using the resulting 16 dikaryons to inoculate the susceptible barley cultivar Vantage. Aggressiveness, defined as the degree of infection, was found to be a continuous character genetically controlled by polygenes which modified the expression of the recessive virulence allele Uhv4. A large amount of variance was found both among and within teliospores. The dikaryons produced by crossing were more aggressive than those from selfing. This implied the operation of heterosis. Additive, dominance and epistatic effects occurred (table 1).

Sixteen smut dikaryons from 8 meiotic products of 2 teliospores were constructed by Emara and Freake (1981). These dikaryons were used on the compatible barley variety, Hannchen, in 5 different macro-environments. Analysis of variance revealed significant differences among dikaryons and among macro-environments. Interactions between parasites and macro-

environments were not significant. Genetic variability was 28.1%, micro-environmental variability was 41.4% and macro-environmental variability was 30.5% of total variability. They concluded that pathogenicity of <u>Ustilago hordei</u> is a highly variable character which is also sensitive to environmental conditions. They stated that disease incidence of covered smut of barley in the same environment on the same cultivar is a direct indication of pathogenicity of different genotypes of <u>Ustilago hordei</u> (table 1).

Caten <u>et al</u>. (1984) examined genetic determination of a quantitative component of pathogenicity (ie. aggressiveness) in <u>Ustilago hordei</u>. They measured the proportion of smutted plants produced from inoculated seeds of a susceptible barley cultivar in progeny populations derived from 3 parent dikaryons. The race 10 strain parental dikaryon was found to be highly homozygous for genes affecting aggressiveness. Highly variable progeny resulted when sporidia from this parent were mated with unrelated sporidia. Aggressiveness was found to be determined by a polygenic system that involved both additive and dominance effects. The number of genes was not determined. A factor affecting aggressiveness was found linked tightly to the mating type locus. Dominance was bidirectional and genotypes with an intermediate genotype were most fit. They stated that aggressiveness is important in determining the severity of epidemics of susceptible hosts. Also, they believe that aggressiveness is a major component of fitness and may even influence the frequency of virulence factors in pathogen

populations and the evolution of new races (table 1).

4.4 CURRENT WORK

Ebba (1974) initiated an investigation into the inheritance of pathogenicity on the barley variety Trebi, in descendents of a cross between two Ustilago hordei teliospores, one from race 11 and one from race 7 (Tapke, 1937, 1945), later renamed T1 and T4, respectively (table 2). Eight F1 dikaryotic lines numbered 17 through 24 were formed by crossing the products of meiosis from the T1 teliospore with those of the T4 teliospore, in all compatible combinations (table 3). Results from his crosses and backcrosses led him to conclude that a series of alleles, at a single locus and with a hierarchy of dominance, controlled pathogenicity on Trebi.

Subsequent classical genetic analysis by Person (personal communication) uncovered the segregation of a single dominant specific virulence gene in the descendents of the T1 x T4 parental cross. Person, Ebba and Christ (1986) found that in addition to the specific virulence gene, other nonspecific pathogenicity genes were segregating and that isolates could be ranked according to the magnitude of their disease phenotype. Ranking reflects the nonspecific genotype of the individual (Vanderplank, 1963, 1968, 1984; Person, 1983). Biometrical analysis of the F2 progeny from the parental cross showed statistically significant variety x dikaryon and inter-dikaryon differences (Pope, 1982). The variety x dikaryon differences were attributed to the segregation of the specific virulence

gene and the inter-dikaryon differences were indicative of the segregation of nonspecific pathogenicity genes. The number of genes was estimated to be between 2 and 4.

The nonspecific pathogenicity genes exhibited dominance and epistatic interactions, ambidirectional dominance, interactions with environmental components and possible interactions with the virulence gene. At least 1 gene with a large effect was found to be tightly linked with the mating locus.

Person isolated 24 sporidia of both mating types (12 with and 12 without the dominant virulence gene) from the 8 F1 dikaryotic lines and crossed them in every possible way to produce a 12 x 12 matrix of 144, F2 dikaryons. The F2 dikaryons could be divided into 3 groups according to virulence genotype. Dikaryons homozygous for the dominant virulence allele produced higher disease readings, on average, than did dikaryons that were heterozygous for the virulence allele. Virulence gene heterozygotes produced higher disease readings than did the recessive homozygotes.

Each F1 sporidium was ranked according to the average quantitative measure of the magnitude of the disease phenotype when combined with all other sporidia of the opposite mating type, on the barley variety Trebi. This represents the first time that concordant ranking of polygenically controlled nonspecific pathogenicity has been conclusively shown. Biological material generated from this study offers the unique and exciting opportunity to study absolute and relative measures of host and pathogen fitnesses and to test the hypothesis of

"constant ranking".

5 PURPOSE

The purpose of this study was investigate important aspects of nonspecific pathogenicity gene effects in a pathosystem.

In cereal-rust systems, as previously described, a relatively large body of information is available concerning the fitness related components infection frequency, latent period, spore production and infectious period. In the monocyclic barley-smut system very little information has been generated concerning characters other than the percent of plants smutted. This disparity is explained by fact that host-pathogen systems involving rust are more prevalent in agriculture than those involving smut. A greater amount of time, energy, and resources have been directed toward investigating rust systems. Also, because of the macrocyclic nature of the life cycle of the rusts, direct measurements of pathogen fitness are virtually impossible. Consequently, attention must be focused on characters that are believed to be closely related to pathogen fitness.

The monocyclic life cycle of the smut pathogen lends itself to direct measurement of reproductivity. Spores counts can be taken at the end of the growing season. As yet, direct measurements have never been made.

Several interesting questions are raised about smut systems in light of the absence of detailed information concerning fitness related components. Do fitness related components similar to those in rust systems exist in the barley-smut system? Can they be identified? Can any of the pathogen or

host fitness related variables provide accurate and cost effective estimations of host and pathogen fitnesses?

These questions are addressed in this study. Other questions specifically tailored to suit the unique biological material chosen for this study also will be addressed. These questions include the following. Will different nonspecific pathogenicity gene combinations have an effect on any of these pathogen fitness related variables? How do nonspecific pathogenicity gene differences affect host fitness related variables?

In cereal-rust host-pathogen systems disease level readings are easily obtained by scoring one or a few plants treated with the rust isolates of interest. Some systems even permit in vitro detached leaf techniques. The disease level readings obtained from these systems are absolute and are easy to handle quantitatively. Conceptually these readings are easy to grasp because genotypically identical plants produce identical disease readings under similar conditions. Also, a minimum of biological material (ie. a small number of plants) is necessary to obtain these readings.

In the barley-smut system, a relatively large number of plants must be treated with the isolates of interest before disease levels can be assessed. A large number of plants must be inoculated with each smut isolate because traditionally the disease level has been measured as the percentage of treated plants, or less frequently, as the percentage of smutted heads that show signs of disease.

These measures of disease level involve probabilities. In the case of the percent plants smutted, the measure of disease damage is the probability that a single susceptible plant will show signs of disease. Conceptually this measure of disease is more difficult to grasp and deal with than that for the cereal-rust system. The conceptual difficulty arises because a diseased plant, treated with a virulent smut isolate, can show signs of disease while another plant of the same genotype treated in an identical way can show no signs of disease. Accurate assessments of disease levels can not be obtained by using one or a few plants because any single plant will be scored either as diseased or not diseased. In order to obtain a measure of disease level many genetically identical plants must be scored as either diseased or not diseased and the ratio (percentage) of diseased plants to nondiseased plants becomes the probability of a plant of that genotype becoming diseased.

Probability (percentage) values have become accepted at face value as indicators of reproductivity (aggressiveness or fitness) of the pathogenic isolates. Their appropriateness as accurate measures of pathogen reproductivity or fitness has never been assessed. Another untested relationship in the barley-smut system is that between host fitness and pathogen fitness. Intuitively, one would expect a negative correlation between pathogen fitness and host fitness. The existence of this correlation has not been tested in the barley-smut system.

Data gathered from this experiment will also allow a test of the "constant ranking" hypothesis not only for the

traditional measures of percent plants smutted and percent heads

smutted but also for pathogen fitness and host fitness

5.1 OBJECTIVES

In light of the questions and challenges described above

the objectives of this study are

1 to measure and compare a total of 58 putative
   fitness related variables in order to identify
   those which may be closely related to host
   and/or pathogen fitness,

   1.1 to determine the relationship between the
       traditional measures of disease level and a
       direct quantitative measure of pathogen
       fitness,

   1.2 to make direct measurements of host fitness
       to determine if the relationship between host
       and pathogen fitnesses can be determined,

   1.3 to generate data dealing with fitness and/or
       reproductive differences among pathogenic
       isolates for estimating selection ("s")
       values in future modeling experiments,

   1.4 to identify particular subsets of fitness
       related variables that can be used to make
       useful predictions of host and pathogen
       fitnesses,

       1.4.1 to reveal aspects of the underlying
             biology involved in the codevelopment
             of the host and the pathogen which are
             controlled by nonspecific pathogenicity
             genes,

2 to determine if additive or nonadditive gene
  effects of the nonspecific pathogenicity genes
  play an important role in the expression of any
  of the fitness related variables,

   2.1 to establish if different nonspecific
       pathogenicity gene combinations cause
       pleitropic effects of any of the putative
       fitness related variables,

3 to test the "constant ranking" hypothesis in this
  system.

6 MATERIAL AND METHODS

6.1 EXPERIMENTAL DESIGN

A single F2 dikaryon was chosen for analysis from Person's ranked matrix (previously described). Parental sporidia of this dikaryon were labelled 18D1+ and 20C1-. The dikaryon was homozygous for the dominant virulence allele. The choice of the dikaryon was based on the consistently high disease readings on Trebi of one of its parents, T1 (race 11), as well as on the low readings of the other parent T2 (race 7), when paired with compatible sporidia known to have the dominant virulence allele. This dikaryon was expected to be highly heterozygous for nonspecific pathogenicity on the barley variety Trebi.

Ten sporidia were isolated at random from the chosen F2 dikaryon, 5 of each mating type. One sporidium was subsequently lost during subculturing. The remaining 9 sporidia were combined in all compatible ways to produce a 5 x 4 North Carolina Mating Design II matrix (Comstock and Robinson, 1952; Singh, 1979). The resulting 20 dikaryotic treatment combinations were used to inoculate Trebi and Odessa seeds (subsequently referred to as T and O respectively). Consult figure 2 for a diagram of the experimental design.

Odessa was included in this experiment because it is considered a universal suscept (ie. without known specific resistance). Also, Odessa was expected to have a low level of nonspecific resistance based on prior performance. Plots were planted in replicated, randomized complete blocks in the field.

There were three replicates.

6.2 SEED PREPARATION

Single seed progeny of the barley varieties Odessa and Trebi were soaked in a dilute formalin solution (0.12 %) for 30 minutes and then washed thoroughly in tap water for 60 minutes. The seeds were spread thinly on newspaper and allowed to air dry for 48 hours before being placed in 110 seeds lots into 25 ml plastic vials.

6.3 PLANTING

During May, seeds were sown in the field in replicated, randomized complete blocks designs. Rows were aligned in an east to west direction. A hand operated, belt driven, single row seeder was used to space seeds evenly in 10 foot rows at a depth of approximately 2 cm. Plots were weeded and watered as necessary.

6.4 HARVESTING AND DATA RECORDING

Approximately 3-4 months after planting, when heads were golden in color and very dry, measurements made on each treatment row were recorded. Some of the measurements made on the first 50 plants in the row included the following:

- number of diseased plants
- number of heads per plant
- number of diseased heads per plant
- weight of spores per diseased head

- spore germination rate per diseased head
- number of healthy heads
- number of seeds per healthy head
- seed weight per healthy head
- seed germination rate per healthy head
- thousand seed weight

In addition to those listed above, other variables were measured. From the recorded data, still other variables were constructed that related specifically to plant and tiller averages, and, host and pathogen fitnesses. The complete set of variables was subdivided into 4 subsets. A mnemonic code character (R,H,C or P) was assigned to each of the subsets for ease of handling and analysis. The R subset of variables relates to aspects of the "row" in general. H variables involve "healthy" plant measurements. Variables in the C subset were made on "completely" diseased plants (ie. with every head diseased). The P subset of variables was obtained from "partially" diseased plants (ie. with at least 1 healthy head and 1 diseased one). Some variables, normally expressed as percentages, were transformed using a modified Freeman and Tukey (1950) angular transformation (Zar, 1984):

$$p' = 1/2 \ [\arcsin((x/(n+1)^{1/2}) + \arcsin(((x+1)/(n+1)^{1/2})]$$
where
        p' = transformed percent smutted plants,
        x = the number of smutted plants, and
        n = the number of plants scored.

Consult tables 4 to 7 for a more detailed description of the variables used. Figure 3 is a schema showing the interrelationship of the subsets of variables.

6.5 HEAD ANALYSIS

Smutted heads were ground in a mortar to release the teliospores. Plant debris was manually removed and the teliospores were brushed into a weighboat and were weighed on a Mettler balance. The weight of the weighboat was subtracted from the reading to give the actual teliospore weight. When possible, 1 mg of teliospores from each smutted head was placed in 5 ml of sterile water for 30 minutes. Two drops of this suspension were spread on a petri plate containing complete medium and the plate was incubated at 22°C for 18 hours. A straight reference line was drawn on the bottom of the plate. The first 100 teliospores touching the line (moving from east to west) were scored for the presence of promycelium with at least one fully formed sporidium. Teliospores with at least one sporidium were considered to have successfully germinated. The seed number, seed weight and seed germination rate was recorded for healthy heads from diseased plants. Seed germination rates were assessed by calculating the percentage germinating after 4 days in large petri plates containing moist vermiculate. Seeds from the first 50 healthy plants were pooled with seeds from remaining healthy plants in each row. A random sample of 1000 of these seeds was taken and weighed to measure the thousand seed weight variable (H6). The germination rate of a random selection of 100 of these seeds was measured.

6.6 SPORIDIA CULTURE MEDIUM

Three media types were employed in this study: minimal agar
medium, complete agar medium and complete liquid medium. See
Appendix A for recipes. Minimal medium was usually used for
short term culturing while complete medium was used for
procedures lasting more than 2 days.

6.7 SPORIDIA ISOLATION

Smutted heads were surface sterilized in a 1% sodium
hypochlorite (household bleach) solution for 30 seconds then
rinsed in sterile water for 2-3 minutes. The heads were cut
open and teliospores, centrally located within a sorus, were
teased out and allowed to imbibe sterile water for 30 minutes.
Under sterile conditions, droplets of the teliospore suspension
were placed in the center of 20 mm x 20 mm x 3 mm blocks of
minimal medium agar. The agar blocks were mounted on 25 sq mm
coverslips and incubated at 22°C, 100% relative humidity, for 18
hours to promote germination and sporidia production. Each agar
block containing coverslip was inverted and placed on a moveable
stage microscope at 150x magnification. A haploid sporidium was
coaxed to each edge of a block with a bulbous tipped, fine glass
needle, mounted in a de Fronbrue micromanipulator (C.H.
Beaudouin, Paris). After 3-4 days of incubation at 22°C
sporidial microcolonies were visible at block edges. These
microcolonies were transferred to petri plates containing
complete agar medium. The mating type of each isolate was

determined by compatibility with known standards using a modified Bauch test (Bauch, 1932). The appearance of microscopic hyphae ("Suchfaden") signalled compatibility (ie. opposite mating types).

6.8 LONG-TERM SPORIDIAL STORAGE

Cells from each sporidial colony were transferred to sterile complete medium, slant agar tubes. After 4 days, cells were emulsified in 1 ml of sterile water plus 1 ml of double strength skim milk.

Screw capped tubes were half filled with silica gel (Perkins, 1962), loosely capped and sterilized in an oven at 180°C for 90 minutes. Caps were tightened and tubes were allowed to air cool to room temperature.

One ml aliquots of the sporidial cell suspension were pipetted into each tube. Tubes were shaken until all traces of moisture disappeared. Tubes were then placed on ice and later stored at 4°C.

6.9 INOCULATION

Sporidial isolates were placed in tubes containing 5 ml of sterile complete medium with tetracycline HCl at a concentration of 0.075 mg/ml and shaken at 22°C for 48 hours. One ml of each suspension was transferred to separate 250 ml flasks containing 60 ml complete medium and tetracycline HCl at a 0.075 mg/ml concentration. Flasks were shaken at 22°C for 48 hours.

Experimental treatments, consisting of every possible pairwise combination of compatible sporidia were premixed in sterile flasks. Vials containing 110 seeds were inoculated with 5 ml volumes of cell suspension treatments. Vials were then subjected to a negative pressure in a bell jar for 30 minutes (Groth and Person, 1976). The excess liquid was drained from the vials and the wet seeds were transferred to labelled coin envelopes and allowed to air dry for 48 hours prior to sowing.

6.10 STATISTICAL ANALYSIS

The data were analysed on an Amdahl computer with the SAS statistical package (1981, 1982a, 1982b). Some variables were changed to angles using a modified angular transformation to satisfy the a priori assumption of normality. A general linear modelling approach was used in the analysis. Statistical techniques employed included the t test, analysis of variance, Duncan's multiple range test, multivariate analysis of variance, correlation coefficients, Spearman rank correlation coefficient and stepwise linear regression.

7 RESULTS

7.1 DESCRIPTION OF VARIABLES

The listing and description of the 4 subsets of variables is found in tables 4 to 7. A schema in representation of the relationship among the 4 subsets is shown in figure 3. Mean values of treatments and controls for each variable are presented in tables 8 to 15.

7.2 REGRESSION OF SPORE NUMBER ON SPORE WEIGHT

A regression of teliospore number on teliospore weight was made to determine if the expected linear relationship between the two spore related variables existed. The regression indicated that the relationship was linear and that a high positive correlation of 0.9931 existed between the two variables. The intercept was forced through the origin, and the slope was significantly different from 0 (figure 4, Tcalc=33.84, P=0.0001). The teliospore weight coefficient, 1.241E10 (+/- 3.667E8), could have been used as a multiplier to convert raw teliospore weights to teliospore numbers. Each milligram of teliospores on average consisted of 1,241,000 teliospores. The simple linear relationship between the two variables made it unnecessary to actually convert the weight values. Instead, teliospore weight was used in place of teliospore number throughout the remainder of the analysis.

7.3 DESCRIPTION OF FITNESS VARIABLES

Many of the variables measured in this system were expected to be highly correlated with pathogen and/or host fitness. Based on this expectation, six composite fitness variables were constructed from others within the set of measured variables. The fitness variables were specifically constructed for use as dependent variables in subsequent multivariate analysis.

Three of the six composite variables (Wp [PATHOGEN], Wc [PATHOGEN] and W [PATHOGEN]) are absolute measures of pathogen fitness. The first of these three variables was derived from pathogen performance on partially diseased plants (Wp [HOST]). The second variable represents pathogen fitness on completely diseased plants (Wc [PATHOGEN]). The third variable is a measure of total pathogen fitness on all plants (W [PATHOGEN]) and was created by summing the first two pathogen fitness values. The remaining three composite variables (Wp [HOST], Wc [HOST] and W [HOST]) quantify aspects of host fitness. One of these variables represents host fitness on partially diseased plants (Wp [HOST]). Another variable quantifies host fitness on healthy plants (Wh [HOST]). The sixth variable combines the first two as a measure of the total fitness of the host (W [HOST]).

The formulae for these composite fitness values are described below. Calculating pathogen fitness on partially diseased plants for each row involved the following variables: the weight of teliospores from partially diseased plants (P8) and the average teliospore germination percent per diseased head

of partially diseased plants converted to decimal form (P11/100). The exact formula is

$$Wp [PATHOGEN] = P8 \times P11/100$$

The following variables were used to calculate pathogen fitness on completely diseased plants: the weight of teliospores from completely diseased plants (C4) and the average teliospore germination rate per head from completely diseased plants converted to a decimal (C7/100). The formula for Wc [PATHOGEN] is

$$Wc [PATHOGEN] = C4 \times C7/100$$

The sum of Wp [PATHOGEN] and Wc [PATHOGEN] totals W [PATHOGEN], the total pathogen fitness.

Variables Wp [HOST] to W [HOST] represent host fitnesses for partially diseased plants, completely diseased plants, and all types of plants, respectively and were built from the following measurements: the number of seeds from partially diseased plants (P12), the average seed germination rate for partially diseased plants in decimal form (P18/100), the number of seeds from healthy plants (H10) and the average seed germination rate of seeds from healthy plants expressed as a decimal (H9/100). Formulae for Wp [HOST] to W [HOST] are

$$Wp [HOST] = P12 \times (P18/100),$$
$$Wh [HOST] = H10 \times H9/100, \text{ and}$$
$$W [HOST] = Wp [HOST] \times Wh [HOST]$$

Values for Wp [PATHOGEN] to W [HOST] are in tables 8 and 12 for

Trebi and Odessa, respectively.

7.4 SPORIDIAL TREATMENTS VERSUS CONTROL COMPARISONS

Based on prior observations, seeds treated with sporidia were expected to suffer reductions, relative to control, for many of the measurements. One-tailed t tests were performed to determine if statistically significant reductions occurred relative to control. The null hypothesis of no difference between a variable's value and that of the corresponding control was tested at the 95% confidence level for 19 degrees of freedom and was rejected when calculated t values exceeded 1.729.

Variables obtained from partially and completely diseased plants had no matching control variables with which they could be compared. This is because control rows were free of disease, as expected. To circumvent this situation variables from subsets C (completely diseased plants) and P (partially diseased plants) were tested against closely related means from control rows. Test results are found in tables 16 and 17. Significant reductions were observed in the following variables: TH1, TH2, TH9, TC3, TP12, TP13, TP14, TP16, TP17, TP18, OR1, OR6, OR8, OWh [HOST], OW [HOST], OH1, OH2, OH3, OH5, OH10, OC3, OP12, OP13, OP14, OP16, OP17 and OP18.

7.5 VARIABLE MEAN COMPARISONS FOR THE VARIETIES

Equality of variable means was tested either with a correlated groups t-test or with a one-way ANOVA. The t-test null hypothesis was for no difference between paired scores. At an alpha of 0.05, with 19 degrees of freedom the null hypothesis was rejected if the calculated t value was more extreme than the tabulated t value of +/-2.093. The ANOVA null hypothesis was for no difference among means. An F value was calculated by placing the "tested means" mean square (with 2 degrees of freedom) over the "error" mean square (with 57 degrees of freedom) and comparing it with the appropriate tabulated F value (alpha=0.05). The ANOVA was used as a simple alternative to performing three t-tests.

Statistical differences among these means were expected to reflect the range of effects that the treatments had on C (completely diseased), P (partially diseased) and H (healthy) plants and how these effects varied between varieties. Important differences were found between many of the variables. Tables 18 to 21 catalogue the results of the comparison of means.

Statistically significant differences were revealed in the t-tests between variable pairs on Trebi and Odessa. The pairs tested are as follows:

- the average number of diseased heads (R7) and healthy heads per plant (R8),

- the number of healthy heads from healthy (H2) and partially diseased plants (P4),

- the average number of seeds per plant for healthy
  (H4) and partially diseased plants (H13),

- the average number of seeds per head for healthy
  (H5) and partially diseased plants (P14),

- the average seed weight per plant for healthy
  (H7) and partially diseased plants (P16),

- the average seed weight per head for healthy (H8)
  and partially diseased plants (P17),

- the average seed germination rate per head for
  healthy (H9) and partially diseased plants (P18),

- the number of seeds from healthy (H10) and
  partially diseased plants (P12),

- the number of diseased heads from completely
  diseased plants (C2) and from partially diseased
  plants (P3),

- the average number of diseased heads per plant
  for completely (C3) and partially diseased plants
  (P6),

- total spore weight for completely (C4) and
  partially diseased plants (P8),

- average spore weight per plant for completely
  (C5) and partially diseased plants (P9),

- the average spore weight per head for completely
  (C6) and partially diseased plants (P10),

- the average spore germination rate per diseased
  head for completely (C7) and partially diseased
  plants (P11),

- the number of diseased (P3) and healthy heads for
  partially diseased plants (P4),

- the average number of healthy (P6) and diseased
  heads for partially diseased plants (P7),

- the pathogen's fitness on partially (Wp) and
  completely diseased plants (Wc) and the fitness
  of the host on partially diseased (Wp [HOST]) and
  healthy plants (Wh [HOST]).

Other differences among means were found in the three
ANOVA's involving: the number of healthy (H1), completely

diseased (C1) and partially diseased plants (P1), the number of heads from healthy (H2), completely diseased (C2), and partially diseased plants (P2), and, the average number of heads per plant for healthy (H3), completely diseased (C3) and partially diseased plants (P5).

7.6 ANOVA

Each variable underwent a different and separate analysis of variance. The objective was to partition total variance for each variable into the seven possible contributing sources. These source components were "+" sporidia, "-" sporidia, "rep" (replicates), "+x-" sporidia interactions, "+xrep" interactions, "-xrep" interactions and "+x-xrep" interactions, which was redefined as the error component. The null hypothesis was for the equality of group means. The level of significance was set at alpha=0.05. Pseudo-F values were calculated and used to test the main effects components because appropriate denominator mean squares were not available. The interaction components were tested against the error mean square. Relative contributions of each variance component to total variability was assessed using the expected mean square table and was expressed as a percentage (tables 22 to 29).

Table 30 was constructed to summarize the results of the ANOVA's. An asterisk was placed in the appropriate column for variance components that had significant F values. Table 31 presents the frequencies of specific combinations of significant components for each subset of variables and for each variety.

$\underline{7}.\underline{7}$ MODELS

Four groups of models, determined by stepwise regression and constructed from specific subsets of independent variables, were designed to estimate host (W [PATHOGEN]) and pathogen fitness (W [HOST]) values and to provide explanations for the underlying biology associated with aspects of fitness. These groups are subsequently referred to as:

- COMPLETE

- TRADITIONAL

- PRACTICAL

- DEVELOPMENTAL

The criteria for inclusion of a variable in a model was that it had an F value with a probability of no more than 0.15, and that this probability was maintained when the variable was included in the model. Variables were excluded or removed from a model if these criteria were not satisfied. Models chosen as being "best" were those that had no term with an F value probability greater than 0.05. "Best" models were chosen so that models with more terms did not have significantly larger R^2 values. Whenever possible, models were refitted with select independent variables known to be influenced by nonspecific pathogenicity (ie. in ANOVA's these variables had statistically significant genetic related components). Models with these independent variables are signified with the letter "G" next to

the dependent variable. Residual analysis of all models revealed no unusual outlying values. Therefore, apriori assumptions about the normal distribution of error values about a mean of zero were supported. Regression results are found in tables 32 to 47.

7.7.1 COMPLETE

These models involved nearly the entire set of variables measured for this host-parasite system (tables 32 and 33). Obvious problems with multicolinearity were avoided by excluding select constructed fitness variables (Wp [PATHOGEN], Wc [PATHOGEN], W [PATHOGEN], Wp [HOST], Wc [HOST], W [HOST]) as independent variables. Which variables were excluded depended upon which dependent variable was used in the model. For example, when pathogen fitness (W [PATHOGEN]) was the dependent variable, Wp [PATHOGEN], Wc [PATHOGEN], W [PATHOGEN] were dropped. Variables Wp [HOST], Wc [HOST] and W [HOST]) were dropped from the model when host fitness (W [HOST]) was the dependent variable.

For each of the varieties, 4 specific models were built. The first was meant to find the best combination of independent variables that estimated pathogen fitness (W [PATHOGEN]). The second model was meant to predict host fitness (W [HOST]). The last two equations were similar to the first two, except that only variables known to be controlled by genetic differences among treatment sporidia were used as independent variables. Note that for Trebi and Odessa, the independent terms in the

models can differ markedly.

7.7.2 TRADITIONAL

This series of models compared the two best known methods of assessing disease damage that involve variables believed to be correlated with pathogen fitness (tables 34 to 36). The two methods are the percent smutted plants (R2), currently the most popular and commonly used, and, the percent smutted heads (R4). Reliability of using these same two independent variables to estimate host fitness was assessed. The effectiveness of combining these two variables as estimators of fitnesses was investigated.

7.7.3 PRACTICAL

This series of models was arranged expressly to investigate certain combinations of independent variables associated with practical and technical aspects of performing this experiment (tables 37 to 41). The models were divided into 3 subgroups:

- MINIMAL COST
- MODERATE COST
- EARLY ASSESSMENT

The first subgroup (MINIMAL COST) involves independent variables that were obtained with minimal cost, in terms of man hours, equipment and financing. The MODERATE COST models

incorporate variables used in the MINIMAL COST model plus a few others which were obtained at a moderate cost. The EARLY ASSESSMENT model tests the adequacy of using certain variables, obtainable well in advance of harvest, as accurate predictors of host and pathogen fitnesses.

7.7.4 DEVELOPMENTAL

Models involving independent variables associated with, or reflecting, sequential stages in the development of the host/parasite association comprised the DEVELOPMENTAL group of equations. These models were used in an attempt to identify particular stages of development in the host which might be associated with physiologic mechanisms affecting host and pathogen fitnesses (tables 42 to 47). There were 2 subgroups of models within this group:

- C (COMPLETELY DISEASED PLANTS) OR H (HEALTHY PLANTS) BASED
- P (PARTIALLY DISEASED PLANTS) BASED: HOST PERSPECTIVE
- P (PARTIALLY DISEASED PLANTS) BASED: PATHOGEN PERSPECTIVE

C (COMPLETELY DISEASED PLANTS) OR H (HEALTHY PLANTS) BASED models incorporated independent variables from either the C (completely diseased plants) or the H (healthy plants) subset of variables depending on which dependent variable was involved. When the dependent variable was W [PATHOGEN] (pathogen fitness), C (completely diseased plants) based, pathogen related,

independent variables were used. Similarly, H (healthy plants) based, host fitness related independent variables were needed when W [HOST] (host fitness) was the dependent variable. The second subgroup of models involved independent variables from the P (partially diseased plants) subset. These variables were either host related or pathogen related.

7.8 "CONSTANT RANKING"

The Spearman rank correlation coefficient, r, for the ranking of the 20 dikaryons on Trebi and Odessa was positive, high and significant (r=0.8714, P=0.0001) for percent smutted plants (R2, table 48). When disease levels were represented by percent smutted tillers the Spearman correlation coefficient was 0.8526 (P=0.0001). Two other variables, used for ranking, generated significant correlations: pathogen fitness on partially diseased plants (Wc [PATHOGEN]) and pathogen fitness on completely diseased plants (W [PATHOGEN]). Rank correlation of pathogen fitness values with host fitness values was not statistically significant on either Trebi or on Odessa (table 49).

8 DISCUSSION

8.1 BIOLOGICAL MATERIAL

Varieties Trebi and Odessa are sufficiently dissimilar genetically, that their reactions to smut isolates differ markedly. Odessa possesses no known specific resistance, while Trebi has specific resistance to race 7, among others (Tapke, 1937, 1945). When challenged with specific virulence genes, Trebi can exhibit differential interactions (Tapke, 1937, 1945). When the two varieties show susceptibility to the same isolates, Trebi invariably has a higher, quantitative level of disease. Non-specific genetic differences were found to control this variation in disease levels (Pope, 1982).

All treatment dikaryons used in this experiment possessed the dominant virulence allele conferring pathogenicity on Trebi (Person, 1983). No known virulence genes are required for pathogenicity on Odessa. Therefore, all treatment dikaryons had the ability to produce disease damage on both varieties. The level of disease damage caused by any dikaryon depended on the combined effects of nonspecific pathogenicity and nonspecific resistance.

8.2 SPORIDIAL TREATMENTS VERSUS CONTROL COMPARISONS

Effects of sporidial treatment common to both varieties were: a reduction in the number of healthy plants and heads, a reduction in tillering of completely and partially diseased plants, a dramatic reduction in seed number, weight and germination rate for partially diseased plants, and a decrease in healthy plant fitness. These effects were present regardless of the resistance genotype of the host. It appears that all plants, diseased or apparently healthy, suffered reductions in seed biomass and reproductivity.

Since all plants of one variety were homozygous for nonspecific resistance, it was expected that each plant would have the same probability of showing disease signs. The fact that not all plants in a row produced spores indicates that some factor(s), other than plant genotype, was important in controlling the probability of a susceptible plant becoming diseased. The factors most likely to be involved are pathogen genotype, and environmental influences, random events and/or combinations of these.

A plausible explanation for disease escape is that an important, as yet unidentified event(s), during a critical period(s) of host development retards, excludes, or removes the smut fungus. Should a dikaryon have the genetic means to escape, avoid or prevent these event(s) it can produce teliospores. A dikaryon could continue to grow and act as a physiological sink and could eventually show signs of its presence by producing teliospores.

Quantitative dissimilarities between the varieties were noticed upon comparison of several variables. Trebi had fewer statistically different departures from control means than Odessa. For Odessa these departures included: a decrease in the number of treated seeds that germinated and reached maturity, and, for healthy plants, a reduction in tillering, seed production and seed weight. Also, a large decrease in host fitness for Odessa was recorded.

Seeds from healthy Trebi plants suffered a reduced germination rate. No such reduction was observed for Odessa. This result would have a nontrivial impact on the contribution of healthy Trebi plants to the next generation (ie. fitness), relative to uninoculated plants. Treatment of Odessa caused several effects to occur that were not seen on Trebi.

Following planting, but prior to maturation, inoculated Odessa seeds showed a pronounced reduction in rate of germination compared with seeds of Trebi. It is hypothesized that Trebi possesses genetically conferred resistance that manifests itself at some time following germination but prior to maturation. It is most likely that this resistance becomes effective soon after germination because there were no immature plants present at harvest and no obvious deaths of immature plants prior to harvest.

Also, genetic dissimilarities between Trebi and Odessa are considered to be responsible for causing healthy plants to have lower tillering, seed production and seed weight than control plants. Trebi did not suffer large reductions in these fitness

related variables.

It appears that the plant may pay a price for resisting the disease in order to remain healthy (Person, personal communication). In an infected plant a "... greatly increased biosynthetic activity occurs at the expense of stored host energy and may ultimately limit plant growth and yield relative to potential growth and yield" (Smedegaard-Petersen, 1985).

An apparent discrepancy was found in the Trebi data. The mean number of healthy plants and heads from healthy plants was different than the mean number for control. Since the average number of heads per healthy plant was similar to that for the control, it was expected that, either a significant decrease in the total number of seeds from healthy plants or an increase in the average seed number per healthy plant, would occur. Neither of these situations happened, indicating a possible Type II error in one, some or all of the following variables: the average number of heads per plant (TH3), the average number of seeds per plant (TH4), the average number of seeds per head (TH5), the total number of seeds (TH10). Two variables, the average number of heads per plant (TH3) and the total number of seeds (TH10), had T(calc) values that were very close to the T(tab) value and were most likely the ones to have been involved in a Type II error. Based on these facts, the parsimonious explanation for this discrepancy involves only 1 of these suspected variables in a Type II error, namely TH10.

8.3 VARIABLE MEAN COMPARISONS FOR THE VARIETIES

Interestingly, statistically significant differences were found when comparisons were made between variable means. There were large differences among the plant types (ie. healthy, completely diseased and partially diseased). There also were striking similarities between the two varieties in terms of relationships between certain variables (compare tables 19 and 21). The only differences between the varieties were for plant type numbers and average tiller numbers. These differences are expected and are consistent with the genetic dissimilarity between the two varieties.

In terms of fitness, completely diseased plants make a larger contribution to pathogen fitness than partially diseased plants. This is explained by a combination of events: a greater number of completely, as compared with partially diseased plants (for Odessa only), a greater number of diseased tillers per plant for completely diseased plants, a greater average spore weight per tiller for completely diseased plants and finally, a larger average spore germination rate for completely diseased plants.

Host fitness values calculated from healthy plants were larger than those from partially diseased plants because of: a larger number of healthy plants, a greater average number of healthy tillers per plant, a larger average number of seeds per tiller and a larger average seed germination rate.

The average seed weight per tiller was greater for healthy plants than for partially diseased plants. Although not

strictly related to host or pathogen fitness, as defined in this study, this difference is believed to be important in relation to the quality of seed set and the yield at harvest. The presence of inoculum at the time of planting may not lead to the production of large amounts of teliospores on partially diseased plants but does cause a dramatic depression of expected yield for partially diseased plants. From these data, it can not be determined if the pathogen, in diseased tillers of partially diseased plants, acts as a metabolic sink and reduces healthy tiller seed weight or if the pathogen continues to live and grow in tissue of healthy tillers.

8.4 ANOVA

Six possible components of variation were measured for all variables. These components were

- "+" sporidia main effects (corresponds to nonspecific pathogenicity)

- "-" sporidia main effects (corresponds to nonspecific pathogenicity)

- "rep" replicate effects (corresponds to environmental differences among blocks)

- "+x-" sporidia interaction (corresponds to dominance and epistatic interaction of pathogenicity genes)

- "+xrep" sporidia by replicate interaction (corresponds to genotype by environment interaction)

- "-xrep" sporidia by replicate interaction (corresponds to genotype by environment interaction)

Forty variables from Trebi and 41 from Odessa, with 30 of these held in common, revealed statistically significant

differences for, at least, one source component. For each of the 3 main effect components, "+", "-", "rep", and the "+x-" interaction component, significant F values appeared in 1, 23, 21 and 7 variables, respectively, on Trebi and 1, 17, 32 and 8 variables, respectively, on Odessa. There were more variables for Odessa than for Trebi where differences among replicates were important in affecting variability. For Trebi, more variables were affected by differences among the "-" sporidia than for Odessa.

Replicate differences ("rep") appeared more frequently in ANOVA's than any of the other 6 components of variation. Replicate differences reflect the effect of environmental heterogeneity on variability. Replicate blocks were handled as similarly as possible and were placed in field locations which were as uniform as possible. Some interplot differences were evident. The experimental field had a slope of about 2 degrees from south to north. A clay hardpan existed at depths varying from approximately 20 to 30 cm below soil surface. These two factors could have affected water drainage and might be the most important environmental factors contributing to variability. Other factors that might have contributed to replicate related variability include: technical (seed preparation, inoculation, planting, etc.), fertilizer distribution, plant density, soil dwelling organisms, and above soil organisms (particularly mildew and barley yellow-dwarf). Significant F values for the replicate component appeared in 13 variables for Trebi and 24 for Odessa (2 were common to both varieties). Environmental

factors played a larger role in generating variability for variables on Odessa than on Trebi.

Genetic differences among treatment sporidia also were an important factor in generating variability. The most frequently occurring genetic component was the "-" mating type component and represents the nonspecific pathogenicity gene differences among sporidia. A large F value was found for the "-" component only, for 15 variables on Trebi and for 4 on Odessa, 2 of which were held in common. Genetic differences among sporidia were more important for producing variability on Trebi than on Odessa.

A total of 26 variables on Trebi and 18 on Odessa was shown by ANOVA to be controlled, to some extent, by nonspecific pathogenicity genes. It is interesting to note that for Trebi these variables were mainly from the R (row), C (completely diseased plants), and P (partially diseased plants) subsets indicating the possibly consequential involvement of these genetic differences in determining host fitness. For Odessa, mainly the R (row) and C (completely diseased plants) subsets had variables with important genetic components. This indicates involvement of nonpathogen related factors in partially diseased plants, possibly heretofore undetected host resistance.

No variable was found where the "+" sporidia component was the sole source of variability. It is highly probable that nonspecific pathogenicity gene(s) are tightly linked to the mating locus, coupled with the "-" mating allele. This result is consistent with that found in an earlier studies (Pope, 1982;

Caten et al, 1984).

The fact that genetically related components for the percent of plants smutted (R2) and for pathogen fitness (W [PATHOGEN]) were significant on Trebi but not on Odessa permits interesting speculation. Genetic differences among dikaryons were observed on Trebi but not on Odessa. Nonspecific pathogenicity genes segregating in the dikaryons were effective on Trebi but not on Odessa.

According to expectations based on Vanderplank's definition of horizontal genes (1982, 1984), in the absence of gene-for-gene interactions all nonspecific pathogenicity genes are effective against all nonspecific resistance genes regardless of their origin or number. Results from this experiment deviate from these expectations. Smut dikaryons are more variable on Trebi than on Odessa which indicates that race 11 (parental teliospore T1) is better adapted to Trebi than to Odessa. Adaptation to Trebi is possibly a result of selection in race 11 for pathogenicity alleles that optimize interactions on Trebi. The history of race 11 is not available. Therefore, this possibility can not be verified.

The effects of the Trebi related "subset" of nonspecific pathogenicity alleles are not present on Odessa. Other reasons for this reaction on Odessa can include the following: Odessa might have the unusual dynamic capacity to interact with the isolates in strengths directly related to the aggressiveness level of each isolate, thereby causing isolates to appear to be genetically identical, or the universal suscept could be devoid

of active resistance polygenes, negating the interaction of pathogenicity and resistance genes (this assumes that these interactions are a prerequisite to the resolution of nonspecific genetic differences among isolates). The first situation is inconsistent with the definition of nonspecific polygenes. The second situation is not supported by experimental evidence here or in the literature.

Interactions among pathogen isolates of the same race and susceptible hosts, similar to those found here, were recorded in the potato-late blight system (Bruyn, 1947; Jeffrey et al, 1962; Caten, 1974) and in the barley-rust system (Clifford and Clothier, 1974; Parlevliet, 1978). These researchers concluded that pathogenic strains may be specifically adapted to varieties from which they were isolated.

There is one major difference between those investigations and this one. For each of those investigations the hosts were consistantly of one type, in terms of specific resistance. They were either without known specific resistance (ie. R0 hosts; Caten, 1974) or they had identical but defeated specific resistance genes (Clifford and Clothier, 1974). In this study, one host (Odessa) was without known specific resistance and the other (Trebi) had a defeated specific resistance gene. This difference plays a non trivial role in the following interpretation.

The presence of a defeated specific resistance gene in the background of Trebi but not in the background of Odessa could be an important determinant of how, when or if the segregating

nonspecific pathogenicity genes function. The different specific gene backgrounds could be involved in generating statistically significant differences among dikaryons for certain variables but not others. Consequently, measurements of host and pathogen reproductivities and fitnesses would differ markedly for the two varieties.

The nonspecific pathogenicity genes could be operative (ie. active in host-pathogen interactions and statistically measurable) only when a certain matching specific resistance/virulence gene combination are present in the background. The nonspecific pathogenicity genes might not adapt an isolate to any variety. They might adapt an isolate to a host variety with a certain defeated specific resistance gene. This variety or background specificity is not the same as, and should not be confused with, gene-for-gene specificity. These conclusions are supported by subsequent analyses described in a later section.

One can only speculate as to how widespread this type of interaction is in any pathosystem. Perhaps every specific gene has its own subset of modifying nonspecific genes; perhaps only a few do. Can a nonspecific gene be a member of more than one subset? Must a nonspecific gene necessarily be a member of any subset. Many other pertinent questions are raised.

Conceivably, under the right conditions, interactions described here could exhibit a quadratic check. Since the check is evidence for a gene-for-gene interaction (Person, 1959; Ellingboe, 1981), care should be taken not confuse these

interactions with those generated by genuine gene-for-gene interactions. Exactly how these interactions will affect the Vanderplankian definition of nonspecific pathogenicity genes and "constant ranking" awaits elucidation.

It is possible that some varieties with long lived resistance which is known to be specific (vertical) in nature, are actually protected from severe damage from compatible pathogenic genotypes because of the absence of disease enhancing alleles within the appropriate subset of nonspecific genes. Individuals in the pathogen population would have to undergo many mutational events, each with a low probability of occurance, to develop the ability to produce severe disease damage.

Further investigation of the interaction between pathogenicity and resistance genes, in this and other systems, should be undertaken especially in view of its involvement in some durably resistant natural and crop pathosystems. As well, useful information could be obtained from studies of the molecular and physiological nature of the interactions.

The following combinations of significant components existed: "-" with "rep" (3 for Trebi, 4 for Odessa, 1 in common), "-" with "+x-" (1 on Trebi, 5 on Odessa), "rep" with "+x-" (1 on Trebi), "+", with "-", and "rep" (1 on Trebi, 1 on Odessa), and, "-", with "rep" and "+x-" (3 on Trebi, 3 on Odessa).

There were no instances of significant contributions to variability by the "+" component alone or by the following

combination of components: "+" with "-", "+" with "rep", "+" with "+x-", "+" with "rep" and "+x-", and, "+" with "-", "rep" and "+x-".

Plus by minus sporidia was the only interaction component that was important in causing variability, with one exception, "-xrep" on Trebi. A significant "+x-" interaction indicates that specific sporidia of the "-" mating type in combination with certain sporidia of the "+" mating type generate a relatively sizable amount of variability because of epistatic and/or dominance interaction. The interactions can be of two types, synergistic or interference (Sokal and Rohlf, 1981).

Most significant "+x-" interactions occurred in association with other significant components, particularly with "-" main effects. Care should be taken when interpreting such results. The simultaneous occurrence of significant differences among "-" sporidia and "+x-" interactions for a variable does not necessarily mean that both components are important contributors to variability. Finney (1947) stated that significant interaction effects ("+x-" interactions) are intimately tied with the main effects components (ie. "+" and "-" main effects). A significant "+" and/or "-" component may be an artifact of a significant "+x-" interaction. Therefore, testing for the significance of the main effects might not be very meaningful (Gilbert, 1973; Sokal and Rohlf, 1981). If the significant "+x-" interaction was absent, significant main effects components might disappear too, especially when the main effects F values are close to the critical F value. When the difference

between the interaction and a main effects F value is large, so is the probability that the main effects component is not an artifact of the interaction. This situation applies not only to the "+x-" interactions but also to any first-order interaction.

Two variables on Trebi revealed only significant "+x-" effects. There were none on Odessa.

8.5 MODELLING

At the start of this section it should be noted that where possible, models involving independent variables with known statistically significant genetic components are favored over models where some or all independent variables have no statistically significant components. Models with one or more independent variables without significant genetic components generally have large environmental components. Such variables are unreliable for inclusion in prediction related models because they are affected by uncontrolled environmental factors.

8.5.1 COMPLETE

Tables 32 and 33 show that three independent variables chosen by the stepwise program predict pathogen fitness (W [PATHOGEN]) on Trebi. These were total spore weight (R12), the number of completely smutted plants (C1) and the average spore weight per tiller (P10). Together they account for 99.1% of the variance in the dependent variable. On Odessa, 98.4% of the variability of pathogen fitness was accounted for by three

variables: the average number of diseased heads per plant (R7), the number of heads from completely smutted plants (C2) and the total spore weight from completely diseased plants (C4).

Host fitness (W [HOST]) was weakly correlated with pathogen fitness on completely diseased plants (Wc [PATHOGEN]) on Trebi ($R^2=0.253$). On Odessa three variables, the percent of plants smutted (R2), the percent of heads smutted (R4) and the number of completely diseased plants (C1) generated on R^2 value of 0.674.

From this group of models it appears that nonspecific genetic differences among treatment sporidia played an important role in controlling pathogen fitness but not host fitness. The correlation between host and pathogen fitnesses was -0.437 (P=0.0538) for Trebi and -0.231 (P=0.3280) for Odessa, both not statistically significant. Most pathogen isolates will depress host fitness (yield) to a certain extent, but an isolate with high fitness will not necessarily depress host fitness more than one with low fitness.

These results are based on the definition of fitness provided in the RESULTS section of this study. The lack of a high positive correlation between the fitnesses of the interacting organisms does not mean that the correlation between their reproductivities is necessarily insignificant. In fact, the r value for spore number (R12) vs seed number (H10) is -0.466 (P=0.0383) and for percent smutted plants (R2) vs seed number (H10) r is -0.447 (P=0.0481) on Trebi. The same correlations on Odessa are -0.317 (P=0.1736) and -0.423

(P=0.0633).

The significant correlations on Trebi indicate that in a farmer's field a large increase in the number of spores (ie. pathogen reproductivity) and/or in the number of smutted plants (ie. disease damage level), will result in a reduction in host reproductivity. No such relationship exists for Odessa. These correlations between reproductivities do not involve any measure of the ability of the spores or the seeds to germinate and survive for the next season.

Similar results were obtained by Hoy, Hollier and Fontenot (1985) in the smut-sugarcane system. They found a highly significant correlation between levels of smut infection and sugarcane yield. Smut reduced the number of healthy canes in diseased plots.

For Trebi the two interpretations of these data seem contradictory at first. In terms of fitness as defined in this study there is no significant correlation between fitnesses of the interacting organisms. In terms of reproductivity (a component usually referred to as fitness by pathologists and epidemiologists), the correlation is significant and negative.

It appears that, on Odessa, selection does not favor high fitness or high aggressiveness in isolates (statistically speaking). On Trebi, extending the definition of fitness to include aspects of post-harvest survival which are not involved in or implied by the definition of aggressiveness or reproductivity, causes the correlation between the fitnesses of the organisms to disappear. This disappearance indicates that

the most aggressive pathogenic isolates will not necessarily contribute relatively more to the establishment of the epidemic in the next season than less aggressive isolates. In fact this is very weak evidence for the absence of selection in favor of the most aggressive isolates (ie. those with the highest reproductivities), or in favor of the least aggressive isolates. All isolates would be considered equally fit based on the definition of fitness used in this study.

There are two additional, less weak, explanations for this phenomenon that stem from the fact that the reproductivity and fitness correlations on Trebi are both borderline in terms of their probabilities. It is possible that a statistical error occurred or that the tests had insufficient power to find both r values significant.

8.5.2 TRADITIONAL

Traditionally, two methods of measuring disease damage have been used in the Ustilago hordei-Hordeum vulgare host-parasite system and both are believed to be highly correlated with pathogen fitness. The methods are: the percent smutted plants and the percent smutted tillers. The former, at present is the one most commonly employed. This group of models was constructed to compare and contrast the two methods and to determine their usefulness for estimating host and pathogen fitnesses (tables 34-36). The accuracy of estimating pathogen fitness on Odessa using the percentage smutted plants (R2) was poor ($R^2=0.57$), whereas, on Trebi, it was much better ($R^2=0.84$).

The correlation between percent smutted plants (R2) and pathogen reproductivity (R12, aggressiveness) was 0.815 (P=0.0001) and 0.902 (P=0.0001) on Odessa and Trebi, respectively. In my opinion, a measure of the percent smutted plants provides a reliable estimate of pathogen fitness on Trebi but a less reliable one on Odessa; at least for the dikaryons involved in this study. The high positive correlation between percent smutted plants and pathogen fitness was expected in light of the fact that both had statistically significant genetic components. In short, nonspecific pathogenicity genes segregating in the sporidia can elicit a response in pathogen fitness. Host fitness, on the other hand, is poorly represented or estimated on both varieties by percent smutted plants (R2, $R^2 < 0.16$).

The independent variable, R4 (percent smutted tillers), is marginally better than R2 as an estimator of pathogen fitness on Odessa ($R^2 = 0.600$) and slightly better on Trebi ($R^2 = 0.905$). As estimators of host fitness, both R4 and R2 alone are poor ($R^2 < 0.24$). The percent plants smutted (R2) and the percent heads smutted (R4) combined act as a moderately accurate predictors of host fitness (W [HOST]) on Odessa ($R^2 = 0.541$).

8.5.3 PRACTICAL

8.5.3.1 MINIMAL COST

This series of models (tables 37-38) was developed to determine if certain easily obtained independent variables would provide good estimates of fitness for both organisms. Five variables were classified as being easily obtainable: germination rate of treated seeds (R5), percentage smutted plants (R2), total heads in the row (R3), percentage smutted heads (R4) and the total number of tillers from diseased plants (R5).

From these five independent variables, only 1 model, involving the percent of heads smutted (R4) offers an acceptable estimation of pathogen fitness on Odessa ($R^2=0.600$). The R4 (percent of heads smutted) variable turned out to be the only independent variable involved in the "best" model for predicting pathogen fitness on Trebi ($R^2=0.905$). Host fitness (W [HOST]) is poorly predicted by percent of heads smutted (R4, $R^2=0.238$) on Trebi and moderately well by the combination of percent plants smutted (R2) and percent heads smutted (R4) on Odessa ($R^2=0.541$).

8.5.3.2 MODERATE COST

The MODERATE COST models (tables 39-40) promise better estimates of fitnesses because of the inclusion of independent variables along with those already in the MINIMAL COST models.

Two models involving independent variables with large genetic components were noteworthy. Pathogen fitness assessed on Trebi was estimated adequately (R^2=0.959) by the average number of diseased tillers per plant (R7) and the number of tillers from completely diseased plants (C2). On Odessa 79.6% of the variability of pathogen fitness (W [PATHOGEN]) was explained by the average number of diseased heads per plant (R7). Host fitness (W [HOST]) is poorly predicted by the percent heads smutted (R4) and the average number of diseased heads per plant (R7) on Trebi (R^2=0.384). Host fitness (W [HOST]) is predicted moderately well on Odessa (R^2=0.674) by the percent plants smutted (R2), the percent heads smutted (R4) and the number of completely diseased plants (C1).

8.5.3.3 EARLY ASSESSMENT

Testing the feasibility of accurately estimating fitnesses with pre-harvest variables was the objective of the EARLY ASSESSMENT models (table 41). Approximately 84.0% of pathogen fitness on Trebi was accounted for by the percent smutted plants (R2). On Odessa, the percent smutted plants was attributed with producing only 57.0% of the variability. Variable R2 can be used to estimate pathogen fitness on Trebi prior to harvest, provided that for every smutted plant at least one smutted head emerges from the boot and is easily scored. Heads emerged from the boot within a relatively short span of time prior to harvest, making this method of estimating pathogen fitness a potentially useful time saver.

Host fitness (W [HOST]) models involved independent variables with no statistically significant genetic component and were considered unreliable.

It is estimated that for any growing season, between 2 to 4 weeks of time can be saved by estimating fitnesses using the predictor variables described above.

8.5.4 DEVELOPMENTAL

Unidentified developmental events in the interaction between the host and the pathogen were expected to influence pathogen fitness. Developments within the infected host that leads to disease expression are not yet separable into discrete events or stages. The elucidation of physiological mechanisms is not yet possible.

8.5.4.1 C (COMPLETELY DISEASED PLANTS) OR H (HEALTHY PLANTS) BASED

The average spore germination rate per tiller (C7) was the only variable among those tested that made a significant contribution to pathogen fitness on Trebi (table 42-43). This variable is related to the last step measurable in the developmental sequence of events which might have an effect of the fitnesses of the interacting organisms. Any number of events occurring between the time of inoculation and the germination of seeds from treated plants could have influenced pathogen fitness. Obviously, this result does not implicate any

specific physiological event as being involved in controlling or affecting pathogen fitness. The fact that this variable was influenced by the pathogen genotype complicates the interpretation of this result. The genetic differences among dikaryons could have been the major cause of the differences in spore germination rate. It is not clear if physiological events initiated by, mediated by, or involving the host, affected spore germination rate.

On Odessa, an event or events leading up to the production of spores (average spore weight per plant, C5) regulates pathogen fitness levels. The independent variable C7 was not included in the equation indicating that the germination rate of the spores is not an important factor in determining pathogen fitness on Odessa. Also, it implies that the environment Trebi provides for the dikaryon is different than that provided by Odessa and that the genetically heterogeneous dikaryons react very differently to the two host environments. The conclusion reached in a previous section concerning nonspecific pathogenicity gene subsets is supported by these results.

No models accurately predict host fitness (W [HOST]) on either variety.

8.5.4.2 P (PARTIALLY DISEASED PLANTS) BASED: HOST PERSPECTIVE

Average seed germination rate for partially diseased plants (P18), which has a large genetic component for the control of pathogen fitness on Trebi, does not provide a definite clue as to where, developmentally, the genetic differences among

dikaryons manifest themselves (tables 44-45). On Odessa, the average number of tillers per partially diseased plant (P5), which has no genetic component, suggests that some plant mediated event(s) leading to, and/or involving, the determination of tiller number, affects pathogen fitness. As a universal suscept, Odessa should be attacked by all dikaryons. This does not imply that Odessa is without any measure of resistance. It appears that Odessa has a barely discernable level of resistance that is operative early, following infection. It is obvious that this resistance is weak and inconsistent, by virtue of the fact that significantly more Odessa plants can become completely diseased than partially diseased.

8.5.4.3 P (PARTIALLY DISEASED PLANTS) BASED: PATHOGEN PERSPECTIVE

Genetic differences among dikaryons for spore germination (P11), on partially diseased Trebi, suggest no specific early events influencing pathogen fitness (tables 46-47). On Odessa variables in the model had no statistically significant genetic components.

No models predicting host fitness were generated.

8.6 "CONSTANT RANKING"

Wehrhahn (1986, personal communication) believes the term "constant ranking" is not "operationally useful" because it implies a rigid consistency in rank order, a difficult condition to find in practice, especially when large confounding environmental and random error effects are possible. In practice ranking can involve occasional rank reversals of near neighbors in an array of interacting genotypes. The term "concordant ranking" was suggested by Wehrhahn as an alternative for "constant ranking" because the new term, statistically speaking, implies the possibility of less rigidity. The term "concordant" is derived from the field of statistics (ie. Kendall's concordant correlation).

"Constant (concordant) ranking" is based on the assumption that pathogen reproductivity is negatively correlated with host reproductivity. "Constant (concordant) ranking" ignores effects of pathogen damage to the host. More precisely, it ignores possible attendant reduction in host reproductivity. "Constant (concordant) ranking" also ignores the possible presence of tolerance. Tolerence is a component of horizontal resistance. A tolerant plant can sustain a certain level of disease and still have a high yield, while a less tolerant plant can have the same level of disease and a lower yield.

In the barley-smut system, "constant (concordant) ranking" occurs for disease damage variables: percent smutted plants (R2), percent smutted tillers (R4) and pathogen fitness (Wc [PATHOGEN] and W [PATHOGEN]; table 48). Although the genetic

differences among dikaryons were not large enough to generate significant F values on Odessa, the dikaryons still maintained a rank order that was highly correlated with that on Trebi. Current evidence for the presence of "constant (concordant) ranking" in this system, supports an earlier finding, the first ever recorded, involving a select population of T1 x T4 descendents (Person et al. , 1983).

Host fitness values (Wp [HOST], Wh [HOST] and W [HOST]) were tested for compliance with the fundamental concept of "constant (concordant) ranking". No significant rank correlation resulted.

Ranking of host and pathogen fitnesses was tested on Trebi and on Odessa with negative results (table 49). Therefore, pathogen fitness rankings can not be used to rate expected host performance (fitness or yield). This finding is not surprising because of the poor performance of pathogen fitness values in predicting host fitness values, in the models discussed earlier.

Regardless of how common polygene subsets targeting different varieties or specific gene backgrounds are, the concept of "constant (concordant) ranking" is still valid. Imagine a situation where an isolate with a subset of pathogenicity polygenes can target variety A. The same isolate also might have a second subset of polygenes targeted for variety B. The genes in both subsets are not necessarily mutually exclusive. That is, a particular gene can be a member of both subsets and can be functional and contribute to pathogenicity on both varieties. Under the simplifying

assumption of equality in magnitude of allele action, ranking of varieties will still occur. On the other hand, if the numbers, and direction and magnitude of action of polygenes in the 2 subsets differ greatly, then the varieties may not display "constant (concordant) ranking".

Another important issue concerning "constant (concordant) ranking", peripheral to the present study, but still an integral complication with its use, is the method of ranking. Jenns et al (1982) and Jenns and Leonard (1985) also recognized that problems with ranking can occur. It is my opinion that because of the use of phenotypic pathogenicity values (disease level values) for ranking, ranking according to nonspecific genotype is not as accurate as it could be. This ties in with Wehrhahn's belief, described earlier concerning the lack of rigidity of ranking. Ranking based on additive gene effects (breeding values) would be more appropriate. The confounding effects of superfluous genetic (dominance and/or epistatic interaction) and nongenetic (environmental and other interaction) effects, known to be associated with phenotypic values, would be excluded from ranking. In other words, ranking of disease phenotypes is expected to be less accurate for assessing host or pathogen performance than ranking of additive gene effects. This is a novel idea to the field of host-pathogen interactions and warrants further consideration.

If we expect to reduce pathogen induced host yield losses with effective host management strategies, all theoretical and practical information concerning every aspect of host-parasite

interactions should be made readily available to breeders.

9 SUMMARY

The following is a summary of the important conclusions reached and hypotheses constructed in this study. These conclusions and hypotheses have been divided into three groups according to the objectives described in the PURPOSE section. Conclusions and hypotheses that are new to the field of host-pathogen interactions or to the _Hordeum vulgare_-_Ustilago hordei_ system, in particular, are suffixed with the term "[DISCOVERY]". Conclusions that support previously reported results are suffixed with the term "[CORROBORATIVE FINDING]".

1 Some fitness related variables measured on treated rows differed significantly from those measured on untreated control rows. [DISCOVERY]

   1.1 Comparison of fitness related variables indicated that the two varieties reacted in dramatically different ways to the dikaryons. [CORROBORATIVE FINDING]

   1.1.1 The traditional measure of the level of disease damage (percent plants smutted, R2) in this system was found to be a reliable estimator of pathogen fitness on Trebi and reproductivity on both varieties. The other less frequently used measure (percent heads smutted, R4), was a slightly better estimator. [DISCOVERY]

   1.2 Inoculation with the pathogen caused reduced host fitness in both diseased and healthy plants. [DISCOVERY]

   1.2.1 A statistically significant negative correlation was found between the reproductivity of the host and the pathogen on Trebi. [CORROBORATIVE FINDING]

   1.2.2 There was no significant correlation between host and pathogen fitnesses (as defined in this study) on either variety. [DISCOVERY]

1.3 Individual selection favored neither high nor low reproductivity (aggressiveness) on Odessa. [DISCOVERY]

    1.3.1 Nonspecific pathogenicity gene differences among dikaryons indicate that selection ("s") values can be calculated for these data for future modeling experiments. [DISCOVERY]

1.4 Modelling

    1.4.1 Neither traditional method of measuring disease damage level should be used to predict host fitness. [DISCOVERY]

    1.4.2 Three variables, the total spore weight (R12), the number of completely diseased plants (C1) and the average spore weight per head (P10) on Trebi, and three variables on Odessa, the average number of diseased heads per plant (R7), the number of heads from completely diseased plants (C2) and the total spore weight from completely diseased plants (C4), can be used as reliable predictors of pathogen fitness (W [PATHOGEN]). [DISCOVERY]

    1.4.3 The variables, percent plants smutted (R2), percent smutted heads (R4) and the number of completely diseased plants (C1) should produce moderately accurate predictions of host fitness (W [HOST]) on Odessa. [DISCOVERY]

    1.4.4 Variables collectable with minimal cost provide acceptable estimations of fitnesses. Specifically, the percent heads smutted (R4) produce accurate and moderately accurate predictions of pathogen fitness on Trebi and on Odessa (respectively). The combination of the percent plants smutted (R2) and the percent heads smutted (R4) on Odessa produce moderately accurate estimates of host fitness (W [HOST]). [DISCOVERY]

    1.4.5 Variables collectable with a moderate cost provide even more accurate predictors of pathogen fitness (W [PATHOGEN]). These variables are the average number of diseased heads per plant (R7) and the total number of heads

from completely diseased heads (C2) on
Trebi and the average number of diseased
heads per plant (R7) on Odessa. The
percent plants smutted (R2), the percent
heads smutted (R4) and the number of
completely diseased plants (C1) are
moderately accurate at predicting host
fitness on Odessa (W [HOST]). [DISCOVERY]

1.4.6 Under conditions of early head emergence,
a preharvest variable, percent smutted
plants (R2), can be collected to provide
time saving, accurate estimates of
pathogen fitness (W [PATHOGEN]) on Trebi
and moderately accurate predictions on
Odessa. [DISCOVERY]

1.4.7 There is little evidence to identify
specific events during the development of
both the host and the pathogen that
affect fitnesses on Trebi and on Odessa.
[DISCOVERY]

2 Statistically significant genetic differences
among dikaryons were displayed for 26 variables
on Trebi and for 17 variables on Odessa. These
differences were attributed to segregating
nonspecific pathogenicity genes with pleiotropic
effects. [DISCOVERY]

2.1 Biometrical analyses uncovered significant
additive gene effects for 15 variables on Trebi
and 4 on Odessa. [DISCOVERY]

2.2 Significant interaction components existed for
many of the fitness related variables (9 on
Trebi and 12 on Odessa) indicating the
importance of dominance and epistatic
interactions. [DISCOVERY]

2.3 Nonspecific genetic differences among
dikaryons played an important role in
controlling pathogen fitness but not host
fitness. [DISCOVERY]

2.4 Environmental (replicate) differences alone,
generated large amounts of variability for 13
variables on Trebi and 24 variables on Odessa.
[DISCOVERY]

2.5 Differential variety reaction to nonspecific
pathogenicity genes indicate that Ebba's
parental teliospore from race 11 was probably

better adapted to Trebi than to Odessa.
[DISCOVERY]

2.6 It is speculated that nonspecific pathogenicity
genes in this biological material may be
targeted to certain varieties or to specific
resistance (vertical gene) backgrounds.
[DISCOVERY]

2.7 A nonspecific pathogenicity gene(s), tightly
linked with the mating locus was revealed
(coupled with the "-" mating allele).
[CORROBORATIVE FINDING]

3 There is "constant (concordant) ranking" of percent
of plants smutted (R2), percent of tillers smutted
(R4) and pathogen fitness (Wc [PATHOGEN] and W
[PATHOGEN]) on the two varieties. [DISCOVERY]

3.1 "Constant (concordant) ranking" of additive
gene effects (breeding values) is suggested.
[DISCOVERY]

10 REFERENCES CITED

Abdalla, M.M.F. and J.G.T. Hermansen. 1971. The concept of breeding for uniform and differential resistance and their integration. Euphytica 20:351-361.

Alexander, M. 1981. Why microbial preditors and parasites do not eliminate their prey and hosts. Annual Review Microbiology 35:113-133.

Andrews, J.H. 1984. Life history strategies of plant parasites. Adv. in Plant Pathology 2:105-130.

Barrett, J. 1985. The gene-for-gene hypothesis:parable or paradigm. In: Ecology and Genetics of Host-Parasite Interactions. Ed: D. Rollinson and R.M. Anderson. Pub: Academic Press. New York. pp. 215-225.

Bassi, F.G.A. and J.H. Burnett. 1980. The genetic architecture of aggressiveness in Ustilago maydis. Ann. Appl. Biol. 94:281.

Batts , C.C.V. and Jeater. 1958. The development of loose smut (Ustilago tritici) in susceptible varieties of wheat, and some observations on field infection. Trans. Brit. Mycol. Soc. 41:115-125.

Bauch, R. 1932 Die Sexualitat von Ustilago scorona-zeae und Ustilago zeae. Phytopath. Zeitschr. 5:315-321.

Biffen, R.H. 1905. Mendel's laws of inheritance and wheat breeding. J. Agric. Sci. Camb. 1:4-48.

Biffen, R.H. 1907. Studies in the inheritance of disease resistance. J. Agr. Sci., Lond. 2:109-128.

Blanch, P.A., M.J.C. Asher and J.B. Burnett. 1981. Inheritance of pathogenicity and characters in Gaeumannomyces graminis var tritici. Trans. Br. mycol. Soc. 77:391-399.

Borlaug, N.D. 1958. The use of multilineal and composite varieties to control airborne epidemic diseases of self-pollinated crop plants. Proc. 1st Int. Wheat Gen. Symp., Univ. of Manitoba, Canada. pp. 12-29.

Borlaug, N.E. 1965. Wheat, rust and people. Phytopathology 55:1088-1098.

Briggs, F.N. 1926. Inheritance of resistance to bunt, Tilletia tritic, in wheat. J. Ag. Res. 32:973-990.

Browning, J.A. and K.J. Frey. 1969. Multiline cultivars as a means of disease control. Annu. Rev. Phytopathol. 7:355-382.

Bruehl, G.W., W.L. Nelson, F. Koehler and O.A Vogel. 1968. Experiments with Cercosporella foot rot (straw breaker) disease of winter wheat. Washington Agriculture Experimental Station Bulletin 694:1-14.

Bruyn, H.L G. de. 1947. Het rassenproblem bij Phytophthora infestans. Vakblad voor biologen 27:147-152.

Caten, C.E. 1974. Intra-racial variation in Phytophthora infestans and adaptation to field resistance for potato blight. Ann. Appl. Biol. 77:259-270.

Caten, C.E. 1979. Quantitative genetic variation in fungi. In: Quantitative genetic variation. Ed: J.N. Thompson, Jr., and J.M. Thoday. Academic Press, New York. pp. 35-59.

Caten, C.E., C. Person, J,V, Groth and S.J. Dhahi. 1984. The genetics of pathogenic aggressiveness in three dikaryons of Ustilago hordei. Can J. Bot. 62:1209-1219.

Chester, K.S. 1946. The nature and the prevention of the cereal rusts as exemplified in the leaf rust of wheat. Chronica Botanica, Waltham, Massachusetts.

Churchward, J.C. 1937-8. Studies on physiologic specialization of the organisms causing bunt in wheat and the genetics of resistance to this and certain other wheat diseases. Royal Soc. N.S.W. 71:362-384.

Clark, J.A., K.S. Quisenberry and L. Powers. 1933. Inheritance of bunt reaction and other characters in Hope wheat crosses. J. Ag. Res. 46:413-425.

Clifford, B.C. 1972. The histology of race-nonspecific resistance to _Puccinia hordei_ Otth. in barley. Proc. Eur. Mediterr. Cereal Rusts Conf. Prague, 1972. I:75-78.

Clifford, B.C. 1975. Stable resistance to cereal diseases: problems and progress. Rep. Welsh Plant Breed. Stn. pp. 107-113.

Clifford, B.C. and R.B. Clothier. 1974. Physiologic specialization of _Puccinia hordei_ on barley hosts with non-hypersensitive resistance. Trans. Br. mycol. Soc. 63:421-430.

Comstock, R.E. and H.F. Robinson. 1964 Estimation of average dominance of genes. In: Heterosis. Ed: W. Gowen. Pub: Hafner Publishing Co., New York.

Day, P.R. 1974. Genetics of Host-Parasite Interactions. Pub: W.H. Freeman and Co. San Francisco. 238 pp.

Deshmukh, M.J. and H.W. Howard. 1956. Field resistance to potato blight (_Phytophthora infestans_). Nature 177:794-795.

Dobzhansky, T. (1950). Evolution in the tropics. Am Sci. 38:209-221.

Driver, C.M. 1962. Breeding for disease resistance. In: Scottish Plant Breeding Station Reports, pp. 1-11.

Ebba. T. 1974. Genetic studies of the host-parasite relationship between _Ustilago hordei_ and _Hordeum vulgare_. Ph.D. Thesis, University of British Columbia, Vancouver, Canada. 140 pp.

Ebba, T. and C. Person. 1975. Genetic control of virulence in _Ustilago hordei_. IV. Duplicate genes for virulence and genetic and environmental modification of a gene-for-gene relationship. Can. J. Genet. Cytol. 17:631-636.

Eide, C.J. and F.L. Lauer. 1967. Testing potatoes for field resistance to late blight. Am. Potato J. 44:9-16.

Ellingboe, A.H. 1975. Horizontal resistance: an artifact of experimental procedure. Aust. Plant Path. Soc. Newsletter 4:44-46

Ellingboe, A.H. 1981. Changing concepts in host-pathogen genetics. Annu. Rev. Phytopathol. 19:125-143.

Emara, Y.A. 1972. Genetic control of aggressiveness in Ustilago hordei. I. Natural variability among physiological races. Can. J. Genet. Cytol. 14:919-924.

Emara, Y.A. and G. Sidhu. 1974. Polygenic inheritance of aggressiveness in Ustilago hordei . Heredity 32:219-224.

Emara, Y.A. and G.W. Freake. 1981. Effect of environment and genotype and their interaction on pathogenicity of Ustilago hordei. I. Parasite- environment effects. J. Heredit. 72:261-263.

FAO. 1981. State of food and agriculture, 1980. Rome, United Nations Food and Agriculture Organization.

Falconer, D.S. 1981. Introduction to Quantitative Genetics. 2nd Edition. Pub: Longman. New York.

Finney, D.J. 1947. Main effects and interactions. J. Amer. Stat. Assoc. 43:566-571.

Fischer, G.W. 1953. Manual of the North American Smut Fungi. Pub: The Ronald Press Co. New York.

Fischer, G.W. and C.S. Holton. 1957. Biological Control of the Smut Fungi. Pub: The Ronald Press co. New York.

Fleming, R.A. 1982. Strategies of cereal rust management: Redesign of an agro-ecosystem to alter its stability properties. Ph.D. Thesis. University of British Columbia, Institute of Animal Resource Ecology, Vancouver, B.C.

Fleming, R.A. and C.O. Person. 1982. Consequences of polygenic determination of resistance and aggressiveness in nonspecific host:parasite relationships. Can. J. Plant Pathol. 4:89-96.

Flor, H.H. 1942. Inheritance of pathogenicity in _Melampsora lini_. Phytopathology 32:653-669.

Flor, H.H. 1947. Inheritance of reaction to rust in flax. J. Agric. Res. 74:241-262.

Flor, H.H. 1955. Host-parasite interaction in flax rust. Its genetics and other implications. Phytopathology 45:680-685.

Flor, H.H. 1956. The complimentary genic systems in flax and flax rust. Adv. Genet. 8:29-59.

Flor, H.H. 1971. Current status of the gene-for-gene concept. Annu. Rev. Phytopathol. 9:275-296.

Freeman, M.F. and J.W. Tukey. 1950. Transformations related to the angular and to the square root. Ann. Math. Statist. 21:607-611.

Frey, K.J., J.A. Browning and M.D. Simons. 1973. Management of host resistance genes to control diseases. Zeit. Pflanzenker. Pflanzensh. 80:160-180.

Gaines, E.F. 1923. Genetics of bunt resistance in wheat. J. Ag. Res. 23:445- 480.

Gaunt, R.E. and J.G. Manners. 1971. Host-parasite relations in loose smut of wheat. I. The effect of infection on host growth. Ann. Bot. 35:1131-1140.

Gilbert, N. 1973. Biometrical Interpretation. Pub: Oxford University Press. London. 125 pp.

Gill, D.E. and B.A. Mock. 1985. Ecological and evolutionary dynamics of parasites: The case of Trypanosoma diemyctyli in the red-spotted newt Notophthalmus viridescens. In: Ecology and Genetics of Host-Parasite Interactions. Ed: D. Rollinson and R.M. Anderson. Pub: Academic Press. New York. pp. 157-183.

Graham, K.M. and W.A. Hodgson. 1965. Effect of major and minor gene interaction on adaptive parasitism in Phytopthora infestans. Phytopathology 55:73-75.

Grant, V. 1975. Genetics of Flowering Plants. Columbia University Press. New York. pp. 514.

Gregory, L. V. 1982. Variation in parasitic fitness within populations of Helminthosporium carbonum race 3. Ph.D. Thesis. Pennsylvania State University, Department of Plant Pathology.

Gregory, L.V., J.E. Ayers and R.R. Nelson. 1984. Effect of host genotype on estimating relative parasitic fitness among populations of Helminthosporium carbonum race 3. Phytopathology 74:1024-1026.

Groth, J.V. Multilines and "super races"; a simple model. Phytopathology 66:937-939.

Groth, J.V. and C. Person. 1976. Estimating the efficiency of partial-vacuum inoculation of barley with Ustilago hordei. Phytopathology 66:65-69.

Groth, J.V., C. Person and T. Ebba. 1976. Relation between two measured of disease expression in barley. Phytopathology 66:1342-1347.

Groth, J.V. and C. Person. 1977. Genetic interdependence of host and parasite in epidemics. Annals of the New York Academy of Sciences 287:97-106.

Groth, J.V. and C. Person. 1978. Smutting patterns in barley and some plant growth effects caused by Ustilago hordei. Phytopathology 68:477-484.

Habgood, R.M. 1973. Variation in Rhynchosporium secalis. Trans. Br. mycol. Soc. 61:41-47.

Habgood, R.M. 1976. Differential aggressiveness of Rhynchosporium secalis isolates towards specified barley genotypes. Trans. Br. mycol. Soc. 66:201-204.

Hamid, A.H., J.E. Ayers, R.D. Schein and R.R. Hill, Jr. 1982. Components of fitness attributes in Cochliobolus carbonum race 3. Phytopathology 72:1166-1169.

Hamid, A.H., J.E. Ayers and R.R. Hill, Jr. 1982. Host x isolate interactions in corn inbreds inoculated with Cochliobolus carbonum race 3. Phytopathology 72:1169-1173.

Hamid, A.H., J.E. Ayers and R.R. Hill, Jr. 1982. The inheritance of resistance in corn to Cochliobolus carbonum race 3. Phytopathology 72:1173-1177.

Harlan, J.R. 1976. Diseases as a factor in plant evolution. Ann. Rev. Phytopathology 14:31-51.

Heagle, A.S. and M.B. Moore. 1970. Some effects of moderate adult resistance to crown rust of oats. Phytopathology 60:461-466.

Heath, M.C. 1985. Implications of nonhost resistance for understanding host- parasite interactions. In: Genetic Basis of Biochemical Mechanisms of Plant Disease. Ed: J.V. Groth and W.R. Bushnell. Pub: APS Press, The American Phytopathological Society. St. Paul, Minnesota. 157 pp.

Hedrick, Philip W. 1983. Genetics of Populations. Pub: Van Nostramd Reinhold Company, New York.

Hoerner, G.H. 1919. Biologic forms of Puccinia coronata on oats. Phytopathology 9:309-314.

Hoy, J.W., C.A. Hollier and D.B. Fontenot. 1985. Effect of smut caused by Ustilago scitamininea on yield of sugarcane in Louisiana. Phytopathology 75:1299. Abstract.

Ingold, C.T. 1973. The biology of fungi. Hutchinson, London.

Istock, C.A. 1982. The extent and consequences of heritable
        variation for fitness characters. In: Population Biology:
        Retrospect and Prospect. Ed: C.E. King and P.S. Dawson.
        Pub: Columbia University Press. New York. pp. 61-96.

Jeffrey, S.I.B., J.L. Jinks and M. Grindle. 1962. Intraracial
        variation in Phytophthora infestans and field resistance
        to potato blight. Genetica 32:323-338.

Jenns, A.E., K.J. Leonard and R.H. Moll. 1982. Stability
        analysis for estimating relative durability of
        quantitative resistance. Theor. Appl. Genet. 63:183-192.

Jenns, A.E. and K.J. Leonard. 1985. Reliability of statistical
        analyses for estimating relative specificity in
        quantitative resistance in a model host- pathogen system.
        Theor. Appl. Genet. 69:503-513.

Johnson, D.A. and R.D. Wilcoxson. 1978. Components of slow
        rusting in barley infected with Puccinia hordei.
        Phytopathology 68:1470-1474.

Johnson, R. 1981. Durable disease resistance. In: Strategies for
        the Control of Cereal Disease. Ed: J.F. Jenkyn and R.T.
        Plumb. Pub: Blackwell Scientific Publications, London.

Johnson, R. 1981. Durable resistance: definition of, genetic
        control, and attainment in plant breeding. Phytopathology
        71:567-568.

Johnson, R. and Law, C.N. 1973. Cytogenetic studies on the
        resistance of the wheat variety Bersee to Puccinia
        striiformis. Cereal Rusts Bull. 1:38-43.

Johnson, R. and Law, C.N. 1975. Genetic control of durable
        resistance to yellow rust (Puccinia striiformis) in the
        wheat cultivar Hybride de Bersee. Ann. Appl. Biol.
        81:385-391.

Johnson, R. and A.J. Taylor. 1972. Isolates of _Puccinia striiformis_ collected in England from the wheat vareities Maris Beacon and Joss Cambier. Nature 238:105-106.

Johnson, R. and A.J. Taylor. 1976. Spore yield of pathogens in investigations of the race-specificity of host resistance. Ann. Rev. Phytopathology 14:97-119.

Johnson, T. 1961. Man-guided evolution in plant rusts. Science 133:357-362.

Katsuya, K. and G.J. Green. 1967. Reproductive potentials of race 15B and 56 of wheat stem rust. Can. J. Bot. 45:1077-1091.

Kniep, H. 1919. Untersuchungan uber den Antherenbrand (_Ustilago violaceae_ Pers.) Ein Beitrag zum sexualitatsproblem. Zeitschr. f. Bot. 11:275-284.

Knutson, K.W. 1962. Studies on the nature of field resistance of the potato to late blight. Am. Potato J. 39:152-161.

Knutson, K.W., C.J. Eide. 1961. Parasitic aggressiveness in _Phytophthora_ _infestans_. Phytopathology 51:286-290.

Kozar, F. 1969. The pathway of infection of _Ustilago_ _hordei_. Can. J. Genet. Cytol. 11:977-986.

Kranz, J. 1983. Epidemiological parameters of plant resistance. In: Durable Resistance in Crops. Ed: F. Lamberti, J.M. Waller and N.A. van der Graaff. Pub: Plenum Press. New York.

Kuhn, R.C., H.W. Ohm and G.E. Shaner. 1978. Slow leaf-rusting resistance in wheat against twenty-two isolates of _Puccinia_ _recondita_. Phytopathology 68:651-656.

Kulkarni, R.N. and V.L. Chopra. 1982. Environment as the cause of differential interactions between host cultivars and pathogenic races. Phytopathology 72:1384-1386.

Lapwood, D.H. 1961. Potato haulm resistance to _Phytophthora infestans_. II. Lesion production and sporulation. Ann. Appl. Biol. 49:316-330.

Lapwood, D.H. 1963. Potato haulm resistance to _Phytophthora infestans_. IV. Laboratory and field estimates compared, and further field analysis. Ann. Appl. Biol. 51:17-28.

Lapwood, D.H. and R.K McKee. 1966. Dose-response relationship for infection of potato leaves by zoospores of _Phytophthora infestans_. Trans. Br. mycol. Soc. 49:679-686.

Latin, R.X., D.R. MacKenzie and H. Cole, Jr. 1981. The influence of host and pathogen genotypes on the apparent infection rates of potato late blight epidemics. Phytopathology 71:82-85.

Lee, T.S. and G. Shaner. 1985. Oligogenic inheritance of length and latent period in six slow leaf-rusting wheat cultivars. Phytopathology 75:636-643.

Leonard, K.J. 1969. Factors affecting rates of stem rust increase in mixed plantings of susceptible and resistant oat varieties. Phytopathology 59:1845-50.

Leonard, K.J. and C.C. Mundt. 1984. Methods for estimating epidemiological effects of quantitative resistance to plant diseases. Theor. Appl. Genet. 67:219-230.

Leonard, K.L. 1969. Selection in heterogeneous populations of _Puccinia graminis_ f. sp. _avenae_. Phytopathology 59:1851-1857.

Lewellen, R.T., E.L. Sharp and E.R. Hehn. 1967 Major and minor genes in wheat for resistance to _Puccinia striiformis_ and their responses to temperature changes. Can. J. Bot. 45:2155-2172.

Lewontin, R.C. 1974. The Genetic Basis of Evolutionary Change. Pub: Columbia University Press.

Loegering, W.Q. 1951. Survival of races of wheat stem rust in mixtures. Phytopathology 41:55-65.

Luig, N.H. and I.A. Watson. 1970 The effect of complex genetic resistance in wheat on the variability of Puccinia graminis f. sp. tritici. Proc. Linn. Soc. N.S.W. 19:22-45.

Luig, N.H. and S. Rajaram. 1972. The effect of temperature and genetic background on the host gene expression and interaction to Puccinia graminis tritici. Phytopathology 62:1171-1174.

MacKenzie, D.R. 1978. Letter to the editor. Estimating parasitic fitness. Phytopathology 68:9-13.

Main, C.E. and M.E. Gallegley. 1964. The disease cycle in relation to multigenic resistance of potato to late blight. Amer. Potato J. 41:387-400.

Marshall, D.R. and B.S. Weir. 1985. Multiline varieties and disease control. Theor. Appl. Genet. 69:463-474.

Mather, K. and J.L. Jinks. 1971. Biometrical Genetics. 2nd Edition. Pub: Chapman and Hall, London.

Mather, S.C. and E.D. Hansing. 1960. Root and shoot development of wheat infected with loose smut Ustilago tritici. Phytopathology 50:645 (Abstr.).

Megginson, F.G.A. and C.O. Person. 1974. Somatic recombination in Ustilago hordei during the parasitic phase on barley. Can. J. Genet. Cytol. 16:851-855.

Mehta, Y.R. and J.C. Zadoks. 1970. Uredospore production and sporulation period of Puccinia recondita f.sp. triticina on primary leaves of wheat. Neth. J. Plant Pathol. 76:267-276.

Nass, H.A., W.L. Pederson, D.R. MacKenzie and R.R. Nelson. 1981. The residual effects of some "defeated" powdery mildew resistance genes in isolines of winter wheat. Phytopathology 71:1315-1318.

Nelson, R.R. 1978. Genetics of horizontal resistance to plant diseases. Annu. Rev. Phytopathol. 16:359-378.

Nelson, R.R. 1979. The Evolution of parasitic fitness. In: Plant Disease and Advanced Treatise. IV. How Pathogens Induce Disease. Eds: J.G. Horsfall and E.B. Cowling. Pub: Academic Press. New York. pp. 23-46.

Nelson, R.R., J.E. Ayers, H. Cole and D.H. Peterson. 1970. Studies and observations on the past occurence and geographical distribution of isolates of race T of Helminthosporium maydis. Plant Dis. Reporter 54:1123-1126.

Ohms, R.E. and W.M. Bever. 1954. Effect of Ustilago tritici infection on third internode elongation in resistant and susceptible winter wheat. Phytopathology 44:500 (Abstr.).

Ohms, R.E. and W.M. Bever. 1955. Types of seedling reactions of Kawvale and Wabash winter wheat to three physiologic races of Ustilago tritici. Phytopathology 45:513-516.

Ostergaard, H. 1983. Problems in estimating parasitic fitness. In: Durable Resistance in Crops. Ed: F. Lamberti, J.M. Waller and N.A. van der Graaff. Pub: Plenum Press. New York.

Parlevliet, J.E. 1975. Partial resistance of barley to leaf rust, Puccinia hordei. I. Effect of cultivar and development stage on latent period. Euphytica 24:21-27.

Parlevliet, J.E. 1978. Further evidence of polygenic inheritance of partial resistance in barley to leaf rust, Puccinia hordei. Euphytica 27:369- 379.

Parlevliet, J.E. 1978. Race-specific aspects of polygenic resistance of barley to leaf rust, Puccinia hordei. Netherlands J. Plant Path. 84:121-126.

Parlevliet, J.E. 1979. Components of resistance that reduce the rate of epidemic development. Annu. Rev. Phytopathol. 17:203-222.

Parlevliet, J.E. 1981. Race-non-specific disease resistance. In: Strategies for the Control of Cereal Disease. Ed: J.F. Jenkyn and R.T. Plumb. Pub: Blackwell Scientific Publications, London. pp.47-54.

Parlevliet, J.E. and A. Van Ommeren. 1975. Partial resistance of barley to leaf rust, _Puccinia_ _hordei_. II. Relationship between field trials, micro plot tests and latent period. Euphytica 24:293-303.

Parlevliet, J.E. and J.C. Zadoks. 1977. The integrated concept of disease resistance: a new view including horizontal and vertical resistance in plants. Euphytica 26:5-21.

Perkins, D.D. 1962. Preservation of _Neurospora_ stock cultures with anhydrous silica gel. C. J. Microbiol. 8:591-594.

Person, C. 1959. Gene-for-gene relationships in host parasite systems. Can J. Bot. 37:1101-1130.

Person, C. 1965. Microevolutionary changes in parasitic systems. In: The Montana Symposium on Integrated Biology; Host-Parasite Interactions, 1:1-12.

Person, C. 1966. Genetic polymorphism in parasitic systems. Nature 212:266-267.

Person, C. 1968. Genetical adjustment of fungi to their environment. In: The Fungi: Vol. III. An Advanced Treatise. Ed: by G.C. Ainsworth and A.S. Sussman. Pub: Academic Press, New York.

Person, C. and W.J. Cherewick. 1964. Infection multiplicity in _Ustilago_. Can. J. Genet. and Cytol. 6:12-18.

Person, C. and G.M.E. Mayo. 1974. Genetic limitations on models of specific interaction between a host and its parasite. Can. J. Bot. 52:1339-1347.

Person, C., J.V. Groth and O.M. Mylyk. 1976. Genetic change in host-parasite populations. Annu. Rev. Phytopathol. 14:177-188.

Person, C. and B. Christ. 1983. Population genetics and evolution of host-parasite interactions. In: Challenging Problems in Plant Health. Ed: T. Kommedahl and P.H. Williams. Pub: APS, St. Paul, Minnesota, pp. 379-386.

Person, C., R. Fleming, L. Cargeeg and B. Christ. 1983. Present knowledge and theories concerning durable resistance. In: Durable Resistance in Crops. Ed: F. Lamberti, J.M. Walter and N.A. Van der Graff. Pub: Plenum Press, New York.

Person, C.O., B.J. Christ and D.D. Pope. 1986. The genetic determination of variation in pathogenicity. In: Populations of Plant Pathogens: Their Dynamics and Genetics. Ed: M.S. Wolfe and C.E. Caten. Pub: Blackwell Scientific Publications. Oxford (in press).

Person, C, T. Ebba and B. Christ. 1986. (in press).

Pimentel, D. 1961. Animal regulation by genetic feedback mechanism. Am Natur. 95:65-79.

Pope, D.D. 1982. Biometrical analysis of pathogenicity in the Ustilago hordei-Hordeum vulgare host-parasite system. M.Sc. Thesis, University of British Columbia, Vancouver, Canada. 156 pp.

Price, P.W. 1980. Evolutionary Biology of Parasites. Pub: Princeton University Press, Princeton, New Jersey.

Rapilly, F. 1979. Yellow rust epidemiology. Annu. Rev. Phytopathol. 17:59-73.

Raymundo, A.D. and A.L. Hooker. 1982. Single and combined effects of monogenetic and polygenic resistance of certain components of northern corn leaf blight development. Phytopathology 72:99-103.

Riley, R. 1973. Genetic changes in hosts and the significance of disease. Ann. Appl. Biol. 75:128-132.

Robinson, R.A. 1969. Disease resistance terminology. Rev. Appl. Mycol. 48:593-606.

Robinson, R.A. 1973. Horizontal resistance. Rev. Plant Pathol. 52:483-501.

Robinson, R.A. 1976. Plant Pathosystems. Pub: Springer-Verlag, New York.

Robinson, R.R. 1980. New concepts in breeding for disease resistance. Annu. Rev. Phytopathol. 18:189-210.

Robinson, R.A. 1986. Host Management. Pub: The MacMillan Co. (in press).

Roelfs, A.P. 1984. Race specificity and methods of study. Ed: W.R. Bushnell and A.P. Roelfs. Pub: Academic Press, Inc. London.

Rosen, H.R. 1949. Oat parentage and procedures for combining resistance to crown rust, including race 45, and Helminthosporium blight. Phytopathology 39:20.

Roughgarden, J. 1983. The theory of coevolution. In: Coevolution. Ed: D.J. Futuyma and M. Slatkin. Sinauer Associates Inc. Sunderland, Massachusetts. pp. 33-64.

Rouse, D.I., R.R. Nelson and D.R. MacKenzie. 1980. A stochastic model of horizontal resistance based on frequency distributions. Phytopathology 70:951-954.

Rouse, D.I., R.R. Nelson, D.R. MacKenzie and C.R. Armitage. 1980. Components of rate-reducing resistance in seedlings of four wheat cultivars and parasitic fitness in six isolates of Erysiphe graminis f. sp. tritici. Phytopathology 70:1097-1100.

Royer, M.H. and R.R. Nelson. 1981. The effect of host resistance on relative parasitic fitness of Helminthosporium maydis race T. Phytopathology 71:351-354.

Rufty, R.C., T.T. Hebert and C.F. Murphy. 1981. Variation in virulence in isolates of Septoria nodorum. Phytopathology 71:593-596.

Ruttle, M.L. 1934. Studies on barley smuts and on loose smut of wheat. New York Agr. Expt Sta. Tech. Bull. No. 221.

Ruttle, M.L. 1934. Studies on barley smuts and on loose smut of wheat. New York Agr. Expt. Sta. Tech. Bull. No. 221.

SAS for Linear Models. 1981. Ed: Randolf J. Freunf and Ramon C. Littell. Pub: SAS Institute Inc. Cary, N. Carolina. 231 pp.

SAS User's Guide: Basics. 1982a. Pub: SAS Institute Inc. Cary, N. Carolina. 923 pp.

SAS User's Guide: Statistics. 1982b. Pub: SAS Institute Inc. Cary, N. Carolina. 584 pp.

Schwarzbach, E. and M.S. Wolfe. 1976. Consideration of the relationship between Erysiphe graminis f. sp. hordei and its host, barley, in the exploitation of host resistance. In: Barley Genetics III. Ed: H. Gaul and K. Thiemig, Munich. pp. 426-432.

Shaner, G. and F.D. Hess. 1978. Equations for integrating components of slow leaf-rusting in wheat. Phytopathology 68:1464-1469.

Shaner, G.E. and R.E. Finney. 1980. New sources of slow leaf rusting resistance in wheat. Phtyopathology 70:1183-1186.

Shattock, R.C. and D.S. Shaw. 1976. Novel phenotypes of Phytopthora infestans from mixed culture of antibiotic resistant mutants. Trans. Br. mycol. Soc. 67:201-206.

Simons, M.D. 1979. Modification of host-parasite interactions through artificial mutagenesis. Annu. Rev. Phytopathol. 17:75-96.

Simons, M.D. and H.C. Murphy. 1967. Determination of relative
    tolerence to Puccinia coronata avena of experimental lines
    of oats. Plant Dis. Reptr. 51:947-950.

Singh, S. 1979. Relative efficiency of North Carolina designs I
    and II and standard design III in three wheat crosses.
    Theor. Appl. Genet. 54:33-35.

Smedegaard-Petersen, V. 1985. The limiting effect of disease
    resistance on yield. Annu. Rev. Phytopathol. 23:475-490.

Sokal, R.R. and F.J. Rohlf. 1981. Biometry. Pub: W.H. Freeman
    and Company. San Francisco. 859 pp.

Stevens, F.L. 1913. The Fungi which Cause Plant Disease. Pub:
    The MacMillan Co. New York.

Tapke, V.F. 1929. Influence of varietal resistance, sap acidity,
    and certain environmental factors on the occurrence of
    loose smut in wheat. J. Ag. Res. 39:313-339.

Tapke, V.F. 1931. Influence of humidity on floral infection of
    wheat and barley by loose smut. J. Ag. Res. 43:503-516.

Tapke, V.F. 1937. Physiologic races of Ustilago hordei. J. Agr.
    Res. 55:683-692.

Tapke, V.F. 1938. Influence of environment, after seedling
    emergence, on covered smut in barley. Phytopathology
    28:370-371.

Tapke, V.F. 1945. New physiologic races of Ustilago hordei. J.
    Agr. Res. 55:683-692.

Tapke, V.F. 1948. Environment and cereal smuts. Bot. Rev.
    14:359-412.

Tapke, V.F. and W.M. Bever. 1942. Effective methods of
    inoculating barley seed with covered smut. Phytopathology
    32:1015-1021.

Tepper, C.S. and A.J. Anderson. 1984. The genetic basis of plant-pathogen interaction. Phytopathology 74:1143-1144.

Umaerus, V. 1969. Studies of field resistance to <u>Phytopthora infestans</u>. A method of screening young potato seedlings for field resistance. Z. Pflanzensuchtg. 61:167-194.

Umaerus, V. 1970. Studies on field resistance to <u>Phytopthora infestans</u>.5. Mechanisms of resistance and applications to potato breeding. Z. Pfl. Zucht. 63:1-23.

Upshall, A. 1969. Intraracial variation in <u>Phytophthora infestans</u>, and its relationship to different host varieties. Can J. Bot. 47:863-867.

Vanderplank, J.E. 1963. Plant Diseases: Epidemics and Control. Pub: Academic Press. New York. 349 pp.

Vanderplank, J.E. 1968. Disease Resistance in Plants. Pub: Academic Press. New York. 206 pp.

Vanderplank, J.E. 1975. Principles of Plant Infection. Pub: Academic Press. New York. 217 pp.

Vanderplank, J.E. 1976. Four essays. Annu. Rev. Phytopathol. 14:1-10.

Vanderplank, J.E. 1978. Genetic and Molecular Basis of Plant Pathogenesis. Pub: Springer-Verlag. New York. 167 pp.

Vanderplank, J.E. 1982. Host-Pathogen Interactions in Plant Disease. Pub: Academic Press. New York. 207 pp.

Vanderplank, J.E. 1984. Disease Resistance in Plants. Pub: Academic Press. New York. 194 pp.

Villareal, R.L., R.R. Nelson, D.R. MacKenzie and W.R. Coffman. 1981. Some components of slow-blasting resistance in rice. Phytopathology 71:608-611.

Vogel, H.J. 1956. A convenient growth medium for <u>Neurospora</u>. Microbiol. Genet. Bull. 13:42-43.

Walsh, E.J. 1984. Developing yield potential in cereals. In: Cereal Production. Ed: E.J. Gallager. Pub: Butterworth's, Toronto.

Wehrhahn, C. 1986. Professor, Department of Animal Resource Ecology, University of British Columbia, Vancouver, B.C.

Welsh, J.N. 1932. The effect of smut on rust development and plant vigor in oats. Sci. Ag. 13:154-164.

Wilson, D.S. 1980. The Natural Selection of Populations and Communities. Pub: The Benjamin/Cummings Publishing Company, Inc. Don Mills, Ontario.

Wittwer, S.H. 1980. The shape of things to come. In: Biology of Crop Protection. Ed: P.S. Carlson. Pub: Academic Press. New York, pp. 413-460.

Wolfe, M.S. 1972. The genetics of barley mildew. Rev. Plant Pathol. 51:507-522.

Wolfe, M.S. 1973. Changes and diversity in populations of fungal pathogens. Ann. Appl. Biol. 75:132-136.

Woodward, R.W. and D.C. Tingey. 1941. Inoculation experiments with covered smut of barley. J. Amer. Soc. Agron. 33:632-642.

Wynne-Edwards, V.C. 1962. Animal Dispersion in Relation to Social Behavior. Pub: Oliver and Boyd. Edinburgh.

Yarwood, C.E. 1961. Uredospore production by <u>Uromyces</u> <u>phaseoli</u>. Phytopathology 51:22-27.

Zadoks, J.C. 1965. Epidemiology of wheat rusts in Europe. FAO Plant Protect. Bull. 13:97-108.

Zadoks, J.C. 1972. Methodology of epidemiological research. Annu. Rev. Phytopathol. 10:253-276.

Zar, J.H. 1984. Biostatistical Analysis. Second Edition. Pub: Prentice-Hall, Inc. N.J.

11 APPENDICES

11.1 APPENDIX A

11.1.1 MINIMAL MEDIUM

Vogel's Solution (50x)	20 ml
Distilled Water	1000 ml
Agar (Bacto)	20 gm
Dextrose (D-Glucose)	10 gm

Sterilize for 15 min in autoclave at 15 lbs, 121°C.

11.1.2 COMPLETE MEDIUM

Vogel's Solution (50x)	20 ml
Distilled Water	1000 ml
Tryptophane	50 mg
Casein Hydrolysate	5 mg
(vitamin and salt free)	
Yeast Extract (Difco)	5 gm
Dextrose (D-Glucose)	10 gm
Vitamin Solution	10 ml
Agar (Bacto)	20 gm

Sterilize for 15 min. in autoclave at 15 lbs, 121°C.

11.1.3 BAUCH MATING TYPE TEST PLATES

Same as Minimal Medium but with only 2 gm Dextrose.

11.1.4 VOGEL'S SOLUTION (50x; VOGEL, 1956)

Na2 citrate 2H2O	123 gm
K2HPO4	250 gm
NH4NO3 anhyd.	100 gm
MgSO4.7H2O	10 gm
CaCl2.2H2O	5 gm
Trace element solution	5 ml
Distilled Water	750 ml
Chloroform	2 ml

Heat solution and add chemicals gradually with stirring.
Store solution at room temperature in a stoppered
bottle.

11.1.5 TRACE ELEMENT SOLUTION

Citric Acid 1H2O	5 gm
Zn SO4.7H2O	5 gm
Fe(NH4)2.(SO4)2.6H2O	1 gm
CuSO4.5H2O	0.25 gm
MnSO4.1H2O	0.05 gm
H3BO3 anhyd.	0.05 gm

Na2MoO4.2H2O   0.05 gm
Chloroform   1 ml
Distilled Water   95 ml

Store at 4°C in tightly stoppered bottle.

11.1.6 VITAMIN SOLUTION (BEADLE AND TATUM, 1945)

Thiamin   100 mg
Riboflavin   50 mg
Pyridoxine   50 mg
Ca Pantothenate   200 mg
p-amino-benzoic Acid   50 mg
Nicotinic Acid   200 mg
Choline chloride   200 mg
Inositol   400 mg
Folic Acid   50 mg
Distilled Water   1000 ml

Dispense in 10 ml aliquots.
Store at -20°C.

11.2 APPENDIX B

This appendix contains all tables associated with this study.

TABLE 1.   Compilation of variance components and estimations
          of heritabilities of the percent smutted plants from
          experiments on pathogenicity in the smut-barley system.
          Bracketed values represent the percent contribution of
          the component to the total phenotypic variance.   (Emara
          (1972); Emara and Sidhu (1974); Pope (1982); Caten   et
          al. (1984))

          Vt = total variance;
          Vg = genetic variance;
          Va = additive genetic variance;
          Vna = nonadditive genetic variance;
          Ve = environmental variance;
          H2 = broad sense heritability;
          h2 = narrow sense heritability;
          s = selfed teliospore;
          i = inbred teliospore;
          n = natural isolate;
          T17 = teliospore from F1 dikaryotic line 17 (on Trebi);
          T21 = teliospore from F1 dikaryotic line 21 (on Trebi);
          T23 = teliospore from F1 dikaryotic line 23 (on Trebi);
          O17 = teliospore from F1 dikaryotic line 17 (on Odessa);
          O21 = teliospore from F1 dikaryotic line 21 (on Odessa);
          O23 = teliospore from F1 dikaryotic line 23 (on Odessa);

TABLE 1

RESEARCHER		Vt	VARIABILITY COMPONENTS				HERITABILITY	
			Vg	Va	Vna	Ve	H2	h2
Emara and Sidhu		301.69	196.76 (65.2)	132.52 (43.9)	64.23 (21.3)	104.93 (34.8)	0.65	0.44
Emara		72.14	36.96 (51.2)	30.35 (42.1)	6.61 (9.1)	35.18 (48.8)	0.51	0.42
Caten et al.	(s)	51.40	23.80 (46.0)			27.60 (54.9)	0.46	
	(i)	28.30	1.50 (5.0)			26.80 (95.0)	0.05	
	(i)	31.60	5.90 (19.9)			25.70 (81.0)	0.19	
	(n)	35.00	3.20 (9.0)			31.80 (91.0)	0.09	
	(n)	40.40	4.10 (10.0)			36.30 (90.0)	0.10	
	(n)	45.50	1.60 (4.0)			43.90 (96.0)	0.04	
	(n)	40.10	0.00 (0.0)			40.10 (100.0)	0.00	
Pope	(T17)	63.12	27.63 (43.8)	19.49 (30.9)	8.14 (12.9)	33.14 (52.5)	0.44	0.31
	(T21)	79.72	48.39 (60.7)	33.23 (41.7)	15.26 (19.0)	31.33 (39.3)	0.61	0.42
	(T23)	69.01	49.61 (71.9)	28.83 (41.8)	20.78 (30.1)	17.10 (24.8)	0.72	0.42
	(O17)	60.33	38.03 (63.0)	1.97 (3.3)	36.06 (59.8)	19.62 (32.5)	0.63	0.03
	(O21)	73.22	30.60 (41.8)	17.52 (23.9)	13.08 (17.8)	35.56 (48.6)	0.42	0.24
	(O23)	57.59	17.23 (30.0)	8.13 (14.1)	9.10 (15.8)	39.05 (67.8)	0.30	0.14

TABLE 2.   Ebba's and Tapke's disease readings from selfing
      teliospores T1 and T4 (ie. race 11 and 7, respectively).
      Disease readings on Trebi and Odessa are expressed as
      the percentage of plants smutted.

TABLE 2

| TELIOSPORE | RACE | SELF | TREBI | | ODESSA |
			EBBA %	TAPKE %	TAPKE %
T1	11	T1-1 x T1-2	42	–	–
		-1 x -4	49	–	–
		-3 x -2	44	–	–
		-3 x -4	43	–	–
		avg. =	44	43	39
T4	7	T4-1 x T4-3	2	–	–
		-1 x -4	3	–	–
		-2 x -3	2	–	–
		-2 x -4	3	–	–
		avg. =	2.5	5	34

TABLE 3.   Eight F1 dikaryotic line (DL) disease readings for
            the cross between teliospores T1 and T4 on Trebi (Ebba,
            1974).

TABLE 3

CROSS		DL	%
T1-1 x	T4-3	17	37
-1 x	-4	18	49
-2 x	-1	19	44
-2 x	-2	20	48
-3 x	-3	21	49
-3 x	-4	22	47
-4 x	-1	23	43
-4 x	-2	24	43
			----
		avg. =	45

TABLE 4.   Description of row (R) related and fitness (W)
        variables.   Codes and descriptions of the R subset of
        variables are catalogued.   Most variables were measured
        for, or, were expressed in relation to, the first 50
        plants scored in each row.   All weights are in mg
        units.

TABLE 4

CODE	DESCRIPTION
R1tw	germination rate of the 110 treated seeds originally planted
R2t	percent of plants smutted
R3	number of heads
R4t	percent of heads smutted
R5	number of heads from diseased plants
R6	average number of heads per plant
R7	average number of diseased heads per plant
R8	average number of healthy heads per plant
R9	average number of heads per diseased plant
R10	average number of diseased heads per diseased plant
R11	average number of healthy heads per diseased plant
R12	spore weight
R13	average spore weight per diseased plant
R14	average spore weight per diseased head
R15t	average spore germination rate per diseased head
R16	average number of seeds per diseased plant
R17	average number of seeds per plant
Wp	[PATHOGEN] pathogen fitness (calculated from P subset of variables)
Wc	[PATHOGEN] pathogen fitness (calculated from C subset of variables)
W	[PATHOGEN] total pathogen fitness (Wp+Wc)
Wp	[HOST] host fitness (calculated from P subset of variables)
Wh	[HOST] host fitness (calculated from H subset of variables)
W	[HOST] total host fitness (Wp+Wh)

t = modified angular transformation
w = measurement may have involved other plants in the row in addition to the first 50 (ie. the whole row)

TABLE 5.   Description of healthy plant (H) related variables.
         Codes and descriptions of the H subset of variables are
         given.   Most variables were measured for, or, were
         expressed in relation to, the first 50 plants scored in
         each row.   All weights are in mg units.

TABLE 5

CODE	DESCRIPTION
H1	number of healthy plants
H2	number of heads
H3	average number of heads per plant
H4	average number of seeds per plant
H5	average number of seeds per head
H6w	thousand seed weight, seeds randomly selected from all healthy plants
H7	average seed weight per plant
H8	average seed weight per head
H9t	seed germination rate (for seeds from H6)
H10	number of seeds

t = modified angular transformation
w = measurement may have involved other plants in the row in addition to the first 50 (ie. the whole row)

TABLE 6.   Description of completely diseased plant (C)
   related variables.   Codes and descriptions of the C
   subset of variables are given.   Most variables were
   measured for, or, were expressed in relation to, the
   first 50 plants scored in each row.   All weights are in
   mg units.

TABLE 6

CODE	DESCRIPTION
C1	number of completely diseased plants
C2	number of heads
C3	average number of heads per plant
C4	spore weight
C5	average spore weight per plant
C6	average spore weight per head
C7t	average spore germination rate per head

t = modified angular transformation

TABLE 7.   Description of partially diseased plant (P) related
          variables.   Codes and descriptions of the P subset of
          variables are given.   Most variables were measured for,
          or, were expressed in relation to, the first 50 plants
          scored in each row.   All weights are in mg units.

TABLE 7

CODE	DESCRIPTION
P1	number of diseased plants with seeds
P2	number of heads
P3	number of diseased heads
P4	number of healthy heads
P5	average number of heads per plant
P6	average number of diseased heads per plant
P7	average number of healthy heads per plant
P8	spore weight
P9	average spore weight per plant
P10	average spore weight per head
P11t	average spore germination rate per head
P12	number of seeds
P13	average number of seeds per plant
P14	average number of seeds per healthy head
P15	seed weight
P16	average seed weight per plant
P17	average seed weight per healthy head
P18t	average seed germination rate per healthy head

t = modified angular transformation

TABLE 8.   Mean values of the Trebi row (R) and fitness (W) subset of variables.   Column "T" identifies the treatment number (c=control).   Columns "+" and "-" identify the particular sporidial combination for each treatment.

TABLE 8

ROW (R) VARIABLE ON TREBI

T	+	-	1	2	3	4	5	6	7	8
c	-	-	47.5	-	111.0	-	-	2.2	-	2.2
1	1	1	48.8	13.5	100.0	11.9	5.3	2.0	0.1	1.9
2	1	2	46.9	12.2	115.0	10.0	4.3	2.3	0.1	2.2
3	1	3	47.8	11.5	119.7	8.7	11.0	2.4	0.1	2.3
4	1	4	51.2	11.5	106.7	7.8	7.0	2.2	0.1	2.1
5	1	5	49.6	16.7	111.0	14.3	10.0	2.2	0.2	2.0
6	2	1	44.5	26.2	145.3	18.4	32.0	2.9	0.3	2.6
7	2	2	45.0	4.0	159.0	2.3	0.0	3.2	0.0	3.2
8	2	3	48.1	12.0	95.0	9.8	4.3	1.9	0.1	1.8
9	2	4	46.9	4.0	105.0	2.8	0.0	2.1	0.0	2.1
10	2	5	49.0	15.7	93.3	17.7	9.3	1.8	0.2	1.7
11	3	1	48.8	24.0	99.0	20.5	16.0	2.0	0.2	1.7
12	3	2	46.9	5.9	117.3	4.3	0.3	2.3	0.0	2.3
13	3	3	48.9	13.9	97.7	12.0	8.3	2.0	0.1	1.9
14	3	4	46.3	12.5	117.3	8.2	2.0	2.3	0.0	2.3
15	3	5	49.1	29.8	88.7	27.6	20.0	1.8	0.4	1.4
16	4	1	45.7	25.2	97.3	25.4	22.7	2.0	0.4	1.6
17	4	2	46.2	4.0	93.0	3.0	0.0	1.9	0.0	1.9
18	4	3	47.6	14.7	114.7	10.4	6.0	2.3	0.1	2.2
19	4	4	46.7	23.6	99.0	20.4	14.3	2.0	0.2	1.7
20	4	5	47.0	24.4	94.0	22.2	14.3	1.9	0.3	1.6

TABLE 8
(continued)

ROW (R) VARIABLE ON TREBI

T	+	-	9	10	11	12	13	14	15	16	17
c	-	-	-	-	-	-	-	-	-	-	-
1	1	1	2.4	1.8	0.6	0.6050	0.2933	0.1323	46.8	11.6	62.2
2	1	2	1.8	1.7	0.2	0.3233	0.1188	0.0641	41.8	5.3	76.5
3	1	3	2.1	1.0	1.1	0.5077	0.0929	0.0515	31.7	36.6	75.6
4	1	4	1.7	0.7	1.0	0.1847	0.0424	0.0384	32.8	26.4	64.0
5	1	5	1.3	1.2	0.2	1.2057	0.1493	0.0860	28.5	4.2	71.9
6	2	1	2.8	1.5	1.3	1.3613	0.1347	0.0902	48.1	44.4	90.0
7	2	2	0.0	0.0	0.0	0.0	0.0	0.0	2.9	0.0	120.1
8	2	3	1.1	0.9	0.2	0.4180	0.0981	0.0704	34.0	8.4	51.3
9	2	4	0.0	0.0	0.0	0.0	0.0	0.0	2.9	0.0	65.3
10	2	5	3.1	2.5	0.6	0.8490	0.3004	0.1173	47.1	17.9	53.2
11	3	1	2.0	1.5	0.5	1.0233	0.1221	0.0827	45.7	13.9	55.9
12	3	2	0.3	0.3	0.0	0.0057	0.0057	0.0057	12.6	0.0	68.8
13	3	3	2.7	1.6	1.1	0.6703	0.2155	0.1240	47.7	32.5	57.5
14	3	4	1.0	1.0	0.0	0.0737	0.0595	0.0595	43.9	0.0	66.1
15	3	5	1.6	1.5	0.2	2.2137	0.1765	0.1196	49.2	3.7	44.6
16	4	1	2.2	2.0	0.3	2.6020	0.2403	0.1199	44.8	8.2	49.2
17	4	2	0.0	0.0	0.0	0.0	0.0	0.0	2.9	0.0	48.3
18	4	3	1.7	1.2	0.5	0.3490	0.1039	0.0882	50.9	14.2	74.2
19	4	4	1.6	1.4	0.3	1.3927	0.1499	0.1061	46.2	9.6	54.6
20	4	5	1.8	1.6	0.1	1.8153	0.2244	0.1392	47.8	4.3	49.1

TABLE 8
(continued)

ROW (R) VARIABLE ON TREBI

			[PATHOGEN]			[HOST]		
T	+	-	Wp	Wc	W	Wp	Wh	W
c	-	-	-	-	-	-	3258.3	3258.3
1	1	1	0.1224	0.2070	0.3294	22.8	2884.6	2907.4
2	1	2	0.0107	0.1322	0.1429	20.1	3538.6	3558.7
3	1	3	0.1693	0.0776	0.2469	213.4	3341.1	3554.5
4	1	4	0.0401	0.0603	0.1004	114.5	2840.2	2954.6
5	1	5	0.0755	0.4588	0.5343	34.6	3284.7	3319.2
6	2	1	0.5315	0.2455	0.7769	541.5	3500.4	4041.9
7	2	2	0	0	0	0	5605.4	5605.4
8	2	3	0.1263	0.1450	0.2712	40.4	2322.2	2362.7
9	2	4	0	0	0	0	3068.3	3068.3
10	2	5	0.1048	0.3487	0.4534	41.0	2474.1	2515.0
11	3	1	0.1491	0.3782	0.5273	94.3	2510.5	2604.8
12	3	2	0	0.0016	0.0016	0	3201.6	3201.6
13	3	3	0.2488	0.1358	0.3846	108.1	2612.4	2720.5
14	3	4	0	0.0385	0.0385	0	3131.4	3131.4
15	3	5	0.1134	1.1877	1.3011	28.6	2053.1	2081.7
16	4	1	0.3425	0.9590	1.3015	114.9	2165.6	2280.5
17	4	2	0	0	0	0	2201.7	2201.7
18	4	3	0.1698	0.0376	0.2074	54.5	3407.9	3462.5
19	4	4	0.1080	0.6046	0.7125	80.7	2451.8	2532.5
20	4	5	0.1061	0.8898	0.9959	36.3	2237.2	2273.6

TABLE 9.   Mean values of the Trebi healthy plant (H) subset
of variables.   Column "T" identifies the treatment
number (c=control).   Columns "+" and "-" identify the
particular sporidial combination for each treatment.

TABLE 9

HEALTHY PLANT (H) VARIABLE ON TREBI

T	+	-	1	2	3	4	5	6	7
c	-	-	50.0	111.0	2.2	68.8	31.0	47.9	3.2957
1	1	1	47.7	94.7	2.0	64.5	31.9	48.8	3.1013
2	1	2	48.0	110.7	2.3	79.9	32.8	48.4	3.9147
3	1	3	47.7	108.7	2.3	76.6	30.5	47.1	3.7182
4	1	4	47.7	99.7	2.1	65.6	30.9	46.0	3.0710
5	1	5	45.0	101.0	2.2	78.9	35.2	48.3	3.8282
6	2	1	40.3	113.3	2.8	96.8	34.9	45.0	4.3510
7	2	2	50.0	159.0	3.2	120.1	37.7	45.9	5.5371
8	2	3	47.7	90.7	1.9	52.9	27.3	43.6	2.2984
9	2	4	50.0	105.0	2.1	65.3	30.9	48.5	3.1798
10	2	5	46.7	84.0	1.8	56.3	31.8	49.8	2.7888
11	3	1	42.0	83.0	2.0	64.0	32.5	48.2	3.0762
12	3	2	49.7	117.0	2.3	69.0	27.3	43.7	3.1487
13	3	3	47.3	89.3	1.9	58.5	30.7	49.1	2.8833
14	3	4	48.0	115.3	2.4	69.1	28.9	48.4	3.3459
15	3	5	37.7	68.7	1.8	56.8	31.5	47.6	2.7195
16	4	1	40.7	74.7	1.8	58.3	30.0	48.9	2.8917
17	4	2	50.0	93.0	1.9	48.3	24.8	47.0	2.3628
18	4	3	47.0	108.7	2.3	77.9	33.5	48.0	3.7356
19	4	4	42.0	84.7	2.0	61.4	30.5	47.5	2.9857
20	4	5	41.7	79.7	1.9	57.4	30.2	47.1	2.7028

TABLE 9
(continued)

HEALTHY PLANT (H)
VARIABLE ON TREBI

T	+	−	8	9	10
c	−	−	1.4854	76.5	3440.2
1	1	1	1.5490	74.6	3082.6
2	1	2	1.5975	75.4	3803.1
3	1	3	1.4574	74.3	3561.4
4	1	4	1.4242	73.2	3081.8
5	1	5	1.7065	73.3	3559.8
6	2	1	1.5710	71.0	3920.8
7	2	2	1.7310	75.2	6006.7
8	2	3	1.1875	72.9	2521.4
9	2	4	1.5093	75.4	3265.0
10	2	5	1.5839	76.1	2618.8
11	3	1	1.5643	75.0	2685.2
12	3	2	1.2259	73.6	3440.6
13	3	3	1.5129	76.6	2752.7
14	3	4	1.3965	76.1	3306.2
15	3	5	1.5044	74.2	2189.2
16	4	1	1.4811	73.7	2334.5
17	4	2	1.1976	71.1	2415.0
18	4	3	1.6051	74.6	3642.1
19	4	4	1.4635	74.5	2642.8
20	4	5	1.4180	74.5	2415.7

TABLE 10.   Mean values of the Trebi completely diseased plant
(C) subset of variables.   Column "T" identifies the
treatment number (c=control).   Columns "+" and "-"
identify the particular sporidial combination for each
treatment.

TABLE 10

COMPLETELY DISEASED PLANT (C)
VARIABLE ON TREBI

T	+	−	1	2	3	4	5	6	7
C	−	−	−	−	−	−	−	−	−
1	1	1	1.0	2.0	2.0	0.3830	0.3830	0.1605	46.3
2	1	2	1.3	3.0	2.0	0.2903	0.1705	0.0699	42.4
3	1	3	1.0	1.7	1.0	0.1520	0.0810	0.0455	32.1
4	1	4	0.7	1.0	0.5	0.1143	0.0572	0.0381	17.4
5	1	5	4.0	7.0	1.2	1.0540	0.1628	0.0930	28.1
6	2	1	4.3	6.3	1.5	0.4737	0.1114	0.0743	45.2
7	2	2	0.0	0.0	0.0	0.0	0.0	0.0	2.9
8	2	3	2.0	2.7	0.8	0.2540	0.0756	0.0584	33.1
9	2	4	0.0	0.0	0.0	0.0	0.0	0.0	2.9
10	2	5	2.7	6.3	2.5	0.6583	0.2939	0.1142	47.7
11	3	1	5.0	8.0	1.6	0.7347	0.1433	0.0923	46.1
12	3	2	0.3	0.3	0.3	0.0057	0.0057	0.0057	12.6
13	3	3	1.0	2.0	1.2	0.2710	0.1593	0.0923	32.5
14	3	4	2.0	2.0	1.0	0.0737	0.0595	0.0595	43.9
15	3	5	11.0	16.3	1.5	2.0350	0.1776	0.1193	48.6
16	4	1	7.0	14.7	2.1	1.9473	0.2563	0.1185	44.4
17	4	2	0.0	0.0	0.0	0.0	0.0	0.0	2.9
18	4	3	1.7	1.0	0.7	0.0930	0.0595	0.0470	32.9
19	4	4	6.3	9.7	1.5	1.2107	0.1733	0.1133	46.8
20	4	5	7.3	11.7	1.7	1.6020	0.2321	0.1377	48.0

TABLE 11.　Mean values of the Trebi partially diseased plant (P) subset of variables.　Column "T" identifies the treatment number (c=control).　Columns "+" and "-" identify the particular sporidial combination for each treatment.

TABLE 11

PARTIALLY DISEASED PLANT (P)
VARIABLE ON TREBI

T	+	−	1	2	3	4	5	6	7	8
--	-	-	---	----	---	----	---	---	---	------
c	−	−	−	−	−	−	−	−	−	−
1	1	1	1.3	3.3	2.0	1.3	2.7	1.7	1.0	0.2220
2	1	2	0.7	1.3	0.7	0.7	0.7	0.3	0.3	0.0330
3	1	3	1.3	9.3	2.7	6.7	2.3	0.7	1.7	0.3557
4	1	4	1.7	6.0	1.7	4.3	2.0	0.7	1.3	0.0703
5	1	5	1.0	3.0	1.7	1.3	2.0	1.2	0.8	0.1517
6	2	1	5.3	25.7	8.7	17.0	3.7	1.4	2.2	0.8877
7	2	2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
8	2	3	0.3	1.7	0.7	1.0	1.7	0.7	1.0	0.1640
9	2	4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
10	2	5	0.7	3.0	1.7	1.3	3.0	1.7	1.3	0.1907
11	3	1	3.0	8.0	4.0	4.0	2.6	1.3	1.3	0.2887
12	3	2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
13	3	3	1.7	6.3	2.3	4.0	2.1	0.8	1.3	0.3993
14	3	4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
15	3	5	1.3	3.7	1.7	2.0	2.8	1.3	1.5	0.1787
16	4	1	2.3	8.0	4.0	4.0	2.2	1.2	0.9	0.6547
17	4	2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
18	4	3	1.3	5.3	2.7	2.7	2.4	1.3	1.1	0.2560
19	4	4	1.7	4.7	2.0	2.7	1.9	0.8	1.1	0.1820
20	4	5	1.0	2.7	1.3	1.3	1.7	0.8	0.8	0.2133

TABLE 11
(continued)

PARTIALLY DISEASED PLANT (P)
VARIABLE ON TREBI

T	+	-	9	10	11	12	13	14
c	-	-	-	-	-	-	-	-
1	1	1	0.2167	0.1138	47.0	26.0	21.8	21.8
2	1	2	0.0165	0.0165	13.6	21.3	10.7	10.7
3	1	3	0.0889	0.0445	16.5	219.3	54.8	11.0
4	1	4	0.0281	0.0281	33.2	118.7	35.7	17.2
5	1	5	0.0955	0.0571	30.0	37.0	20.8	15.4
6	2	1	0.1514	0.1125	48.4	578.0	74.3	32.5
7	2	2	0.0	0.0	2.9	0.0	0.0	0.0
8	2	3	0.1640	0.0820	22.3	42.0	42.0	14.0
9	2	4	0.0	0.0	2.9	0.0	0.0	0.0
10	2	5	0.1907	0.0732	30.9	42.0	42.0	20.9
11	3	1	0.0848	0.0626	44.6	106.7	38.2	30.7
12	3	2	0.0	0.0	2.9	0.0	0.0	0.0
13	3	3	0.1133	0.0816	34.2	122.3	38.1	20.2
14	3	4	0.0	0.0	2.9	0.0	0.0	0.0
15	3	5	0.1572	0.1110	50.7	42.3	32.3	22.8
16	4	1	0.1658	0.0927	33.9	120.3	24.8	16.1
17	4	2	0.0	0.0	2.9	0.0	0.0	0.0
18	4	3	0.1238	0.0619	36.8	65.0	27.4	16.7
19	4	4	0.0616	0.0469	27.5	86.0	36.2	21.5
20	4	5	0.1080	0.0729	31.3	39.7	25.8	21.2

TABLE 11
(continued)

PARTIALLY DISEASED PLANT (P)
VARIABLE ON TREBI

T	+	-	15	16	17	18
--	-	-	-------	------	------	----
c	-	-	-	-	-	-
1	1	1	1.1557	1.0225	1.0225	73.2
2	1	2	1.1010	0.5505	0.5505	27.0
3	1	3	10.9037	2.7259	0.5452	28.5
4	1	4	5.6017	1.5947	0.7042	47.9
5	1	5	1.8583	1.0038	0.7190	44.8
6	2	1	28.6410	3.6745	1.5967	78.2
7	2	2	0.0	0.0	0.0	2.9
8	2	3	2.4120	2.4120	0.8040	28.0
9	2	4	0.0	0.0	0.0	2.9
10	2	5	2.2213	2.2213	1.0849	53.4
11	3	1	4.6863	1.6907	1.3577	70.3
12	3	2	0.0	0.0	0.0	2.9
13	3	3	5.9787	1.8432	0.9659	45.5
14	3	4	0.0	0.0	0.0	2.9
15	3	5	1.5177	1.2810	0.9253	61.0
16	4	1	6.4373	1.2571	0.7861	51.8
17	4	2	0.0	0.0	0.0	2.9
18	4	3	3.2487	1.3407	0.7955	48.5
19	4	4	4.1373	1.7347	1.0343	51.2
20	4	5	1.8667	1.2630	1.0618	50.9

TABLE 12.   Mean values of the Odessa row (R) and fitness (W)
        subset of variables.   Column "T" identifies the
        treatment number (c=control).   Columns "+" and "-"
        identify the particular sporidial combination for each
        treatment.

TABLE 12

ROW (R) VARIABLE ON ODESSA

T	+	−	1	2	3	4	5	6	7	8
c	−	−	56.2	−	78.7	−	−	1.6	−	1.6
1	1	1	49.7	16.6	88.0	15.6	6.7	1.7	0.1	1.6
2	1	2	50.2	18.7	79.0	16.8	7.7	1.6	0.1	1.4
3	1	3	50.9	11.0	82.3	10.1	3.3	1.7	0.1	1.6
4	1	4	55.7	17.0	66.3	16.5	5.3	1.3	0.1	1.2
5	1	5	55.9	26.5	56.7	27.1	12.7	1.1	0.3	0.9
6	2	1	51.1	25.0	56.7	26.6	11.7	1.1	0.2	0.9
7	2	2	49.8	4.0	83.3	3.2	0.0	1.7	0.0	1.7
8	2	3	51.5	18.2	81.3	19.5	15.7	1.6	0.2	1.4
9	2	4	50.3	16.0	80.7	14.1	5.3	1.6	0.1	1.5
10	2	5	50.7	25.8	68.3	24.5	12.3	1.4	0.2	1.1
11	3	1	45.5	26.2	63.7	27.9	17.0	1.3	0.3	1.0
12	3	2	51.7	4.0	63.0	3.6	0.0	1.3	0.0	1.3
13	3	3	52.6	21.4	92.7	18.1	13.7	1.9	0.2	1.6
14	3	4	50.7	22.1	81.3	20.5	12.7	1.6	0.2	1.4
15	3	5	50.4	23.5	57.7	23.3	11.7	1.2	0.2	1.0
16	4	1	50.1	27.1	63.7	26.7	12.3	1.3	0.2	1.0
17	4	2	53.7	4.0	64.0	3.6	0.0	1.3	0.0	1.3
18	4	3	51.2	22.5	68.7	22.9	11.7	1.4	0.2	1.2
19	4	4	48.7	27.2	101.7	24.7	22.0	2.0	0.3	1.7
20	4	5	50.4	25.8	76.3	25.3	20.3	1.5	0.3	1.2

TABLE 12
(continued)

ROW (R) VARIABLE ON ODESSA

T	+	-	9	10	11	12	13	14	15	16	17
c	-	-	-	-	-	-	-	-	-	-	-
1	1	1	1.8	1.6	0.2	1.0490	0.2822	0.1677	36.0	5.8	64.3
2	1	2	1.4	1.3	0.2	0.5487	0.0940	0.0652	40.3	3.6	49.9
3	1	3	0.9	0.9	0.0	0.4293	0.0880	0.0501	25.2	0.0	60.2
4	1	4	1.3	1.3	0.0	0.4797	0.1102	0.0849	45.7	0.0	46.0
5	1	5	1.2	1.2	0.0	1.3643	0.1246	0.1068	41.2	0.0	27.5
6	2	1	1.3	1.3	0.0	1.3787	0.1508	0.1141	46.4	0.0	28.7
7	2	2	0.0	0.0	0.0	0.0	0.0	0.0	2.9	0.0	65.1
8	2	3	2.4	1.9	0.5	1.9600	0.2822	0.1396	53.1	18.3	49.8
9	2	4	1.4	1.2	0.2	0.1423	0.0338	0.0278	43.8	6.2	57.3
10	2	5	1.4	1.2	0.1	1.1980	0.1310	0.1066	49.6	6.7	39.1
11	3	1	1.3	1.3	0.0	2.5190	0.1911	0.1439	54.7	1.1	32.3
12	3	2	0.0	0.0	0.0	0.0	0.0	0.0	2.9	0.0	37.7
13	3	3	1.6	1.4	0.2	1.5870	0.1907	0.1340	49.9	6.6	64.0
14	3	4	1.7	1.4	0.3	0.8783	0.1045	0.0744	46.3	9.5	51.5
15	3	5	1.3	1.1	0.2	1.4137	0.1702	0.1492	47.3	6.7	33.5
16	4	1	1.2	1.2	0.1	1.4913	0.1630	0.1340	48.8	0.9	36.4
17	4	2	0.0	0.0	0.0	0.0	0.0	0.0	2.9	0.0	41.9
18	4	3	1.5	1.4	0.1	1.9013	0.2434	0.1709	52.0	3.4	42.8
19	4	4	2.1	1.6	0.5	2.3137	0.2181	0.1340	51.1	16.0	60.4
20	4	5	2.1	1.8	0.3	1.9123	0.2413	0.1300	45.8	10.8	39.9

TABLE 12
(continued)

ROW (R) VARIABLE ON ODESSA

| | | | [PATHOGEN] | | | [HOST] | | |
T	+	−	Wp	Wc	W	Wp	Wh	W
c	−	−	−	−	−	−	2755.5	2755.5
1	1	1	0.1558	0.2506	0.4064	18.8	3109.0	3127.7
2	1	2	0.0149	0.2258	0.2407	20.2	2388.1	2408.4
3	1	3	0	0.2207	0.2207	0	2905.1	2905.1
4	1	4	0	0.2512	0.2512	0	2213.4	2213.4
5	1	5	0	0.5769	0.5769	0	1269.2	1269.2
6	2	1	0	0.7497	0.7497	0	1413.9	1413.9
7	2	2	0	0	0	0	3093.2	3093.2
8	2	3	0.5466	0.6780	1.2247	120.6	2220.9	2341.5
9	2	4	0.0122	0.0500	0.0622	21.7	2750.4	2772.1
10	2	5	0.1007	0.6006	0.7012	51.0	1827.7	1878.6
11	3	1	0.1313	1.5212	1.6526	17.0	1541.8	1558.8
12	3	2	0	0	0	0	1766.5	1766.5
13	3	3	0.3621	0.6796	1.0417	85.6	2907.1	2992.7
14	3	4	0.0377	0.4582	0.4959	50.4	2450.6	2501.0
15	3	5	0.0873	0.7513	0.8386	77.4	1528.8	1606.2
16	4	1	0.0596	0.7868	0.8464	4.6	1755.1	1759.7
17	4	2	0	0	0	0	1941.9	1941.9
18	4	3	0.0958	1.1014	1.1972	27.1	2038.7	2065.8
19	4	4	0.3502	1.0542	1.4044	154.9	2715.0	2869.9
20	4	5	0.2459	0.8026	1.0485	139.9	1744.6	1884.4

TABLE 13.   Mean values of the Odessa healthy plant (H) subset
of variables.   Column "T" identifies the treatment
number (c=control).   Columns "+" and "-" identify the
particular sporidial combination for each treatment.

TABLE 13

HEALTHY PLANT (H) VARIABLE ON ODESSA

T	+	-	1	2	3	4	5	6	7
c	-	-	50.0	78.7	1.6	57.8	37.0	39.2	2.2922
1	1	1	46.3	81.3	1.8	69.0	39.2	40.0	2.8382
2	1	2	45.0	71.3	1.6	55.1	34.9	40.0	2.2333
3	1	3	48.0	79.0	1.7	63.0	38.7	40.5	2.5522
4	1	4	46.0	61.0	1.3	50.4	36.7	40.4	2.0815
5	1	5	39.7	44.0	1.1	34.8	31.6	36.2	1.2614
6	2	1	41.0	45.0	1.1	34.5	31.5	41.0	1.4037
7	2	2	50.0	83.3	1.7	65.1	38.4	38.5	2.5475
8	2	3	45.0	65.7	1.5	53.2	36.2	38.9	2.1139
9	2	4	46.3	75.3	1.6	60.9	37.3	41.4	2.5064
10	2	5	40.7	56.0	1.4	46.1	33.2	37.2	1.7308
11	3	1	39.0	46.7	1.2	40.2	34.4	39.7	1.5872
12	3	2	50.0	63.0	1.3	37.7	29.1	33.3	1.3688
13	3	3	43.0	79.0	1.9	76.8	38.9	41.4	3.2846
14	3	4	43.0	68.7	1.6	59.4	36.6	40.2	2.4448
15	3	5	42.0	46.0	1.1	37.6	34.9	39.7	1.4876
16	4	1	39.7	51.3	1.3	44.0	33.1	38.0	1.6586
17	4	2	50.0	64.0	1.3	41.9	32.0	37.6	1.6133
18	4	3	42.3	57.0	1.3	49.1	35.9	38.5	1.8938
19	4	4	39.7	79.7	2.0	73.0	36.3	39.5	2.9518
20	4	5	39.7	56.0	1.4	48.2	33.0	40.9	2.0286

TABLE 13
(continued)

HEALTHY PLANT (H)
VARIABLE ON ODESSA

T	+	-	8	9	10
c	-	-	1.4453	77.0	2889.0
1	1	1	1.5697	80.4	3189.8
2	1	2	1.3952	78.4	2472.7
3	1	3	1.5593	78.9	3011.7
4	1	4	1.4927	77.2	2302.0
5	1	5	1.1469	73.5	1373.8
6	2	1	1.2865	81.7	1435.6
7	2	2	1.4771	76.1	3255.0
8	2	3	1.4118	76.4	2354.4
9	2	4	1.5416	78.7	2841.2
10	2	5	1.2189	78.5	1891.7
11	3	1	1.3638	78.7	1601.5
12	3	2	1.0251	74.5	1883.3
13	3	3	1.6158	78.9	31.5.5
14	3	4	1.4802	79.8	2514.9
15	3	5	1.3788	78.5	1591.9
16	4	1	1.2579	77.4	1813.5
17	4	2	1.2183	74.2	2093.3
18	4	3	1.3805	80.9	2107.8
19	4	4	1.4453	77.6	2851.8
20	4	5	1.3664	76.9	1835.1

TABLE 14.   Mean values of the Odessa completely diseased
         plant (C) subset of variables.   Column "T" identifies
         the treatment number (c=control).   Columns "+" and "-"
         identify the particular sporidial combination for each
         treatment.

TABLE 14

COMPLETELY DISEASED PLANT (C)
VARIABLE ON ODESSA

T	+	-	1	2	3	4	5	6	7
C	-	-	-	-	-	-	-	-	-
1	1	1	3.0	4.3	1.6	0.6763	0.2535	0.1562	36.0
2	1	2	4.3	6.0	1.3	0.5097	0.1032	0.0671	40.3
3	1	3	2.0	3.3	0.9	0.4293	0.0880	0.0501	25.2
4	1	4	4.0	5.3	1.3	0.4797	0.1102	0.0849	45.7
5	1	5	10.3	12.7	1.2	1.3643	0.1246	0.1068	41.2
6	2	1	9.0	11.7	1.3	1.3787	0.1508	0.1141	46.4
7	2	2	0.0	0.0	0.0	0.0	0.0	0.0	2.9
8	2	3	3.7	7.0	1.7	1.1237	0.2462	0.1435	52.7
9	2	4	3.3	4.0	1.1	0.1170	0.0289	0.0251	44.6
10	2	5	8.7	10.0	1.2	1.0607	0.1291	0.1058	48.6
11	3	1	10.3	15.3	1.3	2.3280	0.1880	0.1420	54.7
12	3	2	0.0	0.0	0.0	0.0	0.0	0.0	2.9
13	3	3	5.0	6.7	1.3	1.0577	0.1849	0.1401	49.9
14	3	4	5.7	9.7	1.5	0.8090	0.1161	0.0783	46.5
15	3	5	7.3	8.7	1.1	1.2980	0.1698	0.1481	47.2
16	4	1	10.0	11.7	1.2	1.4050	0.1624	0.1306	48.6
17	4	2	0.0	0.0	0.0	0.0	0.0	0.0	2.9
18	4	3	7.0	9.7	1.4	1.7683	0.2531	0.1765	51.8
19	4	4	8.3	12.7	1.5	1.7383	0.2067	0.1341	51.3
20	4	5	7.3	12.7	1.9	1.5250	0.2513	0.1285	44.0

TABLE 15.   Mean values of the Trebi partially diseased plant
        (P) subset of variables.   Column "T" identifies the
        treatment number (c=control).   Columns "+" and "-"
        identify the particular sporidial combination for each
        treatment.

TABLE 15

PARTIALLY DISEASED PLANT (P)
VARIABLE ON ODESSA

T	+	-	1	2	3	4	5	6	7	8
c	-	-	-	-	-	-	-	-	-	-
1	1	1	0.7	2.3	1.7	0.7	1.2	0.8	0.3	0.3727
2	1	2	0.7	1.7	0.7	1.0	1.7	0.7	1.0	0.0390
3	1	3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
4	1	4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
5	1	5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
6	2	1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
7	2	2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
8	2	3	1.3	8.7	5.0	3.7	3.8	1.9	1.9	0.8363
9	2	4	0.3	1.3	0.7	0.7	1.3	0.7	0.7	0.0253
10	2	5	0.7	2.3	1.0	1.3	1.2	0.5	0.7	0.1373
11	3	1	0.7	1.7	1.0	0.7	1.7	1.0	0.7	0.1910
12	3	2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
13	3	3	2.0	7.0	3.7	3.3	1.2	0.6	0.6	0.5293
14	3	4	1.3	3.0	1.3	1.7	2.3	1.0	1.3	0.0693
15	3	5	0.7	3.0	0.7	2.3	1.5	0.3	1.2	0.1157
16	4	1	0.3	0.7	0.3	0.3	0.7	0.3	0.3	0.0863
17	4	2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
18	4	3	0.7	2.0	1.0	1.0	2.0	1.0	1.0	0.1330
19	4	4	2.0	9.3	4.3	5.0	3.1	1.4	1.7	0.5753
20	4	5	3.0	7.7	3.3	4.3	1.7	0.7	0.9	0.3873

TABLE 15
(continued)

PARTIALLY DISEASED PLANT (P)
VARIABLE ON ODESSA

T	+	-	9	10	11	12	13	14
c	-	-	-	-	-	-	-	-
1	1	1	0.1863	0.0745	15.4	23.3	11.7	11.7
2	1	2	0.0390	0.0390	26.7	22.0	22.0	13.2
3	1	3	0.0	0.0	2.9	0.0	0.0	0.0
4	1	4	0.0	0.0	2.9	0.0	0.0	0.0
5	1	5	0.0	0.0	2.9	0.0	0.0	0.0
6	2	1	0.0	0.0	2.9	0.0	0.0	0.0
7	2	2	0.0	0.0	2.9	0.0	0.0	0.0
8	2	3	0.2908	0.0765	34.0	137.0	68.6	24.3
9	2	4	0.0253	0.0127	16.6	24.7	24.7	12.3
10	2	5	0.0687	0.0458	21.5	60.7	30.3	15.2
11	3	1	0.1910	0.1205	36.5	17.3	17.3	17.3
12	3	2	0.0	0.0	2.9	0.0	0.0	0.0
13	3	3	0.0882	0.0481	20.5	93.0	15.5	9.3
14	3	4	0.0527	0.0527	45.2	61.0	46.0	38.5
15	3	5	0.0578	0.0578	22.0	80.7	40.3	11.5
16	4	1	0.0863	0.0863	20.6	5.7	5.7	5.7
17	4	2	0.0	0.0	2.9	0.0	0.0	0.0
18	4	3	0.1330	0.0682	36.3	32.7	32.7	24.8
19	4	4	0.1899	0.0895	34.7	168.0	56.8	22.7
20	4	5	0.0859	0.0797	36.4	159.3	33.4	23.4

TABLE 15
(continued)

PARTIALLY DISEASED PLANT (P)
VARIABLE ON ODESSA

T	+	-	15	16	17	18
c	-	-	-	-	-	-
1	1	1	0.7880	0.3940	0.3940	23.1
2	1	2	0.9540	0.9540	0.5630	53.7
3	1	3	0.0	0.0	0.0	2.9
4	1	4	0.0	0.0	0.0	2.9
5	1	5	0.0	0.0	0.0	2.9
6	2	1	0.0	0.0	0.0	2.9
7	2	2	0.0	0.0	0.0	2.9
8	2	3	5.6077	2.8192	0.9978	49.1
9	2	4	0.9070	0.9070	0.4535	25.1
10	2	5	1.6727	0.8363	0.4182	24.0
11	3	1	0.6260	0.6260	0.6260	55.5
12	3	2	0.0	0.0	0.0	2.9
13	3	3	3.8843	0.6474	0.3884	26.3
14	3	4	2.1137	1.5147	1.2593	65.3
15	3	5	3.4253	1.7127	0.4893	27.8
16	4	1	0.1780	0.1780	0.1780	23.5
17	4	2	0.0	0.0	0.0	2.9
18	4	3	1.2853	1.2853	0.9920	44.6
19	4	4	7.0423	2.4114	0.9646	50.2
20	4	5	6.4520	1.3514	0.9469	48.0

TABLE 16.   Single sample t test results between treatment and
control means on Trebi (T).   Means of select variables
were tested for the probability of statistically
significant difference from mean control values.
Significant differences between means are shown by an
asterisk in the "SIG" column.   Absence of an asterisk
indicates no significant difference between the means.
(Ttab(a=.05(1),df=19)=1.729))

[P] = [PATHOGEN], [H] = [HOST]

TABLE 16

VARIABLE	VARIABLE MEAN	SE	CONTROL MEAN	Tcalc	SIG
TR1	47.6	0.36	47.5	0.138	
TR3	108.4	3.89	111.0	0.669	
TR6	2.18	0.077	2.2	0.326	
TR8	2.03	0.891	2.2	1.963	*
TWh[H]	2941.6	172.95	3258.3	1.831	*
TW[H]	3018.9	176.74	3258.3	1.354	
TH1	45.85	0.814	50.0	5.106	*
TH2	99.1	4.34	111.0	2.753	*
TH3	2.2	0.77	2.2	1.650	
TH4	68.8	3.61	68.8	0.220	
TH5	31.2	0.64	31.0	0.298	
TH6	47.4	0.38	47.9	1.479	
TH7	3.2820	0.16265	3.2957	0.084	
TH8	1.4843	0.32460	1.4854	0.033	
TH9	92.8	0.31	94.7	6.129	*
TH10	3162.3	186.18	3440.2	1.493	
TC3	1.2	0.16	2.2	6.441	*
TP5	1.7	0.25	2.2	2.014	*
TP12	83.3	28.25	3440.2	118.829	*
TP13	26.2	4.44	68.8	9.595	*
TP14	14.6	2.22	31.0	7.376	*
TP16	1.2808	0.22167	3.2957	9.090	*
TP17	0.6977	0.10460	1.4854	7.530	*
TP18	45.9	7.17	94.7	6.806	*

TABLE 17.   Single sample t test results between treatment and control means on Odessa (O).   Means of select variables were tested for the probability of statistically significant difference from mean control values. Significant differences between means are shown by an asterisk in the "SIG" column.   Absence of an asterisk indicates no significant difference between the means. (Ttab(a=.05(1),df=19)=1.729))

[P] = [PATHOGEN], [H] = [HOST]

TABLE 17

VARIABLE	VARIABLE MEAN	SE	CONTROL MEAN	Tcalc	SIG
OR1	51.0	0.50	56.2	10.413	*
OR3	73.8	2.77	78.7	1.781	*
OR6	1.5	0.06	1.6	2.198	*
OR8	1.3	0.06	1.6	5.222	*
OWh[H]	2179.1	127.20	2755.5	4.532	*
OW[H]	2218.5	128.69	2755.5	4.173	*
OH1	43.8	.82	50.0	7.555	*
OH2	63.7	2.87	78.7	5.231	*
OH3	1.5	0.06	1.6	2.394	*
OH4	52.0	2.81	57.8	2.063	*
OH5	35.1	0.62	37.0	3.096	*
OH6	39.1	0.43	39.2	0.127	
OH7	2.0794	0.12594	2.2922	1.689	
OH8	1.3816	0.03332	1.4453	1.912	*
OH9	95.6	0.35	95.3	0.857	
OH10	2232.7	125.18	2889.0	5.243	*
OC3	1.1	0.12	1.6	3.913	*
OP5	1.2	2.42	1.6	1.779	*
OP12	44.27	12.12	2889.0	234.674	*
OP13	20.3	4.58	57.8	8.205	*
OP14	11.5	2.42	37.0	10.518	*
OP16	0.7819	0.18488	2.2922	8.169	*
OP17	0.4336	0.09083	1.4453	11.139	*
OP18	31.1	6.25	95.3	10.272	*

TABLE 18.   One-way ANOVA and Duncan's multiple range test for select variables measured on Trebi (T).   The probability of statistically significant differences among variable means was calculated.   The "TEST" label in the source column represents the among means source of variability.   If significant differences exist between one variable mean and at least one of the other two, an asterisk is found in the "SIG" column.   Means were grouped with Duncan's multiple range test and were assigned an alphabetic character in the "GROUPING" column.   Means not differing significantly have the same alphabetic character.

TABLE 18

ANOVA (Variables TH1,TC1 and TP1)

SOURCE	DF	SS	MS	F	PR > F	SIG
TEST	2	25549.733333	12774.866670	1546.95	0.0001	*
ERROR	57	470.711111	8.258089			
TOTAL	59	26020.444444				

DUNCAN'S MULTIPLE RANGE TEST

VARIABLE	GROUPING	MEAN	N
TH1	A	45.833	20
TC1	B	2.933	20
TP1	B	1.233	20

TABLE 18
(continued)

ANOVA (Variables TH2,TC2 and TP2)

SOURCE	DF	SS	MS	F	PR > F	SIG
TEST	2	118671.670370	59335.835150	390.50	0.0001	*
ERROR	57	8661.061111	151.948440			
TOTAL	59	127332.731481				

DUNCAN'S MULTIPLE RANGE TEST

VARIABLE	GROUPING	MEAN	N
TH2	A	99.033	20
TC2	B	4.783	20
TP2	B	4.600	20

TABLE 18
(continued)

ANOVA (Variables TH3,TC3 and TP5)

SOURCE	DF	SS	MS	F	PR > F	SIG
TEST	2	9.82059259	4.91029630	7.27	0.0015	*
ERROR	57	38.49294444	0.67531481			
TOTAL	59	48.31353704				

DUNCAN'S MULTIPLE RANGE TEST

VARIABLE	GROUPING	MEAN	N
TH3	A	2.1417	20
TC3	A	1.6850	20
TP5	B	1.1517	20

TABLE 19.   Correlated groups t test results measured on Trebi
        (T).   The probability of a statistically significant
        difference between paired scores of certain variables
        was calculated.   A significant difference between
        paired scores was shown with an asterisk in the "SIG"
        column.   (N=20,Ttab(a=.05(2),df=19)=+/- 2.093))

        [P] = [PATHOGEN], [H] = [HOST]

TABLE 19

PAIRED VARIABLES			N	MEAN DIFFERENCE	SE	Tcalc	SIG
TR7	vs	TR8	20	-1.9	0.11	-17.72	*
TH2	vs	TP4	20	96.3	4.55	21.18	*
TH3	vs	TP7	20	1.3	0.18	7.11	*
TH4	vs	TP13	20	42.6	5.94	7.18	*
TH5	vs	TP14	20	16.5	2.16	7.66	*
TH7	vs	TP16	20	2.0013	0.29042	6.89	*
TH8	vs	TP17	20	0.7866	0.10270	7.66	*
TH9	vs	TP18	20	35.5	5.65	6.29	*
TH10	vs	TP12	20	2968.4	236.02	12.58	*
TC2	vs	TP3	20	2.9	1.05	2.77	*
TC3	vs	TP6	20	0.4	0.11	3.12	*
TC4	vs	TP8	20	0.3553	0.13741	2.59	*
TC5	vs	TP9	20	0.0418	0.01553	2.69	*
TC6	vs	TP10	20	0.0191	0.00668	2.86	*
TC7	vs	TP11	20	7.1	2.95	2.41	*
TP3	vs	TP4	20	-0.8	0.47	-1.79	
TP6	vs	TP7	20	-0.1	0.09	-1.07	
TWp[P]	vs	TWc[P]	20	-0.1748	0.07533	-2.32	*
TWp[H]	vs	TWh[H]	20	-2858.9	177.14	-16.14	*

TABLE 20.   One-way ANOVA and Duncan's multiple range test for
        select variables measured on Odessa (O).   The
        probability of statistically significant differences
        among variable means was calculated.   The "TEST" label
        in the source column represents the among means source
        of variability.   If significant differences exist
        between one variable mean and at least one of the other
        two, an asterisk is found in the "SIG" column.   Means
        were grouped with Duncan's multiple range test and were
        assigned an alphabetic character in the "GROUPING"
        column.   Means not differing significantly have the
        same alphabetic character.

TABLE 20

ANOVA (Variables OH1,OC1 and OP1)

SOURCE	DF	SS	MS	F	PR > F	SIG
TEST	2	22339.300000	11169.650000	1248.84	0.0001	*
ERROR	57	509.811111	8.944054			
TOTAL	59	22849.111111	2.990661			

DUNCAN'S MULTIPLE RANGE TEST

VARIABLE	GROUPING	MEAN	N
OH1	A	43.817	20
OC1	B	5.467	20
OP1	C	0.717	20

TABLE 20
(continued)

ANOVA (VARIABLES OH2,OC2 and OP2)

SOURCE	DF	SS	MS	F	PR > F	SIG
TEST	2	46065.525925	23032.762960	336.74	0.0001	*
ERROR	57	3898.777777	68.399610			
TOTAL	59	49964.303703				

DUNCAN'S MULTIPLE RANGE TEST

VARIABLE	GROUPING	MEAN	N
OH2	A	63.667	20
OC2	B	7.567	20
OP2	B	2.533	20

TABLE 20
(continued)

ANOVA (Variables OH3,OC3 and OP5)

SOURCE	DF	SS	MS	F	PR > F	SIG
TEST	2	1.21737037	0.60868519	1.15	0.3231	
ERROR	57	30.10816667	0.52821345			
TOTAL	59	31.32553704				

DUNCAN'S MULTIPLE RANGE TEST

VARIABLE	GROUPING	MEAN	N
OH3	A	1.4517	20
OC3	A	1.1600	20
OP5	A	1.1400	20

TABLE 21.   Correlated groups t test results measured on
      Odessa (O).   The probability of a statistically
      significant difference between paired scores of certain
      variables was calculated.   A significant difference
      between paired scores was shown with an asterisk in the
      "SIG" column.   (N=20,Ttab(a=.05(2),df=19)=+/- 2.093))

      [P] = [PATHOGEN], [H] = [HOST]

TABLE 21

PAIRED VARIABLES		N	MEAN DIFFERENCE	SE	Tcalc	SIG
OR7	vs OR8	20	-1.1	0.07	-15.85	*
OH2	vs OP4	20	62.4	2.91	21.42	*
OH3	vs OP7	20	0.8	0.13	6.53	*
OH4	vs OP13	20	31.8	4.87	6.51	*
OH5	vs OP14	20	23.6	2.42	9.75	*
OH7	vs OP16	20	1.2975	0.20050	6.47	*
OH8	vs OP17	20	0.9480	0.08970	10.57	*
OH9	vs OP18	20	51.0	4.69	10.89	*
OH10	vs OP12	20	2081.4	130.76	15.92	*
OC2	vs OP3	20	6.3	1.00	6.39	*
OC3	vs OP6	20	0.6	0.10	5.75	*
OC4	vs OP8	20	0.7785	0.13729	5.67	*
OC5	vs OP9	20	0.0636	0.01311	4.85	*
OC6	vs OP10	20	0.0540	0.00847	6.38	*
OC7	vs OP11	20	19.8	3.03	6.55	*
OP3	vs OP4	20	-0.1	0.14	-0.48	
OP6	vs OP7	20	-0.1	0.06	-1.03	
OWp[P] vs OWc[P]		20	-0.4263	0.08265	-5.16	*
OWp[H] vs OWh[H]		20	-2147.5	131.61	-16.32	*

TABLE 22.   Analysis of variance of R and fitness (W)
     variables on Trebi (T).   Sources of variability include
     three main effects components; plus sporidia (+), minus
     sporidia (-), and replicates (rep); as well as all
     possible second order interactions; sporidial
     interactions (+x-), and two types of sporidia replicate
     interactions (+xrep, and -xrep).   The third order
     interaction component (+x-xrep) was redefined as the
     error component.   Degrees of freedom, mean squares, F
     and pseudo-F values were calculated.   It was necessary
     to calculate pseudo-F values for the three main effects
     components because of the absence of suitable
     denominator mean squares.   Components with
     statistically significant F values (alpha=.05) have an
     asterisk in the "SIG" column.   The relative
     contribution of each component to total variability (%
     VAR) was determined using the following expected mean
     squares table:

```
EMS+      = Verror + 3V+x-    + 5V+xrep + 15V+
EMS-      = Verror + 3V+x-    + 4V-xrep + 12V-
EMSrep    = Verror + 5V+xrep + 4V-xrep + 20Vrep
EMS+x-    = Verror + 3V+x-
EMS+xrep  = Verror + 5V+xrep
EMS-xrep  = Verror + 4V-xrep
EMSerror  = Verror
```

EMS = expected mean square
V = variance

TABLE 22

VARIABLE TR1

GERMINATION RATE OF THE 110 TREATED SEEDS
ORIGINALLY PLANTED

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	17.31305556	2.25		7.1
2	-	4	11.06266667	1.28		2.6
3	rep	2	77.72866667	5.39	*	26.7
4	+ x -	12	5.03777778	.64		0
5	+ x rep	6	6.14222222	.79		0
6	- x rep	8	9.72679167	1.24		3.6
7	error	24	7.82423611			59.9

VARIABLE TR2

PERCENT OF PLANTS SMUTTED

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	132.56861111	1.27		2.3
2	-	4	527.62775000	5.56	*	38.5
3	rep	2	165.37016667	3.78		7.3
4	+ x -	12	88.26819444	2.95		19.6
5	+ x rep	6	39.66727778	1.32		2.0
6	- x rep	8	11.96225000	.40		0
7	error	24	29.95852778			30.3

VARIABLE TR3

NUMBER OF HEADS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	1125.11111111	1.30		3.0
2	-	4	916.01666667	1.24		2.8
3	rep	2	2599.80000000	4.50	*	14.1
4	+ x -	12	923.52777778	1.53		12.0
5	+ x rep	6	409.04444445	.68		0
6	- x rep	8	303.09166667	.50		0
7	error	24	605.16944444			68.2

TABLE 22
(continued)

VARIABLE TR4

PERCENT OF HEADS SMUTTED

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	134.50733333	1.66		4.8
2	-	4	527.75275000	7.10	*	43.6
3	rep	2	93.73216667	2.83		4.4
4	+ x -	12	67.23219444	2.17		13.2
5	+ x rep	6	32.62150000	1.05		.4
6	- x rep	8	11.45737500	.37		0
7	error	24	30.96448611			33.7

VARIABLE TR5

NUMBER OF HEADS FROM DISEASED PLANTS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	39.13333333	.65		0
2	-	4	578.19166667	2.58		19.4
3	rep	2	619.81666667	3.95	*	15.0
4	+ x -	12	143.14722222	1.52		9.2
5	+ x rep	6	63.28333333	.67		0
6	- x rep	8	117.50416667	1.25		3.3
7	error	24	94.02638889			53.1

VARIABLE TR6

AVERAGE NUMBER OF HEADS PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.41022222	1.24		2.3
2	-	4	.39225000	1.32		3.6
3	rep	2	1.03516667	4.50	*	13.6
4	+ x -	12	.37258333	1.44		10.3
5	+ x rep	6	.16738889	.65		0
6	- x rep	8	.12037500	.47		0
7	error	24	.25870833			70.2

TABLE 22
(continued)

VARIABLE TR7

AVERAGE NUMBER OF DISEASED HEADS PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.02775111	1.49		3.2
2	-	4	.12836667	4.49	*	34.0
3	rep	2	.05952667	4.31	*	10.2
4	+ x -	12	.02022889	1.88		11.9
5	+ x rep	6	.00557111	.52		0
6	- x rep	8	.01075167	1.00		0
7	error	24	.01077389			40.7

VARIABLE TR8

AVERAGE NUMBER OF HEALTHY HEADS PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.63794444	1.65		6.3
2	-	4	.76141667	1.89		10.8
3	rep	2	.71266667	2.91		8.6
4	+ x -	12	.35863889	1.66		13.4
5	+ x rep	6	.15977778	.74		0
6	- x rep	8	.15954167	.74		0
7	error	24	.21609722			60.9

VARIABLE TR9

AVERAGE NUMBER OF HEADS PER DISEASED PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.70444444	.58		0
2	-	4	6.44666667	2.55		21.3
3	rep	2	2.98716667	1.75		4.8
4	+ x -	12	1.75722222	1.84		15.2
5	+ x rep	6	1.11027778	1.16		1.7
6	- x rep	8	1.14716667	1.20		2.7
7	error	24	.95638889			54.3

TABLE 22
(continued)

VARIABLE TR10

AVERAGE NUMBER OF DISEASED HEADS PER DISEASED PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.28416667	.56		0
2	-	4	3.44766667	2.82	*	25.1
3	rep	2	.18150000	1.08		.3
4	+ x -	12	1.02722222	2.50	*	24.9
5	+ x rep	6	.20750000	.50		0
6	- x rep	8	.34129167	.83		0
7	error	24	.41118056			49.8

VARIABLE TR11

AVERAGE NUMBER OF HEALTHY HEADS PER DISEASED PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.33814444	.85		0
2	-	4	.97423917	1.57		7.9
3	rep	2	1.75704000	2.43		12.0
4	+ x -	12	.41792917	1.08		1.9
5	+ x rep	6	.43588445	1.12		1.8
6	- x rep	8	.44748792	1.15		2.8
7	error	24	.38831292			73.7

VARIABLE TR12

SPORE WEIGHT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	1.57609766	2.35		6.5
2	-	4	4.91638560	4.29	*	25.8
3	rep	2	2.23564712	3.94	*	8.1
4	+ x -	12	.79862030	.93		0
5	+ x rep	6	.23776090	.28		0
6	- x rep	8	.54646264	.64		0
7	error	24	.85448630			59.7

TABLE 22
(continued)

VARIABLE TR13

AVERAGE SPORE WEIGHT PER DISEASED PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.00484705	.90		0
2	-	4	.07684853	3.61	*	26.4
3	rep	2	.00773068	1.36		1.4
4	+ x -	12	.01538425	1.03		.7
5	+ x rep	6	.00663535	.44		0
6	- x rep	8	.01002367	.67		0
7	error	24	.01496982			71.5

VARIABLE TR14

AVERAGE SPORE WEIGHT PER DISEASED HEAD

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.00316823	1.42		2.5
2	-	4	.01973971	4.82	*	34.1
3	rep	2	.00385515	2.32		4.1
4	+ x -	12	.00290201	1.23		4.2
5	+ x rep	6	.00099843	.42		0
6	- x rep	8	.00167958	.71		0
7	error	24	.00235890			55.1

VARIABLE TR15

AVERAGE SPORE GERMINATION RATE PER DISEASED HEAD

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	503.79066667	.97		0
2	-	4	1926.41108333	2.56		25.4
3	rep	2	310.32516667	1.41		1.5
4	+ x -	12	568.66997222	4.61	*	36.3
5	+ x rep	6	75.22516667	.61		0
6	- x rep	8	231.81808333	1.88		6.6
7	error	24	123.32113889			30.1

TABLE 22
(continued)

VARIABLE TR16

AVERAGE NUMBER OF SEEDS PER DISEASED PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	271.52061111	.81		0
2	-	4	954.36983333	1.50		7.0
3	rep	2	1808.96550000	2.52		12.3
4	+ x -	12	441.67672222	1.05		1.4
5	+ x rep	6	413.24994445	.99		0
6	- x rep	8	471.96383333	1.13		2.4
7	error	24	418.81022222			76.8

VARIABLE TR17

AVERAGE NUMBER OF SEEDS PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	1430.05483333	1.78		6.9
2	-	4	879.81641667	1.25		2.8
3	rep	2	2593.72516667	4.99	*	15.0
4	+ x -	12	809.04775000	1.49		10.6
5	+ x rep	6	299.95050000	.55		0
6	- x rep	8	329.17954167	.61		0
7	error	24	543.44904167			64.8

VARIABLE TWp [PATHOGEN]

PATHOGEN FITNESS
(CALCULATED FROM P SUBSET OF VARIABLES)

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.01665366	1.33		1.5
2	-	4	.15645464	1.94		12.2
3	rep	2	.15823547	2.13		8.1
4	+ x -	12	.02720927	.61		0
5	+ x rep	6	.01887408	.42		0
6	- x rep	8	.07610722	1.71		11.8
7	error	24	.04442487			66.4

TABLE 22
(continued)

VARIABLE TWc [PATHOGEN]

PATHOGEN FITNESS
(CALCULATED FROM C SUBSET OF VARIABLES)

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.38715007	2.08		7.3
2	-	4	.97784340	5.23	*	30.6
3	rep	2	.15266710	2.58		3.5
4	+ x -	12	.17482332	1.39		6.7
5	+ x rep	6	.07180571	.57		0
6	- x rep	8	.03645610	.29		0
7	error	24	.12611129			51.9

VARIABLE TW [PATHOGEN]

TOTAL PATHOGEN FITNESS
(Wp [PATHOGEN] + Wc [PATHOGEN])

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.43708659	2.34		6.4
2	-	4	1.40965597	4.35	*	26.6
3	rep	2	.59755085	4.31	*	8.0
4	+ x -	12	.23556903	1.02		.3
5	+ x rep	6	.05059528	.22		0
6	- x rep	8	.14175317	.61		0
7	error	24	.23157868			58.5

VARIABLE TWp [HOST]

HOST FITNESS
(CALCULATED FROM P SUBSET OF VARIABLES)

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	18083.99743427	.81		0
2	-	4	66032.07611782	1.27		3.8
3	rep	2	153248.09576362	3.12		13.5
4	+ x -	12	44277.72421213	1.18		4.7
5	+ x rep	6	23927.03935402	.64		0
6	- x rep	8	37174.06968837	.99		0
7	error	24	37470.31685946			78.0

TABLE 22
(continued)

VARIABLE TWh [HOST]

HOST FITNESS
(CALCULATED FROM H SUBSET OF VARIABLES)

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	2597099.20169904	1.63		5.7
2	-	4	2111770.22994770	1.29		3.6
3	rep	2	5260971.73765141	4.34	*	14.6
4	+ x -	12	1637807.68537828	1.50		10.9
5	+ x rep	6	622548.47648524	.57		0
6	- x rep	8	840503.07338025	.77		0
7	error	24	1089905.48815647			65.2

VARIABLE TW [HOST]

TOTAL HOST FITNESS
(Wp [HOST] + Wh [HOST])

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	3002297.28216660	1.80		6.6
2	-	4	1870533.04314847	1.26		2.9
3	rep	2	6196930.11705426	5.66	*	16.2
4	+ x -	12	1749705.85335652	1.42		9.1
5	+ x rep	6	607651.50908114	.49		0
6	- x rep	8	705162.13728359	.57		0
7	error	24	1233359.50130055			65.2

TABLE 23.   Analysis of variance of H variables on Trebi (T).
    Sources of variability include three main effects
    components; plus sporidia (+), minus sporidia (-), and
    replicates (rep); as well as all possible second order
    interactions; sporidial interactions (+x-), and two
    types of sporidia replicate interactions (+xrep, and
    -xrep).   The third order interaction component
    (+x-xrep) was redefined as the error component.
    Degrees of freedom, mean squares, F and pseudo-F values
    were calculated.   It was necessary to calculate
    pseudo-F values for the three main effects components
    because of the absence of suitable denominator mean
    squares.   Components with statistically significant F
    values (alpha=.05) have an asterisk in the "SIG" column.
    The relative contribution of each component to total
    variability (% VAR) was determined using the following
    expected mean square table:

```
EMS+      = Verror + 3V+x-    + 5V+xrep + 15V+
EMS-      = Verror + 3V+x-    + 4V-xrep + 12V-
EMSrep    = Verror + 5V+xrep + 4V-xrep + 20Vrep
EMS+x-    = Verror + 3V+x-
EMS+xrep  = Verror + 5V+xrep
EMS-xrep  = Verror + 4V-xrep
EMSerror  = Verror
-----------------------------------------------------------
EMS = expected mean square
V = variance
```

TABLE 23

VARIABLE TH1

NUMBER OF HEALTHY PLANTS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	31.71111111	1.39		3.3
2	–	4	108.16666667	4.29	*	32.0
3	rep	2	40.81666667	4.22	*	8.1
4	+ x –	12	22.26666667	2.55	*	19.3
5	+ x rep	6	6.72777778	.77		0
6	– x rep	8	5.00416667	.57		0
7	error	24	8.72083333			37.3

VARIABLE TH2

NUMBER OF HEADS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	1411.44444444	1.54		5.5
2	–	4	2235.77500000	2.00		14.1
3	rep	2	1312.71666667	1.77		4.9
4	+ x –	12	789.31944444	1.64		12.7
5	+ x rep	6	442.29444445	.92		0
6	– x rep	8	570.92500000	1.18		2.7
7	error	24	482.66944444			60.1

VARIABLE TH3

AVERAGE NUMBER OF HEADS PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.40994444	1.18		2.0
2	–	4	.40041667	1.21		2.9
3	rep	2	.79216667	2.86		10.4
4	+ x –	12	.35286111	1.53		12.7
5	+ x rep	6	.19061111	.83		0
6	– x rep	8	.16716667	.72		0
7	error	24	.23061111			72.0

TABLE 23
(continued)

VARIABLE TH4

AVERAGE NUMBER OF SEEDS PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	1012.02911111	1.29		2.8
2	-	4	522.91125000	.92		0
3	rep	2	2956.13316667	5.23	*	16.8
4	+ x -	12	879.60980556	1.50		11.6
5	+ x rep	6	355.77361111	.61		0
6	- x rep	8	321.83462500	.55		0
7	error	24	584.85034722			68.8

VARIABLE TH5

AVERAGE NUMBER OF SEEDS PER HEAD

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	29.56194444	.89		0
2	-	4	11.33025000	.47		0
3	rep	2	232.23316667	12.42	*	41.4
4	+ x -	12	29.54791667	4.55	*	29.0
5	+ x rep	6	10.98694445	1.69		3.4
6	- x rep	8	8.24020833	1.27		1.6
7	error	24	6.49695833			24.5

VARIABLE TH6

THOUSAND SEED WEIGHT,
SEEDS RANDOMLY SELECTED FROM ALL HEALTHY PLANTS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	4.46905556	.77		0
2	-	4	6.73641667	.78		0
3	rep	2	150.89116667	13.70	*	46.8
4	+ x -	12	10.67919444	1.50		7.6
5	+ x rep	6	4.35938889	.61		0
6	- x rep	8	7.16929167	1.01		.1
7	error	24	7.09723611			45.4

TABLE 23
(continued)

VARIABLE TH7

AVERAGE SEED WEIGHT PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	1.81376015	1.18		1.6
2	-	4	.97111379	.92		0
3	rep	2	10.64086477	7.20	*	24.2
4	+ x -	12	1.86823488	1.28		6.3
5	+ x rep	6	.90255312	.62		0
6	- x rep	8	.77737790	.53		0
7	error	24	1.46140572			67.9

VARIABLE TH8

AVERAGE SEED WEIGHT PER HEAD

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.04737899	1.19		1.0
2	-	4	.04000611	1.00		.0
3	rep	2	1.01391597	14.50	*	37.1
4	+ x -	12	.08017736	.94		0
5	+ x rep	6	.03134368	.37		0
6	- x rep	8	.04444303	.52		0
7	error	24	.08516773			61.8

VARIABLE TH9

SEED GERMINATION RATE (FOR SEEDS FROM H6)

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	5.46727778	.70		0
2	-	4	3.43858333	.66		0
3	rep	2	65.65216667	3.05		19.2
4	+ x -	12	7.94880556	.85		0
5	+ x rep	6	13.17594445	1.41		5.9
6	- x rep	8	11.38820833	1.22		3.9
7	error	24	9.32143056			71.0

TABLE 23
(continued)

VARIABLE TH10

NUMBER OF SEEDS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	3126059.88711178	1.63		6.1
2	–	4	2460746.50558384	1.27		3.6
3	rep	2	5351548.64216757	3.63	*	12.9
4	+ x –	12	1864419.80002758	1.54		11.8
5	+ x rep	6	792952.22861079	.65		0
6	– x rep	8	1016804.55070808	.84		0
7	error	24	1211146.45298617			65.7

TABLE 24.   Analysis of variance of C variables on Trebi (T).
    Sources of variability include three main effects
    components; plus sporidia (+), minus sporidia (-), and
    replicates (rep); as well as all possible second order
    interactions; sporidial interactions (+x-), and two
    types of sporidia replicate interactions (+xrep, and
    -xrep).   The third order interaction component
    (+x-xrep) was redefined as the error component.
    Degrees of freedom, mean squares, F and pseudo-F values
    were calculated.   It was necessary to calculate
    pseudo-F values for the three main effects components
    because of the absence of suitable denominator mean
    squares.   Components with statistically significant F
    values (alpha=.05) have an asterisk in the "SIG" column.
    The relative contribution of each component to total
    variability (% VAR) was determined using the following
    expected mean squares table:

```
EMS+       = Verror + 3V+x-    + 5V+xrep + 15V+
EMS-       = Verror + 3V+x-    + 4V-xrep + 12V-
EMSrep     = Verror + 5V+xrep + 4V-xrep + 20Vrep
EMS+x-     = Verror + 3V+x-
EMS+xrep   = Verror + 5V+xrep
EMS-xrep   = Verror + 4V-xrep
EMSerror   = Verror
----------------------------------------------------------
EMS = expected mean square
V = variance
```

TABLE 24

VARIABLE TC1

NUMBER OF COMPLETELY DISEASED PLANTS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	31.42222222	2.24		9.3
2	-	4	66.18333333	4.69	*	32.3
3	rep	2	7.31666667	3.15	*	2.9
4	+ x -	12	13.78333333	2.67	*	19.8
5	+ x rep	6	2.53888889	.49		0
6	- x rep	8	1.42083333	.27		0
7	error	24	5.17083333			35.7

VARIABLE TC2

NUMBER OF HEADS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	70.59444444	2.03		7.0
2	-	4	199.56666667	4.93	*	34.6
3	rep	2	39.21666667	3.88	*	4.9
4	+ x -	12	35.84444444	2.37	*	16.8
5	+ x rep	6	6.32777778	.42		0
6	- x rep	8	7.65416667	.51		0
7	error	24	15.09861111			36.7

VARIABLE TC3

AVERAGE NUMBER OF HEADS PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.35261111	.59		0
2	-	4	3.85391667	2.62		21.6
3	rep	2	.59116667	1.46		1.8
4	+ x -	12	1.25191667	2.09		20.5
5	+ x rep	6	.37094445	.62		0
6	- x rep	8	.44429167	.74		0
7	error	24	.59795833			56.2

TABLE 24
(continued)

VARIABLE TC4

SPORE WEIGHT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	1.39219033	5.11	*	9.6
2	–	4	3.37456973	12.46	*	28.4
3	rep	2	3.53597800	10.34	*	17.4
4	+ x –	12	.14230979	.31		0
5	+ x rep	6	.22092943	.48		0
6	– x rep	8	.16569707	.36		0
7	error	24	.46309152			44.6

VARIABLE TC5

AVERAGE SPORE WEIGHT PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.01728456	1.09		.8
2	–	4	.08472522	2.82	*	21.3
3	rep	2	.01455143	1.13		.8
4	+ x –	12	.02050058	1.00		0
5	+ x rep	6	.01413103	.69		0
6	– x rep	8	.01685805	.82		0
7	error	24	.02053559			77.2

VARIABLE TC6

AVERAGE SPORE WEIGHT PER HEAD

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.00365424	1.02		.2
2	–	4	.02042098	4.07	*	34.1
3	rep	2	.00646162	1.92		4.9
4	+ x –	12	.00329793	1.70		10.9
5	+ x rep	6	.00218005	1.12		1.1
6	– x rep	8	.00219975	1.13		1.5
7	error	24	.00194486			47.2

TABLE 24
(continued)

VARIABLE TC7

AVERAGE SPORE GERMINATION RATE PER HEAD

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	310.81661111	.60		0
2	-	4	1809.78641667	2.42		21.9
3	rep	2	364.28816667	1.18		1.0
4	+ x -	12	596.97063889	3.20	*	30.7
5	+ x rep	6	237.68461111	1.27		2.3
6	- x rep	8	227.69004167	1.22		2.3
7	error	24	186.62676389			41.9

TABLE 25.   Analysis of variance of P variables on Trebi (T).
       Sources of variability include three main effects
       components; plus sporidia (+), minus sporidia (−), and
       replicates (rep); as well as all possible second order
       interactions; sporidial interactions (+x−), and two
       types of sporidia replicate interactions (+xrep, and
       −xrep).   The third order interaction component
       (+x−xrep) was redefined as the error component.
       Degrees of freedom, mean squares, F and pseudo-F values
       were calculated.   It was necessary to calculate
       pseudo-F values for the three main effects components
       because of the absence of suitable denominator mean
       squares.   Components with statistically significant F
       values (alpha=.05) have an asterisk in the "SIG" column.
       The relative contribution of each component to total
       variability (% VAR) was determined using the following
       expected mean squares table:

```
EMS+      = Verror + 3V+x-   + 5V+xrep + 15V+
EMS-      = Verror + 3V+x-   + 4V-xrep + 12V-
EMSrep    = Verror + 5V+xrep + 4V-xrep + 20Vrep
EMS+x-    = Verror + 3V+x-
EMS+xrep  = Verror + 5V+xrep
EMS-xrep  = Verror + 4V-xrep
EMSerror  = Verror
-------------------------------------------------------
EMS = expected mean square
V = variance
```

TABLE 25

VARIABLE TP1

NUMBER OF DISEASED PLANTS WITH SEEDS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.02222222	.46		0
2	-	4	13.43333333	2.41		18.0
3	rep	2	16.11666667	3.89	*	16.1
4	+ x -	12	3.24444444	1.52		8.8
5	+ x rep	6	1.47222222	.69		0
6	- x rep	8	3.22083333	1.51		6.5
7	error	24	2.13194444			50.6

VARIABLE TP2

NUMBER OF HEADS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	16.84444444	.71		0
2	-	4	208.80833333	1.62		8.5
3	rep	2	358.35000000	3.13		13.9
4	+ x -	12	83.99722222	1.11		2.6
5	+ x rep	6	46.86111111	.62		0
6	- x rep	8	91.87083333	1.21		3.8
7	error	24	75.82638889			71.3

VARIABLE TP3

NUMBER OF DISEASED HEADS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	1.08333333	.72		0
2	-	4	35.27500000	2.22		16.3
3	rep	2	37.61666667	2.74		12.0
4	+ x -	12	7.37500000	.98		0
5	+ x rep	6	4.55000000	.61		0
6	- x rep	8	11.88750000	1.59		9.2
7	error	24	7.48750000			62.5

TABLE 25
(continued)

VARIABLE TP4

NUMBER OF HEALTHY HEADS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	10.99444444	.74		0
2	-	4	73.85833333	1.38		5.2
3	rep	2	163.81666667	3.27		14.3
4	+ x -	12	42.48055556	1.13		3.4
5	+ x rep	6	23.06111111	.61		0
6	- x rep	8	38.48333333	1.03		.5
7	error	24	37.50555556			76.7

VARIABLE TP5

AVERAGE NUMBER OF HEADS PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.48238889	.66		0
2	-	4	14.02391667	3.95	*	24.0
3	rep	2	9.01550000	1.93		6.5
4	+ x -	12	1.61447222	.55		0
5	+ x rep	6	3.52172222	1.20		2.7
6	- x rep	8	2.67404167	.91		0
7	error	24	2.92776389			66.7

VARIABLE TP6

AVERAGE NUMBER OF DISEASED HEADS PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.12977778	.98		0
2	-	4	3.82975000	7.27	*	29.7
3	rep	2	.56816667	1.46		1.9
4	+ x -	12	.31241667	.41		0
5	+ x rep	6	.59394445	.78		0
6	- x rep	8	.31900000	.42		0
7	error	24	.76200000			68.5

TABLE 25
(continued)

VARIABLE TP7

AVERAGE NUMBER OF HEALTHY HEADS PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.17298833	.53		0
2	-	4	3.46061833	2.18		13.4
3	rep	2	5.12148167	2.31		11.6
4	+ x -	12	.77145500	.81		0
5	+ x rep	6	1.36824167	1.43		5.5
6	- x rep	8	1.25847333	1.32		5.1
7	error	24	.95688333			64.4

VARIABLE TP8

SPORE WEIGHT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.03656099	1.57		2.0
2	-	4	.48579490	2.06		12.7
3	rep	2	.50772827	2.53		9.3
4	+ x -	12	.07926974	.57		0
5	+ x rep	6	.03240491	.23		0
6	- x rep	8	.22345354	1.61		10.0
7	error	24	.13881002			65.9

VARIABLE TP9

AVERAGE SPORE WEIGHT PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.00238528	1.38		1.6
2	-	4	.05836304	4.07	*	20.3
3	rep	2	.00666079	1.01		0
4	+ x -	12	.00450695	.24		0
5	+ x rep	6	.01068481	.57		0
6	- x rep	8	.01441916	.77		0
7	error	24	.01863158			78.1

TABLE 25
(continued)

VARIABLE TP10

AVERAGE SPORE WEIGHT PER HEAD

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.00004321	1.36		1.3
2	-	4	.01865939	4.16	*	22.3
3	rep	2	.00371102	1.36		1.7
4	+ x -	12	.00150026	.30		0
5	+ x rep	6	.00221715	.44		0
6	- x rep	8	.00418991	.84		0
7	error	24	.00501163			74.6

VARIABLE TP11

AVERAGE SPORE GERMINATION RATE PER HEAD

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	128.72994444	.56		0
2	-	4	2719.19875000	5.85	*	33.3
3	rep	2	749.96016667	1.59		3.3
4	+ x -	12	359.02230556	.96		0
5	+ x rep	6	535.59927778	1.43		5.0
6	- x rep	8	170.02537500	.45		0
7	error	24	375.05143056			58.4

VARIABLE TP12

NUMBER OF SEEDS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	18501.55555556	.80		0
2	-	4	75857.20833333	1.28		4.0
3	rep	2	173688.46666667	3.17		13.6
4	+ x -	12	49894.37500000	1.18		4.8
5	+ x rep	6	25883.48888889	.61		0
6	- x rep	8	42259.00833333	1.00		.1
7	error	24	42105.97500000			77.6

TABLE 25
(continued)

VARIABLE TP13

AVERAGE NUMBER OF SEEDS PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	339.11438889	.60		0
2	-	4	3088.60308333	1.75		9.4
3	rep	2	5729.90016667	2.33		12.4
4	+ x -	12	853.77786111	.84		0
5	+ x rep	6	1400.94972222	1.38		4.9
6	- x rep	8	1494.56245833	1.47		7.7
7	error	24	1017.43923611			65.5

VARIABLE TP14

AVERAGE NUMBER OF SEEDS PER HEALTHY HEAD

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	9.54866667	.51		0
2	-	4	935.39891667	4.50	*	24.0
3	rep	2	431.23266667	1.97		5.1
4	+ x -	12	179.09547222	.85		0
5	+ x rep	6	251.19000000	1.19		2.5
6	- x rep	8	75.96204167	.36		0
7	error	24	211.86326389			68.4

VARIABLE TP15

SEED WEIGHT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	51.09973451	.83		0
2	-	4	186.98489757	1.27		3.8
3	rep	2	430.33494527	3.08		13.5
4	+ x -	12	121.30637884	1.17		4.5
5	+ x rep	6	65.28459505	.63		0
6	- x rep	8	108.18709952	1.05		.9
7	error	24	103.42683238			77.4

TABLE 25
(continued)

VARIABLE TP16

AVERAGE SEED WEIGHT PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	1.40939377	.71		0
2	-	4	7.71249796	1.65		8.2
3	rep	2	14.93139679	2.17		11.5
4	+ x -	12	1.99044076	.74		0
5	+ x rep	6	3.79408857	1.40		5.3
6	- x rep	8	4.32460045	1.60		9.8
7	error	24	2.70237471			65.2

VARIABLE TP17

AVERAGE SEED WEIGHT PER HEALTHY HEAD

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.01921060	.54		0
2	-	4	2.08465134	4.14	*	21.0
3	rep	2	1.17580962	1.87		5.1
4	+ x -	12	.39450527	.70		0
5	+ x rep	6	.68476766	1.22		3.0
6	- x rep	8	.24494189	.43		0
7	error	24	.56345846			70.8

VARIABLE TP18

AVERAGE SEED GERMINATION RATE PER HEALTHY HEAD

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	365.70061111	.67		0
2	-	4	6346.54150000	5.91	*	31.2
3	rep	2	2050.60266667	1.77		4.1
4	+ x -	12	784.38727778	.78		0
5	+ x rep	6	1264.16711111	1.26		3.2
6	- x rep	8	459.46037500	.46		0
7	error	24	1001.24731944			61.5

TABLE 26.   Analysis of variance of R and fitness (W)
       variables on Odessa (O).   Sources of variability
       include three main effects components; plus sporidia
       (+), minus sporidia (-), and replicates (rep); as well
       as all possible second order interactions; sporidial
       interactions (+x-), and two types of sporidia replicate
       interactions (+xrep, and -xrep).   The third order
       interaction component (+x-xrep) was redefined as the
       error component.   Degrees of freedom, mean squares, F
       and pseudo-F values were calculated.   It was necessary
       to calculate pseudo-F values for the three main effects
       components because of the absence of suitable
       denominator mean squares.   Components with
       statistically significant F values (alpha=.05) have an
       asterisk in the "SIG" column.   The relative
       contribution of each component to total variability (%
       VAR) was determined using the following expected mean
       squares table:

```
EMS+      = Verror + 3V+x-   + 5V+xrep + 15V+
EMS-      = Verror + 3V+x-   + 4V-xrep + 12V-
EMSrep    = Verror + 5V+xrep + 4V-xrep + 20Vrep
EMS+x-    = Verror + 3V+x-
EMS+xrep  = Verror + 5V+xrep
EMS-xrep  = Verror + 4V-xrep
EMSerror  = Verror
----------------------------------------------------------
EMS = expected mean square
V = variance
```

TABLE 26

VARIABLE OR1

GERMINATION RATE OF THE 110 TREATED SEEDS ORIGINALLY PLANTED

	#	SOURCE	DF	MS	F	SIG	% VAR
1	+		3	14.67927778	1.23		1.4
2	-		4	14.48541667	.45		0
3	rep		2	87.51616667	2.14		11.3
4	+ x -		12	16.04441667	1.43		6.9
5	+ x rep		6	4.93994445	.44		0
6	- x rep		8	41.27616667	3.67	*	32.2
7	error		24	11.23216667			48.2

VARIABLE OR2

PROPORTION OF PLANTS SMUTTED

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	40.21822222	.69		0
2	-	4	583.17650000	5.00	*	33.3
3	rep	2	637.79850000	14.54	*	25.3
4	+ x -	12	89.56350000	2.91	*	15.9
5	+ x rep	6	12.83405556	.42		0
6	- x rep	8	33.15787500	1.08		.5
7	error	24	30.74287500			25.0

VARIABLE OR3

NUMBER OF HEADS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	31	.51		0
2	-	4	746.64166667	.93		0
3	rep	2	2527.01666667	2.55		14.2
4	+ x -	12	510.70833333	1.16		3.8
5	+ x rep	6	402.75000000	.92		0
6	- x rep	8	760.57916667	1.73		12.7
7	error	24	439.39583333			69.3

TABLE 26
(continued)

VARIABLE OR4

PROPORTION OF HEADS SMUTTED

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	35.94088889	.71		0
2	-	4	638.83041667	4.96	*	34.7
3	rep	2	552.26616667	8.91	*	20.2
4	+ x -	12	89.41630556	2.18	*	12.4
5	+ x rep	6	18.92438889	.46		0
6	- x rep	8	47.64179167	1.16		1.3
7	error	24	40.94834722			31.4

VARIABLE OR5

NUMBER OF HEADS FROM DISEASED PLANTS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	104.24444444	1.35		2.3
2	-	4	269.93333333	2.59		13.5
3	rep	2	778.95000000	9.03	*	29.7
4	+ x -	12	79.74444444	1.26		4.4
5	+ x rep	6	44.32777778	.70		0
6	- x rep	8	48.97083333	.78		0
7	error	24	63.18194444			50.1

VARIABLE OR6

AVERAGE NUMBER OF HEADS PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.01172222	.50		0
2	-	4	.30141667	.95		0
3	rep	2	1.06016667	2.64		15.2
4	+ x -	12	.20297222	1.17		3.8
5	+ x rep	6	.16972222	.97		0
6	- x rep	8	.29704167	1.71		12.2
7	error	24	.17409722			68.9

TABLE 26
(continued)

VARIABLE OR7

AVERAGE NUMBER OF DISEASED HEADS PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.02135111	1.13		.8
2	-	4	.08477667	2.74	*	15.9
3	rep	2	.22232000	8.28	*	31.3
4	+ x -	12	.02030111	1.30		4.7
5	+ x rep	6	.01247111	.80		0
6	- x rep	8	.01626167	1.04		.5
7	error	24	.01557944			46.7

VARIABLE OR8

AVERAGE NUMBER OF HEALTHY HEADS PER PLANT

#.	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.03127778	.60		0
2	-	4	.42225000	1.12		2.2
3	rep	2	.47016667	1.38		3.9
4	+ x -	12	.18391667	1.22		4.9
5	+ x rep	6	.12127778	.80		0
6	- x rep	8	.33037500	2.18		20.3
7	error	24	.15120833			68.6

VARIABLE OR9

AVERAGE NUMBER OF HEADS PER DISEASED PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.10977778	.48		0
2	-	4	3.36016667	3.29	*	26.6
3	rep	2	.89716667	3.38	*	5.5
4	+ x -	12	.89505556	2.33	*	20.8
5	+ x rep	6	.13494445	.35		0
6	- x rep	8	.24404167	.63		0
7	error	24	.38459722			47.1

TABLE 26
(continued)

VARIABLE OR10

AVERAGE NUMBER OF DISEASED HEADS PER DISEASED PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.12994444	.58		0
2	-	4	2.62350000	4.58	*	35.3
3	rep	2	.22316667	1.82		2.0
4	+ x -	12	.50550000	2.07		16.5
5	+ x rep	6	.13561111	.56		0
6	- x rep	8	.12087500	.50		0
7	error	24	.24387500			46.2

VARIABLE OR11

AVERAGE NUMBER OF HEALTHY HEADS PER DISEASED PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.04534444	1.17		1.3
2	-	4	.08850583	1.08		1.2
3	rep	2	.26132667	4.47	*	16.1
4	+ x -	12	.07346250	1.34		8.1
5	+ x rep	6	.01186445	.22		0
6	- x rep	8	.05890583	1.07		1.3
7	error	24	.05485750			71.9

VARIABLE OR12

SPORE WEIGHT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	1.70851133	1.34		2.6
2	-	4	4.43700396	2.44		14.6
3	rep	2	9.30956402	4.64	*	21.9
4	+ x -	12	1.06771579	.96		0
5	+ x rep	6	1.03879182	.93		0
6	- x rep	8	1.20947725	1.09		1.3
7	error	24	1.11266455			59.6

TABLE 26
(continued)

VARIABLE OR13

AVERAGE SPORE WEIGHT PER DISEASED PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.00796154	.75		0
2	-	4	.06535069	3.44	*	29.5
3	rep	2	.00643195	.98		0
4	+ x -	12	.01412462	1.64		12.3
5	+ x rep	6	.00806812	.93		0
6	- x rep	8	.00735857	.85		0
7	error	24	.00863637			58.2

VARIABLE OR14

AVERAGE SPORE WEIGHT PER DISEASED HEAD

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.00335809	.91		0
2	-	4	.03008313	5.77	*	50.8
3	rep	2	.00063911	.81		0
4	+ x -	12	.00407365	4.45	*	25.1
5	+ x rep	6	.00062684	.69		0
6	- x rep	8	.00130209	1.42		2.3
7	error	24	.00091452			21.8

VARIABLE OR15

AVERAGE SPORE GERMINATION RATE PER DISEASED HEAD

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	20.89422222	.18		0
2	-	4	2747.64150000	5.81	*	50.2
3	rep	2	.56466667	1.17		.1
4	+ x -	12	451.67172222	7.28	*	33.6
5	+ x rep	6	21.60155556	.35		0
6	- x rep	8	31.89987500	.51		0
7	error	24	62.03093056			16.1

TABLE 26
(continued)

VARIABLE OR16

AVERAGE NUMBER OF SEEDS PER DISEASED PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	63.26950000	1.19		1.5
2	-	4	119.51608333	1.36		4.4
3	rep	2	288.61316667	4.80	*	14.9
4	+ x -	12	87.45963889	1.27		6.6
5	+ x rep	6	23.33983333	.34		0
6	- x rep	8	51.19045833	.74		0
7	error	24	68.80684722			72.6

VARIABLE OR17

AVERAGE NUMBER OF SEEDS PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	120.12111111	.71		0
2	-	4	860.80600000	1.11		2.2
3	rep	2	462.23450000	.87		0
4	+ x -	12	402.17000000	1.19		4.8
5	+ x rep	6	239.98561111	.71		0
6	- x rep	8	673.44137500	2.00		18.6
7	error	24	336.90804167			74.4

VARIABLE OWp [PATHOGEN]

PATHOGEN FITNESS
(CALCULATED FROM P SUBSET OF VARIABLES)

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.04023427	1.06		.5
2	-	4	.09557794	1.80		6.4
3	rep	2	.16386699	2.72		7.4
4	+ x -	12	.06928482	.73		0
5	+ x rep	6	.05851552	.61		0
6	- x rep	8	.03668752	.38		0
7	error	24	.09545937			85.8

TABLE 26
(continued)

VARIABLE OWc [PATHOGEN]

PATHOGEN FITNESS
(CALCULATED FROM C SUBSET OF VARIABLES)

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.67255677	1.76		5.7
2	-	4	1.08300876	2.15		12.6
3	rep	2	2.10390383	3.97	*	18.3
4	+ x -	12	.29429220	.98		0
5	+ x rep	6	.25714967	.86		0
6	- x rep	8	.34795047	1.16		2.5
7	error	24	.29931392			61.0

VARIABLE OW [PATHOGEN]

TOTAL PATHOGEN FITNESS
(Wp [PATHOGEN] + Wc [PATHOGEN])

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.96751576	1.54		4.5
2	-	4	1.56063792	2.20		12.3
3	rep	2	3.10532772	3.58	*	17.1
4	+ x -	12	.44170166	.89		0
5	+ x rep	6	.51042054	1.02		.3
6	- x rep	8	.49659848	1.00		0
7	error	24	.49896609			65.8

VARIABLE OWp [HOST]

HOST FITNESS
(CALCULATED FROM P SUBSET OF VARIABLES)

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	8569.43529033	1.74		5.0
2	-	4	10383.70323932	1.73		7.0
3	rep	2	20464.46626115	4.82	*	12.7
4	+ x -	12	6317.10778844	1.00		.1
5	+ x rep	6	2233.41231140	.35		0
6	- x rep	8	3316.96389569	.53		0
7	error	24	6294.72001496			75.2

TABLE 26
(continued)

VARIABLE OWh [HOST]

HOST FITNESS
(CALCULATED FROM H SUBSET OF VARIABLES)

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	425748.21119924	.82		0
2	-	4	1943631.35600586	1.11		2.1
3	rep	2	845109.14484295	.76		0
4	+ x -	12	863660.96068215	1.21		5.0
5	+ x rep	6	525031.87772750	.74		0
6	- x rep	8	1538370.42225049	2.15		20.8
7	error	24	714149.44967887			72.0

VARIABLE OW [HOST]

TOTAL HOST FITNESS
(Wp [HOST] + Wh [HOST])

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	325583.74482073	.75		0
2	-	4	1946995.42645697	1.08		1.7
3	rep	2	1062041.41176370	.87		0
4	+ x -	12	925795.61069632	1.20		4.9
5	+ x rep	6	534066.65325863	.69		0
6	- x rep	8	1586665.13315334	2.05		19.5
7	error	24	772457.77180752			74.0

TABLE 27.   Analysis of variance of H variables on Odessa (O).
      Sources of variability include three main effects
      components; plus sporidia (+), minus sporidia (-), and
      replicates (rep); as well as all possible second order
      interactions; sporidial interactions (+x-), and two
      types of sporidia replicate interactions (+xrep, and
      -xrep).   The third order interaction component
      (+x-xrep) was redefined as the error component.
      Degrees of freedom, mean squares, F and pseudo-F values
      were calculated.   It was necessary to calculate
      pseudo-F values for the three main effects components
      because of the absence of suitable denominator mean
      squares.   Components with statistically significant F
      values (alpha=.05) have an asterisk in the "SIG" column.
      The relative contribution of each component to total
      variability (% VAR) was determined using the following
      expected mean squares table:

```
EMS+      = Verror + 3V+x-    + 5V+xrep + 15V+
EMS-      = Verror + 3V+x-    + 4V-xrep + 12V-
EMSrep    = Verror + 5V+xrep + 4V-xrep + 20Vrep
EMS+x-    = Verror + 3V+x-
EMS+xrep  = Verror + 5V+xrep
EMS-xrep  = Verror + 4V-xrep
EMSerror  = Verror
-----------------------------------------------------------
EMS = expected mean square
V = variance
```

TABLE 27

VARIABLE OH1

NUMBER OF HEALTHY PLANTS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	22.95000000	1.30		1.5
2	-	4	123.89166667	3.77	*	23.3
3	rep	2	231.11666667	10.78	*	30.9
4	+ x -	12	20.26944444	1.66		7.5
5	+ x rep	6	6.71666667	.55		0
6	- x rep	8	15.86666667	1.30		2.5
7	error	24	12.24444444			34.3

VARIABLE OH2

NUMBER OF HEADS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	143.37777778	.67		0
2	-	4	1124.79166667	1.22		4.3
3	rep	2	929.21666667	1.17		1.8
4	+ x -	12	416.00277778	1.25		5.5
5	+ x rep	6	293.66111111	.88		0
6	- x rep	8	782.59166667	2.35		22.3
7	error	24	333.20277778			66.1

VARIABLE OH3

AVERAGE NUMBER OF HEADS PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.01527778	.49		0
2	-	4	.35475000	.88		0
3	rep	2	.88066667	1.96		9.4
4	+ x -	12	.23875000	1.27		6.2
5	+ x rep	6	.17177778	.92		0
6	- x rep	8	.37400000	2.00		16.8
7	error	24	.18733333			67.6

TABLE 27
(continued)

VARIABLE OH4

AVERAGE NUMBER OF SEEDS PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	46.83133333	.52		0
2	-	4	869.62900000	.98		0
3	rep	2	945.34016667	1.12		1.4
4	+ x -	12	490.78466667	1.19		4.9
5	+ x rep	6	396.39016667	.97		0
6	- x rep	8	809.94162500	1.97		18.3
7	error	24	410.71995833			75.4

VARIABLE OH5

AVERAGE NUMBER OF SEEDS PER HEAD

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	12.30283333	.65		0
2	-	4	43.42691667	1.67		10.4
3	rep	2	13.57800000	.86		0
4	+ x -	12	20.42325000	1.67		15.3
5	+ x rep	6	17.10866667	1.40		5.5
6	- x rep	8	12.83150000	1.05		.9
7	error	24	12.20905556			68.0

VARIABLE OH6

THOUSAND SEED WEIGHT,
SEEDS RANDOMLY SELECTED FROM ALL HEALTHY PLANTS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	1.38994444	.54		0
2	-	4	17.76858333	1.24		1.9
3	rep	2	246.83750000	18.00	*	54.2
4	+ x -	12	12.37869444	1.49		6.1
5	+ x rep	6	5.58461111	.67		0
6	- x rep	8	8.58833333	1.03		.3
7	error	24	8.31127778			37.4

TABLE 27
(continued)

VARIABLE OH7

AVERAGE SEED WEIGHT PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.08928833	.48		0
2	-	4	1.75870841	1.01		.1
3	rep	2	3.69165893	1.85		8.9
4	+ x -	12	.97864675	1.24		5.5
5	+ x rep	6	.86281089	1.10		1.3
6	- x rep	8	1.55269703	1.97		16.5
7	error	24	.78753073			67.8

VARIABLE OH8

AVERAGE SEED WEIGHT PER HEAD

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.02511772	.98		0
2	-	4	.13610154	1.38		5.5
3	rep	2	.20194638	2.20		8.7
4	+ x -	12	.05934858	.89		0
5	+ x rep	6	.03444043	.52		0
6	- x rep	8	.08783123	1.32		6.3
7	error	24	.06665947			79.5

VARIABLE OH9

SEED GERMINATION RATE (FOR SEEDS FROM H6)

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	2.48022222	.86		0
2	-	4	27.47941667	1.58		6.7
3	rep	2	6.52866667	.85		0
4	+ x -	12	12.74897222	.62		0
5	+ x rep	6	14.31688889	.69		0
6	- x rep	8	17.69116667	.86		0
7	error	24	20.67105556			93.3

TABLE 27
(continued)

VARIABLE OH10
_____

NUMBER OF SEEDS
_____

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	405144.08777807	.77		0
2	-	4	2148896.21233355	1.14		2.8
3	rep	2	897264.65150047	.75		0
4	+ x -	12	930321.95555548	1.21		5.1
5	+ x rep	6	584074.40594429	.76		0
6	- x rep	8	1628950.16295821	2.12		20.2
7	error	24	766701.29018058			71.9

TABLE 28.   Analysis of variance of C variables on Odessa (O).
Sources of variability include three main effects
components; plus sporidia (+), minus sporidia (-), and
replicates (rep); as well as all possible second order
interactions; sporidial interactions (+x-), and two
types of sporidia replicate interactions (+xrep, and
-xrep).   The third order interaction component
(+x-xrep) was redefined as the error component.
Degrees of freedom, mean squares, F and pseudo-F values
were calculated.   It was necessary to calculate
pseudo-F values for the three main effects components
because of the absence of suitable denominator mean
squares.   Components with statistically significant F
values (alpha=.05) have an asterisk in the "SIG" column.
The relative contribution of each component to total
variability (% VAR) was determined using the following
expected mean squares table:

```
EMS+       = Verror + 3V+x-    + 5V+xrep + 15V+
EMS-       = Verror + 3V+x-    + 4V-xrep + 12V-
EMSrep     = Verror + 5V+xrep + 4V-xrep + 20Vrep
EMS+x-     = Verror + 3V+x-
EMS+xrep   = Verror + 5V+xrep
EMS-xrep   = Verror + 4V-xrep
EMSerror   = Verror
----------------------------------------------------------
EMS = expected mean square
V = variance
```

TABLE 28

VARIABLE OC1

NUMBER OF COMPLETELY DISEASED PLANTS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	10	.88		0
2	-	4	107.65000000	3.42	*	24.0
3	rep	2	165.26666667	9.04	*	27.1
4	+ x -	12	18.47222223	1.96		10.5
5	+ x rep	6	3.53333333	.37		0
6	- x rep	8	15.78750000	1.67		5.5
7	error	24	9.44305556			32.9

VARIABLE OC2

NUMBER OF HEADS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	29.80000000	1.12		.7
2	-	4	178.97500000	2.96	*	18.5
3	rep	2	396.86666667	8.91	*	30.7
4	+ x -	12	35.23055556	1.40		5.5
5	+ x rep	6	13.66666667	.54		0
6	- x rep	8	33.70000000	1.34		3.5
7	error	24	25.13888889			41.2

VARIABLE OC3

AVERAGE NUMBER OF HEADS PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.18177778	.58		0
2	-	4	2.44058333	4.24	*	35.8
3	rep	2	.18050000	1.46		1.2
4	+ x -	12	.50247222	2.64	*	22.2
5	+ x rep	6	.13627778	.72		0
6	- x rep	8	.11758333	.62		0
7	error	24	.19030556			40.7

TABLE 28
(continued)

VARIABLE OC4

SPORE WEIGHT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	1.24118798	1.31		2.4
2	-	4	3.30745789	3.45	*	19.1
3	rep	2	4.18379995	2.31		11.2
4	+ x -	12	.25063370	.38		0
5	+ x rep	6	1.19797666	1.84		8.9
6	- x rep	8	.89848249	1.38		5.0
7	error	24	.65186424			53.3

VARIABLE OC5

AVERAGE SPORE WEIGHT PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.01059327	.89		0
2	-	4	.05891309	3.46	*	31.9
3	rep	2	.00558766	.85		0
4	+ x -	12	.01185602	2.09		17.2
5	+ x rep	6	.00638486	1.13		1.2
6	- x rep	8	.00682236	1.20		2.4
7	error	24	.00567392			47.3

VARIABLE OC6

AVERAGE SPORE WEIGHT PER HEAD

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.00348274	.91		0
2	-	4	.02932797	5.10	*	48.3
3	rep	2	.00119590	.91		0
4	+ x -	12	.00421105	5.28	*	27.2
5	+ x rep	6	.00051235	.64		0
6	- x rep	8	.00169060	2.12		5.3
7	error	24	.00079805			19.1

TABLE 28
(continued)

VARIABLE OC7

AVERAGE SPORE GERMINATION RATE PER HEAD

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	18.51172222	.18		0
2	-	4	2729.20041667	5.82	*	49.9
3	rep	2	1.15800000	1.23		.2
4	+ x -	12	448.00852778	6.88	*	33.0
5	+ x rep	6	21.69088889	.33		0
6	- x rep	8	32.18716667	.49		0
7	error	24	65.14394444			16.9

TABLE 29.   Analysis of variance of P variables on Odessa (O).
     Sources of variability include three main effects
     components; plus sporidia (+), minus sporidia (-), and
     replicates (rep); as well as all possible second order
     interactions; sporidial interactions (+x-), and two
     types of sporidia replicate interactions (+xrep, and
     -xrep).   The third order interaction component
     (+x-xrep) was redefined as the error component.
     Degrees of freedom, mean squares, F and pseudo-F values
     were calculated.   It was necessary to calculate
     pseudo-F values for the three main effects components
     because of the absence of suitable denominator mean
     squares.   Components with statistically significant F
     values (alpha=.05) have an asterisk in the "SIG" column.
     The relative contribution of each component to total
     variability (% VAR) was determined using the following
     expected mean squares table:

```
EMS+       = Verror + 3V+x-    + 5V+xrep + 15V+
EMS-       = Verror + 3V+x-    + 4V-xrep + 12V-
EMSrep     = Verror + 5V+xrep + 4V-xrep + 20Vrep
EMS+x-     = Verror + 3V+x-
EMS+xrep   = Verror + 5V+xrep
EMS-xrep   = Verror + 4V-xrep
EMSerror   = Verror
------------------------------------------------------------
EMS = expected mean square
V = variance
```

TABLE 29

VARIABLE OP1

NUMBER OF DISEASED PLANTS WITH SEEDS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	2.72777778	1.58		4.8
2	-	4	1.94166667	1.21		2.4
3	rep	2	5.81666667	3.91	*	12.5
4	+ x -	12	1.96388889	1.14		3.6
5	+ x rep	6	.86111111	.50		0
6	- x rep	8	1.06666667	.62		0
7	error	24	1.72222222			76.7

VARIABLE OP2

NUMBER OF HEADS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	25.64444444	1.34		2.6
2	-	4	33.56666667	1.52		5.0
3	rep	2	82.91666667	4.41	*	12.1
4	+ x -	12	27.97777778	.98		0
5	+ x rep	6	12.49444445	.44		0
6	- x rep	8	12.79166667	.45		0
7	error	24	28.53611111			80.3

VARIABLE OP3

NUMBER OF DISEASED HEADS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	4.64444444	.99		0
2	-	4	8.68333333	1.54		5.1
3	rep	2	20.06666667	3.19		9.9
4	+ x -	12	7.67222222	.91		0
5	+ x rep	6	5.51111111	.66		0
6	- x rep	8	3.42083333	.41		0
7	error	24	8.39305556			85.1

TABLE 29
(continued)

VARIABLE OP4

NUMBER OF HEALTHY HEADS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	8.73333333	1.74		5.1
2	-	4	9.44166667	1.50		5.2
3	rep	2	21.65000000	4.60	*	12.9
4	+ x -	12	6.66388889	1.02		.5
5	+ x rep	6	2.11666667	.32		0
6	- x rep	8	4.00416667	.61		0
7	error	24	6.52638889			76.3

VARIABLE OP5

AVERAGE NUMBER OF HEADS PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	2.48755556	1.20		1.4
2	-	4	3.75308333	1.28		2.9
3	rep	2	14.88200000	9.68	*	18.9
4	+ x -	12	3.93186111	1.36		8.2
5	+ x rep	6	.56422222	.20		0
6	- x rep	8	1.27220833	.44		0
7	error	24	2.89331944			68.6

VARIABLE OP6

AVERAGE NUMBER OF DISEASED HEADS PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.45177778	.97		0
2	-	4	.98500000	1.40		3.9
3	rep	2	3.48750000	5.73	*	16.2
4	+ x -	12	.95066667	1.12		3.0
5	+ x rep	6	.39194445	.46		0
6	- x rep	8	.36562500	.43		0
7	error	24	.85229167			76.9

TABLE 29
(continued)

VARIABLE OP7

AVERAGE NUMBER OF HEALTHY HEADS PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.84565500	1.15		1.2
2	-	4	1.11180667	1.07		1.0
3	rep	2	4.05798167	5.27	*	17.8
4	+ x -	12	1.11138000	1.47		10.9
5	+ x rep	6	.28448833	.38		0
6	- x rep	8	.62931292	.83		0
7	error	24	.75404458			69.0

VARIABLE OP8

SPORE WEIGHT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.06504438	.94		0
2	-	4	.20282789	1.61		5.1
3	rep	2	.44130515	3.19	*	8.6
4	+ x -	12	.18827850	.81		0
5	+ x rep	6	.12888513	.56		0
6	- x rep	8	.08198650	.35		0
7	error	24	.23173982			86.3

VARIABLE OP9

AVERAGE SPORE WEIGHT PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.00742927	1.09		.6
2	-	4	.02785290	1.57		4.9
3	rep	2	.07084056	3.89	*	10.6
4	+ x -	12	.02220151	.76		0
5	+ x rep	6	.01149667	.39		0
6	- x rep	8	.01424267	.49		0
7	error	24	.02934757			83.8

TABLE 29
(continued)

VARIABLE OP10

AVERAGE SPORE WEIGHT PER HEAD

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.00652572	2.68	*	6.3
2	-	4	.00571639	2.50	*	7.0
3	rep	2	.01278546	6.61	*	9.3
4	+ x -	12	.00355481	.51		0
5	+ x rep	6	.00147161	.21		0
6	- x rep	8	.00151332	.22		0
7	error	24	.00695823			77.4

VARIABLE OP11

AVERAGE SPORE GERMINATION RATE PER HEAD

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	911.31755556	2.22		7.1
2	-	4	477.21150000	1.27		2.4
3	rep	2	1923.06050000	9.18	*	14.5
4	+ x -	12	606.62005556	1.07		1.8
5	+ x rep	6	59.37938889	.11		0
6	- x rep	8	211.65987500	.37		0
7	error	24	564.97459722			74.1

VARIABLE OP12

NUMBER OF SEEDS

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	10549.91111111	1.74		5.0
2	-	4	13051.64166667	1.74		7.1
3	rep	2	25258.61666667	4.59	*	12.4
4	+ x -	12	7707.84166667	.97		0
5	+ x rep	6	2897.86111111	.37		0
6	- x rep	8	4320.49166667	.55		0
7	error	24	7908.87500000			75.5

TABLE 29
(continued)

VARIABLE OP13

AVERAGE NUMBER OF SEEDS PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	1226.18533333	1.40		3.0
2	-	4	1798.93358333	1.64		6.4
3	rep	2	4473.22066667	6.13	*	16.2
4	+ x -	12	1187.41936111	1.22		5.0
5	+ x rep	6	388.13200000	.40		0
6	- x rep	8	501.40045833	.51		0
7	error	24	976.97123611			69.4

VARIABLE OP14

AVERAGE NUMBER OF SEEDS PER HEALTHY HEAD

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	366.68505556	1.39		3.1
2	-	4	400.38733333	1.44		4.5
3	rep	2	1038.61550000	7.03	*	14.7
4	+ x -	12	363.16644444	1.39		8.9
5	+ x rep	6	89.48505556	.34		0
6	- x rep	8	95.49570833	.36		0
7	error	24	261.64331944			68.9

VARIABLE OP15

SEED WEIGHT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	17.93020739	1.62		4.6
2	-	4	20.65359102	1.68		6.6
3	rep	2	41.40557082	4.69	*	12.4
4	+ x -	12	13.67217384	1.06		1.5
5	+ x rep	6	5.33826566	.41		0
6	- x rep	8	6.24042582	.48		0
7	error	24	12.87820711			74.9

TABLE 29
(continued)

VARIABLE OP16

AVERAGE SEED WEIGHT PER PLANT

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	1.81415413	1.27		2.1
2	-	4	2.73604594	1.49		5.2
3	rep	2	7.52095115	6.74	*	17.2
4	+ x -	12	2.05253858	1.37		8.3
5	+ x rep	6	.54956405	.37		0
6	- x rep	8	.78920615	.53		0
7	error	24	1.49650386			67.1

VARIABLE OP17

AVERAGE SEED WEIGHT PER HEALTHY HEAD

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	.54884986	1.43		3.3
2	-	4	.55839826	1.47		4.5
3	rep	2	1.83318920	8.47	*	17.7
4	+ x -	12	.50164149	1.39		8.5
5	+ x rep	6	.13488174	.37		0
6	- x rep	8	.12421348	.34		0
7	error	24	.36170730			66.0

VARIABLE OP18

AVERAGE SEED GERMINATION RATE PER HEALTHY HEAD

#	SOURCE	DF	MS	F	SIG	% VAR
1	+	3	1282.60727778	1.35		2.5
2	-	4	673.45691667	.84		0
3	rep	2	4845.36866667	13.53	*	17.4
4	+ x -	12	1674.48936111	1.60		13.3
5	+ x rep	6	56.53444445	.05		0
6	- x rep	8	378.83116667	.36		0
7	error	24	1046.92527778			66.7

TABLE 30.   A comparison of the pattern of significant
        components of variability on Trebi (T) and Odessa (O).
        Variance components 1 to 6 (1=+ sporidia, 2=- sporidia,
        3=replicates, 4=+x- interactions, 5=+xreplicate
        interactions, 6=-xreplicate interactions) are listed.
        When statistically significant differences among
        constituent members of a component were detected, an
        asterix was placed in the corresponding column.   The
        similar variables measured on Trebi and on Odessa are
        shown side by side for ease of comparison.

        [P] = [PATHOGEN], [H] = [HOST]

TABLE 30

	VARIANCE COMPONENT							VARIANCE COMPONENT					
TR	1	2	3	4	5	6	OR	1	2	3	4	5	6
1			*				1						*
2		*					2		*	*	*		
3			*				3						
4		*					4		*	*	*		
5			*				5			*			
6			*				6						
7		*	*				7		*	*			
8							8						
9							9		*	*	*		
10		*		*			10		*				
11							11			*			
12		*	*				12			*			
13		*					13		*				
14		*					14		*		*		
15				*			15		*		*		
16							16			*			
17			*				17						
Wp[P]							Wp[P]						
Wc[P]		*					Wc[P]			*			
W[P]		*	*				W[P]			*			
Wp[H]							Wp[H]			*			
Wh[H]			*				Wh[H]						
W[H]			*				W[H]						

TABLE 30
(continued)

VARIANCE COMPONENT

TH	1	2	3	4	5	6
1		*	*	*		
2						
3						
4			*			
5			*	*		
6			*			
7			*			
8			*			
9						
10			*			

VARIANCE COMPONENT

OH	1	2	3	4	5	6
1		*	*			
2						
3						
4						
5						
6			*			
7						
8						
9						
10						

VARIANCE COMPONENT

TC	1	2	3	4	5	6
1		*	*	*		
2		*	*	*		
3						
4	*	*	*			
5		*				
6		*				
7				*		

VARIANCE COMPONENT

OC	1	2	3	4	5	6
1		*	*			
2		*	*			
3		*		*		
4		*				
5		*				
6		*		*		
7		*		*		

TABLE 30
(continued)

	VARIANCE COMPONENT								VARIANCE COMPONENT					
TP	1	2	3	4	5	6	OP	1	2	3	4	5	6
1			*				1			*			
2							2			*			
3							3						
4							4			*			
5		*					5			*			
6		*					6			*			
7							7			*			
8							8			*			
9		*					9			*			
10		*					10	*	*	*			
11		*					11			*			
12							12			*			
13							13			*			
14		*					14			*			
15							15			*			
16							16			*			
17		*					17			*			
18		*					18			*			

TABLE 31.   Frequencies of combinations of variance components
        contributing significantly to total variance.
        Frequencies of all possible combinations of variance
        components 1 to 4 are shown for each individual subset.
        The total number of types of combinations for each
        subset is given as well as the total number of types of
        combinations for each variety.

TABLE 31

COMPONENT* COMBINATIONS	TREBI					ODESSA				
	VARIABLE SUBSET					VARIABLE SUBSET				
	R	H	C	P	TOTAL	R	H	C	P	TOTAL
1	–	–	–	–	–	–	–	–	–	–
2	5	–	2	8	15	2	–	2	–	4
3	7	5	–	1	13	7	1	–	16	24
4	1	–	1	–	2	–	–	–	–	–
12	–	–	–	–	–	–	–	–	–	–
13	–	–	–	–	–	–	–	–	–	–
14	–	–	–	–	–	–	–	–	–	–
23	3	–	–	–	3	1	1	2	–	4
24	1	–	–	–	1	2	–	3	–	5
34	–	1	–	–	1	–	–	–	–	–
123	–	–	1	–	1	–	–	–	1	1
134	–	–	–	–	–	–	–	–	–	–
234	–	1	2	–	3	3	–	–	–	3
1234	–	–	–	–	–	–	–	–	–	–
TOTAL	17	7	6	9	39	15	2	7	17	41

* 1 = + sporidia; 2 = – sporidia; 3 = replicates;
  4 = + x – sporidial interaction; 12 = + sporidia
    and – sporidia
234 = – sporidia, replicates and + x – sporidial
    interactions; etc.

TABLE 32.   Stepwise regression results of the COMPLETE models
        for the dependent variables W [PATHOGEN] (pathogen
        fitness) and W [HOST] (host fitness).   Independent
        variables employed in these models are shown above the R
        SQUARE value.   The TERMS column contains the Y
        intercept and any independent variable with a
        statistically significant F value.   Intercept and term
        coefficients are in the B column.   Remaining columns
        hold the standard error (SE), sum of squares (SS), F,
        and probability of significance values (PROB>F).

TABLE 32

COMPLETE: PATHOGEN FITNESS (W [PATHOGEN]) ON TREBI

R1   R2   R3   R4   R5   R6   R7   R8   R9   R10
R11 R12 R13 R14 R15 R16 R17
Wp [HOST] Wh [HOST] W [HOST]
H1   H2   H3   H4   H5   H6   H7   H8   H9   H10
C1   C2   C3   C4   C5   C6   C7
P1   P2   P3   P4   P5   P6   P7   P8   P9   P10
P11 P12 P13 P14 P15 P16 P17 P18

R SQUARE = 0.99409972

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-0.02529980				
R12	0.40560957	0.03103345	0.20529614	170.83	0.0001
C1	0.02526359	0.00688430	0.01618432	13.47	0.0021
P10	0.96666443	0.28802940	0.01353637	11.26	0.0040

COMPLETE: PATHOGEN FITNESS (W [PATHOGEN]) ON ODESSA

R1   R2   R3   R4   R5   R6   R7   R8   R9   R10
R11 R12 R13 R14 R15 R16 R17
Wp [HOST] Wh [HOST] W [HOST]
H1   H2   H3   H4   H5   H6   H7   H8   H9T H10
C1   C2   C3   C4   C5   C6   C7
P1   P2   P3   P4   P5   P6   P7   P8   P9   P10
P11 P12 P13 P14 P15 P16 P17 P18

R SQUARE = 0.98374694

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	0.06577155				
R4	-0.01076009	0.00362184	0.04317179	8.83	0.0090
R12	0.80254383	0.05129552	1.19730763	244.78	0.0001
R13	-0.87927227	0.34046053	0.03262424	6.67	0.0200

TABLE 32
(continued)

COMPLETE: HOST FITNESS (W [HOST]) ON TREBI

R1   R2   R3   R4   R5   R6   R7   R8   R9   R10
R11 R12 R13 R14 R15 R16 R17
Wp [PATHOGEN] Wc [PATHOGEN] W [PATHOGEN]
H1   H2   H3   H4   H5   H6   H7   H8   H9   H10
C1   C2   C3   C4   C5   C6   C7
P1   P2   P3   P4   P5   P6   P7   P8   P9   P10
P11 P12 P13 P14 P15 P16 P17 P18

R SQUARE = 0.99729422

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	19.983571				
R17	46.195638	0.567151	12461355.625647	6634.43	0.0001

COMPLETE: HOST FITNESS (W [HOST]) ON ODESSA

R1   R2   R3   R4   R5   R6   R7   R8   R9   R10
R11 R12 R13 R14 R15 R16 R17
Wp [PATHOGEN] Wc [PATHOGEN] W [PATHOGEN]
H1   H2   H3   H4   H5   H6   H7   H8   H9   H10
C1   C2   C3   C4   C5   C6   C7
P1   P2   P3   P4   P5   P6   P7   P8   P9   P10
P11 P12 P13 P14 P15 P16 P17 P18

R SQUARE = 0.99648852

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-4.792663				
R17	47.905550	0.670283	6601497.392234	5108.05	0.0001

TABLE 33. Stepwise regression results of the COMPLETE models
for the dependent variables W [PATHOGEN]G (pathogen
fitness) and W [HOST]G (host fitness). Independent
variables employed in these models were those with
statistically significant genetic component(s) and are
shown above the R SQUARE value. The TERMS column
contains the Y intercept and any independent variable
with a statistically significant F value. Intercept
and term coefficients are in the B column. Remaining
columns hold the standard error (SE), sum of squares
(SS), F, and probability of significance values
(PROB>F).

TABLE 33

COMPLETE: PATHOGEN FITNESS (W [PATHOGEN]G) ON TREBI
```
         R2  R4  R7  R10 R12 R13 R14 R15
         H1
         C1  C2  C4  C5  C6  C7
         P5  P6  P9  P10 P11 P14 P17 P18
```

R SQUARE = 0.99409972

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-0.02529980				
R12	0.40560957	0.03103345	0.20529614	170.83	0.0001
C1	0.02526359	0.00688430	0.01618432	13.47	0.0021
P10	0.96666443	0.28802940	0.01353637	11.26	0.0040

COMPLETE: PATHOGEN FITNESS (W [PATHOGEN]G) ON ODESSA
```
         R1  R2  R4  R7  R9  R10 R13 R14 R15
         H1
         C1  C2  C3  C4  C5  C6  C7
         P10
```

R SQUARE = 0.98093828

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-0.01198516				
R7	4.63272255	0.60986663	0331021991	57.70	0.0001
C2	-0.12873443	0.01447878	0.45350218	79.05	0.0001
C4	0.85865437	0.07441344	0.76381553	133.15	0.0001

TABLE 33
(continue)

COMPLETE: HOST FITNESS (W [HOST]G) ON TREBI

       R2   R4   R7   R10 R12 R13 R14 R15
       Wc [PATHOGEN] W [PATHOGEN]
       H1
       C1   C2   C4   C5   C6   C7
       P5   P6   P9   P10 P11 P14 P17 P18

              R SQUARE = 0.25327987

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	3358.812774				
Wc [P]	-1150.670752	465.686247	3164773.719389	6.11	0.0237

COMPLETE: HOST FITNESS (W [HOST]G) ON ODESSA

       R1   R2   R4   R7   R9   R10 R13 R14 R15
       H1
       C1   C2   C3   C4   C5   C6   C7
       P10

              R SQUARE = 0.67439628

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	1962.578000				
R2	256.156791	67.034552	1968583.549921	14.60	0.0015
R4	-178.492415	78.531384	696454.193075	5.17	0.0372
C1	-243.975454	95.279342	883963.425815	6.56	0.0209

TABLE 34.   Stepwise regression results of the TRADITIONAL
            models for the dependent variables W [PATHOGEN]
            (pathogen fitness) and W [HOST] (host fitness).   Only 1
            independent variable was employed (R2) and is shown
            above the R SQUARE value.   The TERMS column contains
            the Y intercept and the independent variable, if it had
            a statistically significant F value.   Intercept and
            term coefficients are in the B column.   Remaining
            columns hold the standard error (SE), sum of squares
            (SS), F, and probability of significance values
            (PROB>F).

TABLE 34

TRADITIONAL: PATHOGEN FITNESS (W [PATHOGEN]) ON TREBI
```
------------------------------------------------------------
                              R2
------------------------------------------------------------
               R SQUARE = 0.83622239
------------------------------------------------------------
   TERMS        B           SE          SS        F    PROB>F
---------  ----------  ----------  ----------  -----  ------
INTERCEPT  -0.31431579
R2          0.04787175  0.00499355  2.72516849  91.91  0.0001
```

TRADITIONAL: PATHOGEN FITNESS (W [PATHOGEN]) ON ODESSA
```
------------------------------------------------------------
                              R2
------------------------------------------------------------
               R SQUARE = 0.57033495
------------------------------------------------------------
   TERMS        B           SE          SS        F    PROB>F
---------  ----------  ----------  ----------  -----  ------
INTERCEPT  -0.27599497
R2          0.04832365  0.00988607  2.74626142  23.89  0.0001
```

TABLE 34
(continued)

TRADITIONAL: HOST FITNESS (W [HOST]) ON TREBI
```
------------------------------------------------------------
                              R2
------------------------------------------------------------
                R SQUARE = 0.15130233
------------------------------------------------------------
   TERMS         B          SE           SS          F   PROB>F
---------  ------------  ---------  --------------  ----  ------
INTERCEPT  3627.452314
R2          -39.872768  22.258394  1890547.575347  3.21  0.0901
```


TRADITIONAL: HOST FITNESS (W [HOST]) ON ODESSA
```
------------------------------------------------------------
                              R2
------------------------------------------------------------
                R SQUARE = 0.13125757
------------------------------------------------------------
   TERMS         B          SE           SS          F   PROB>F
---------  ------------  ---------  --------------  ----  ------
INTERCEPT  2738.408861
R2          -27.191680  16.488575  869549.905872   2.72  0.1165
```

TABLE 35.   Stepwise regression results of the TRADITIONAL
models for the dependent variables W [PATHOGEN]
(pathogen fitness) and W [HOST] (host fitness).   Only 1
independent variable was employed (R4) and is shown
above the R SQUARE value.   The TERMS column contains
the Y intercept and the independent variable, if it had
a statistically significant F value.   Intercept and
term coefficients are in the B column.   Remaining
columns hold the standard error (SE), sum of squares
(SS), F, and probability of significance values
(PROB>F).

TABLE 35

TRADITIONAL: PATHOGEN FITNESS (W [PATHOGEN]) ON TREBI
```
------------------------------------------------------------
                              R4
------------------------------------------------------------
                R SQUARE = 0.90528508
------------------------------------------------------------
    TERMS        B          SE          SS        F     PROB>F
------------------------------------------------------------
INTERCEPT  -0.24946328
R4          0.05162188 0.00393563 2.95023716 172.04 0.0001
```

TRADITIONAL: PATHOGEN FITNESS (W [PATHOGEN]) ON ODESSA
```
------------------------------------------------------------
                              R4
------------------------------------------------------------
                R SQUARE = 0.59992019
------------------------------------------------------------
    TERMS        B          SE          SS        F     PROB>F
------------------------------------------------------------
INTERCEPT  -0.24464349
R4          0.04816169 0.00927026 2.88871949 26.99 0.0001
```

TABLE 35
(continued)

TRADITIONAL: HOST FITNESS (W [HOST]) ON TREBI

R4					
R SQUARE = 0.23843417					
TERMS	B	SE	SS	F	PROB>F
INTERCEPT	3687.945767				
R4	-51.875292	21.852130	2979274.293383	5.64	0.0289

TRADITIONAL: HOST FITNESS (W [HOST]) ON ODESSA

R4					
R SQUARE = 0.21172367					
TERMS	B	SE	SS	F	PROB>F
INTERCEPT	2840.478148				
R4	-33.559760	15.262902	1402618.535973	4.83	0.0412

TABLE 36.   Stepwise regression results of the TRADITIONAL
models for the dependent variables W [PATHOGEN]
(pathogen fitness) and W [HOST] (host fitness).   Two
independent variables were employed (R2 and R4) and are
shown above the R SQUARE value.   The TERMS column
contains the Y intercept and any independent variable
with a statistically significant F value.   Intercept
and term coefficients are in the B column.   Remaining
columns hold the standard error (SE), sum of squares
(SS), F, and probability of significance values
(PROB>F).

TABLE 36

TRADITIONAL: PATHOGEN FITNESS (W [PATHOGEN]) ON TREBI

R2 R4
R SQUARE = 0.90528508

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-0.24946328				
R4	0.05162188	0.00393563	2.95023716	172.04	0.0001

TRADITIONAL: PATHOGEN FITNESS (W [PATHOGEN]) ON ODESSA

R2 R4
R SQUARE = 0.59992019

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-0.24464349				
R4	0.04816169	0.00927026	2.88871949	26.99	0.0001

TABLE 36
(continued)

TRADITIONAL: HOST FITNESS (W [HOST]) ON TREBI

R2 R4

R SQUARE = 0.23843417

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	3687.945767				
R4	-51.875292	21.852130	2979274.293383	5.64	0.0289

TRADITIONAL: HOST FITNESS (W [HOST]) ON ODESSA

R2 R4

R SQUARE = 0.54096301

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	2479.498203				
R2	268.888345	77.004393	2181131.598273	12.19	0.0028
R4	-291.482343	74.830004	2714200.228374	15.17	0.0012

TABLE 37.   Stepwise regression results of the PRACTICAL:
          MINIMAL COST models for the dependent variables W
          [PATHOGEN] (pathogen fitness) and W [HOST] (host
          fitness).   Independent variables employed in these
          models are shown above the R SQUARE value.   The TERMS
          column contains the Y intercept and any independent
          variable with a statistically significant F value.
          Intercept and term coefficients are in the B column.
          Remaining columns hold the standard error (SE), sum of
          squares (SS), F, and probability of significance values
          (PROB>F).

TABLE 37

PRACTICAL-MINIMAL COST: PATHOGEN FITNESS
(W [PATHOGEN]) ON TREBI

R1 R2 R3 R4 R5

R SQUARE = 0.90528508

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-0.24946328				
R4	0.05162188	0.00393563	2.95023716	172.04	0.0001

PRACTICAL-MINIMAL COST: PATHOGEN FITNESS
(W [PATHOGEN]) ON ODESSA

R1 R2 R3 R4 R5

R SQUARE = 0.80171475

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-0.06116624				
R5	0.07020985	0.00822995	3.86039517	72.78	0.0001

TABLE 37
(continued)

PRACTICAL-MINIMAL COST: HOST FITNESS
(W [HOST]) ON TREBI

R1 R2 R3 R4 R5

R SQUARE = .94494173

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-1546.463710				
R3	43.439697	2.588944	11393151.5556	281.53	0.0001
R5	-15.317301	5.411363	324240.1646	8.01	0.0115

PRACTICAL-MINIMAL COST: HOST FITNESS
(W [HOST]) ON ODESSA

R1 R2 R3 R4 R5

R SQUARE = 0.94998665

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-743.732847				
R3	44.929569	2.559785	6004329.957726	308.08	0.0001
R5	-34.858192	5.067681	922143.020531	47.31	0.0001

TABLE 38.   Stepwise regression results of the PRACTICAL:
MINIMAL COST models for the dependent variables W
[PATHOGEN]G (pathogen fitness) and W [HOST]G (host
fitness).   Independent variables employed in these
models had significant genetic components and are shown
above the R SQUARE value.   The TERMS column contains
the Y intercept and any independent variable with a
statistically significant F value.   Intercept and term
coefficients are in the B column.   Remaining columns
hold the standard error (SE), sum of squares (SS), F,
and probability of significance values (PROB>F).

TABLE 38

PRACTICAL-MINIMAL COST: PATHOGEN FITNESS
(W [PATHOGEN]G) ON TREBI

R2 R4

SAME AS TRADITIONAL MODEL TW [PATHOGEN]
(SEE TABLE 36)

PRACTICAL-MINIMAL COST: PATHOGEN FITNESS
(W [PATHOGEN]G) ON ODESSA

R1 R2 R4

R SQUARE = 0.59992019

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-0.24464349				
R4	0.04816169	0.00927026	2.88871949	26.99	0.0001

TABLE 38
(continued)

PRACTICAL-MINIMAL COST: HOST FITNESS
(W [HOST]G) ON TREBI

R2 R4

SAME AS TRADITIONAL MODEL TW [HOST]
(SEE TABLE 36)

PRACTICAL-MINIMAL COST: HOST FITNESS
(W [HOST]G) ON ODESSA

R1 R2 R4

R SQUARE = 0.54096301

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	2479.498203				
R2	268.888345	77.004393	2181131.598273	12.19	0.0028
R4	-291.482343	74.830004	2714200.228374	15.17	0.0012

TABLE 39.   Stepwise regression results of the PRACTICAL:
            MODERATE COST models for the dependent variables W
            [PATHOGEN] (pathogen fitness) and W [HOST] (host
            fitness).   Independent variables employed in these
            models are shown above the R SQUARE value.   The TERMS
            column contains the Y intercept and any independent
            variable with a statistically significant F value.
            Intercept and term coefficients are in the B column.
            Remaining columns hold the standard error (SE), sum of
            squares (SS), F, and probability of significance values
            (PROB>F).

TABLE 39

PRACTICAL-MODERATE COST: PATHOGEN FITNESS
(W [PATHOGEN]) ON TREBI
```
------------------------------------------------------
        R1  R2  R3  R4  R5  R6  R7  R8  R9  R10  R11
        H1  H2  H3
        C1  C2  C3
        P1  P2  P3  P4  P5  P6  P7
------------------------------------------------------
              R SQUARE = 0.96195777
------------------------------------------------------
```

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-0.00507149				
R7	3.74983604	0.21441740	2.23045331	305.85	0.0001
P1	-0.06374580	0.02040064	0.07120395	9.76	0.0062

PRACTICAL-MODERATE COST: PATHOGEN FITNESS
(W [PATHOGEN]) ON ODESSA
```
------------------------------------------------------
        R1  R2  R3  R4  R5  R6  R7  R8  R9  R10  R11
        H1  H2  H3
        C1  C2  C3
        P1  P2  P3  P4  P5  P6  P7
------------------------------------------------------
              R SQUARE = 0.80171475
------------------------------------------------------
```

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-0.06116624				
R5	0.07020985	0.00822995	3.86039517	72.78	0.0001

TABLE 39
(continued)

PRACTICAL-MODERATE COST: HOST FITNESS
(W [HOST]) ON TREBI

```
R1  R2  R3  R4  R5  R6  R7  R8  R9  R10  R11
H1  H2  H3
C1  C2  C3
P1  P2  P3  P4  P5  P6  P7
```

R SQUARE = 0.93088798

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-1740.45405				
H3	2222.27928	142.721850	11631598.6730	242.45	0.0001

PRACTICAL-MODERATE COST: HOST FITNESS
(W [HOST]) ON ODESSA

```
R1  R2  R3  R4  R5  R6  R7  R8  R9  R10  R11
H1  H2  H3
C1  C2  C3
P1  P2  P3  P4  P5  P6  P7
```

R SQUARE = 0.96367010

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-808.428914				
H2	44.420031	2.091957	6383185.19730	450.87	0.0001
C3	174.436444	51.552209	162093.39193	11.45	0.0035

248

TABLE 40.   Stepwise regression results of the PRACTICAL:
    MODERATE COST models for the dependent variables W
    [PATHOGEN]G (pathogen fitness) and W [HOST]G (host
    fitness).   Independent variables employed in these
    models had significant genetic components and are shown
    above the R SQUARE value.   The TERMS column contains
    the Y intercept and any independent variable with a
    statistically significant F value.   Intercept and term
    coefficients are in the B column.   Remaining columns
    hold the standard error (SE), sum of squares (SS), F,
    and probability of significance values (PROB>F).

TABLE 40

PRACTICAL-MODERATE COST: PATHOGEN FITNESS
(W [PATHOGEN]G) ON TREBI

```
-----------------------------------------------------------
                    R2  R4  R7  R10
                    H1
                    C1  C2  P5  P6
-----------------------------------------------------------
              R SQUARE = 0.95899962
-----------------------------------------------------------
```

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-0.00780581				
R7	1.86926327	0.54100846	0.09383036	11.94	0.0030
C2	0.03655558	0.01306162	0.06156363	7.83	0.0123

PRACTICAL-MODERATE COST: PATHOGEN FITNESS
(W [PATHOGEN]G) ON ODESSA

```
-----------------------------------------------------------
                  R1  R2  R4  R7  R9  R10
                  H1
                  C1  C2  C3
-----------------------------------------------------------
              R SQUARE = 0.79592837
-----------------------------------------------------------
```

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-0.09410876				
R7	4.21626124	0.50320574	3.83253276	70.20	0.0001

TABLE 40
(continued)

PRACTICAL-MODERATE COST: HOST FITNESS
(W [HOST]G) ON TREBI

                    R2 R4 R7 R10
                    H1
                    C1 C2 P5 P6

            R SQUARE = 0.38410192

    TERMS   B   SE   SS   F   PROB>F
--------- ----------- ---------- -------------- ---- ------
INTERCEPT 4331.736400
R4   -204.471232   78.741865 3052494.948951 6.74 0.0188
R7   9931.412577 4952.896846 1820142.520083 4.02 0.0611

PRACTICAL-MODERATE COST: HOST FITNESS
(W [HOST]G) ON ODESSA

                    R1 R2 R4 R7 R9 R10
                    H1
                    C1 C2 C3

            R SQUARE = 0.67439628

    TERMS   B   SE   SS   F   PROB>F
--------- ----------- --------- -------------- ----- ------
INTERCEPT 1962.578000
R2   256.156791 67.034552 1968583.549921 14.60 0.0015
R4   -178.492415 78.531384   696454.193075   5.17 0.0372
C1   -243.975454 95.279342   883963.425815   6.56 0.0209

TABLE 41.   Stepwise regression results of the PRACTICAL:
EARLY ASSESSMENT models for the dependent variables W
[PATHOGEN] (pathogen fitness) and W [HOST] (host
fitness).   Independent variables employed in these
models are shown above the R SQUARE value.   The TERMS
column contains the Y intercept and any independent
variable with a statistically significant F value.
Intercept and term coefficients are in the B column.
Remaining columns hold the standard error (SE), sum of
squares (SS), F, and probability of significance values
(PROB>F).

TABLE 41

PRACTICAL-EARLY ASSESSMENT: PATHOGEN FITNESS
(W [PATHOGEN]) ON TREBI

R1 R2 R3

R SQUARE = 0.83622239

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-0.31431579				
R2	0.04787175	0.00499355	2.72516849	91.91	0.0001

PRACTICAL-EARLY ASSESSMENT: PATHOGEN FITNESS
(W [PATHOGEN]) ON ODESSA

R1 R2 R3

R SQUARE = 0.57033495

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-0.27599497				
R2	0.04832365	0.00988607	2.74626142	23.89	0.0001

TABLE 41
(continued)

PRACTICAL-EARLY ASSESSMENT: HOST FITNESS
(W [HOST]) ON TREBI

R1 R2 R3

R SQUARE = 0.93593024

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-5666.986634				
R1	74.906008	35.333830	211640.1419	4.49	0.0490
R3	47.269452	3.285085	9750214.0476	207.05	0.0001

PRACTICAL-EARLY ASSESSMENT: HOST FITNESS
(W [HOST]) ON ODESSA

R1 R2 R3

R SQUARE = 0.89578095

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-367.867456				
R2	-21.950300	5.895271	563043.254653	13.86	0.0017
R3	40.750942	3.649147	5064783.977911	124.71	0.0001

TABLE 42.   Stepwise regression results of the DEVELOPMENTAL:
C (COMPLETELY DISEASED PLANTS) OR H (HEALTHY PLANTS)
BASED models for the dependent variables W [PATHOGEN]
(pathogen fitness) and W [HOST] (host fitness).
Independent variables employed in these models are shown
above the R SQUARE value.   The TERMS column contains
the Y intercept and any independent variable with a
statistically significant F value.   Intercept and term
coefficients are in the B column.   Remaining columns
hold the standard error (SE), sum of squares (SS), F,
and probability of significance values (PROB>F).

TABLE 42

DEVELOPMENTAL: PATHOGEN FITNESS
(W [PATHOGEN]) ON TREBI

R1 C3 C5 C7

R SQUARE = 0.44246371

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-0.13541350				
C7	0.01679538	0.00444377	1.44194676	14.28	0.0014

DEVELOPMENTAL: PATHOGEN FITNESS
(W [PATHOGEN]) ON ODESSA

R1 C3 C5 C7

R SQUARE = 0.65934546

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-0.01904564				
C5	4.82156232	0.81686843	3.17486245	34.84	0.0001

TABLE 42
(continued)

DEVELOPMENTAL: HOST FITNESS
(W [HOST]) ON TREBI

R1 H3 H4 H7 H9

R SQUARE = 0.97013702

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-5376.783602				
H3	1132.967435	369.140380	219688.268372	9.42	0.0073
H4	24.548005	7.807633	230541.325208	9.89	0.0063
H9	57.611473	24.655888	127330.307603	5.46	0.0328

DEVELOPMENTAL: HOST FITNESS
(W [HOST]) ON ODESSA

R1 H3 H4 H7 H9

R SQUARE = 0.91899888

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-60.391428				
H4	43.827720	3.066907	6088147.116651	204.22	0.0001

TABLE 43. Stepwise regression results of the DEVELOPMENTAL:
C (COMPLETELY DISEASED PLANTS) OR H (HEALTHY PLANTS)
BASED models for the dependent variables W [PATHOGEN]G
(pathogen fitness) and W [HOST]G (host fitness).
Independent variables employed in this model had
significant genetic components and are shown above the R
SQUARE value.   The TERMS column contains the Y
intercept and any independent variable with a
statistically significant F value.   Intercept and term
coefficients are in the B column.   Remaining columns
hold the standard error (SE), sum of squares (SS), F,
and probability of significance values (PROB>F).

TABLE 43

DEVELOPMENTAL: PATHOGEN FITNESS
(W [PATHOGEN]G) ON TREBI
```
-------------------------------------------------------------
                          C5 C7
-------------------------------------------------------------
               R SQUARE = 0.44246371
-------------------------------------------------------------
   TERMS        B          SE         SS        F    PROB>F
--------- ----------- ---------- ---------- ----- ------
INTERCEPT -0.13541350
C7         0.01679538 0.00444377 1.44194676 14.28 0.0014
```


DEVELOPMENTAL: PATHOGEN FITNESS
(W [PATHOGEN]G) ON ODESSA
```
-------------------------------------------------------------
                        R1 C3 C5 C7
-------------------------------------------------------------
               R SQUARE = 0.65934546
-------------------------------------------------------------
   TERMS        B          SE         SS        F    PROB>F
--------- ----------- ---------- ---------- ----- ------
INTERCEPT -0.01904564
C5         4.82156232 0.81686843 3.17486245 34.84 0.0001
```

TABLE 43
(continued)

DEVELOPMENTAL: HOST FITNESS
(W [HOST]G) ON TREBI

NO APPROPRIATE INDEPENDENT VARIABLES

NO MODEL GENERATED

DEVELOPMENTAL: HOST FITNESS
(W [HOST]G) ON ODESSA

R1

NO MODEL GENERATED

260

TABLE 44.   Stepwise regression results of the DEVELOPMENTAL:
    P (PARTIALLY DISEASED PLANTS) BASED (HOST PERSPECTIVE)
    models for the dependent variables W [PATHOGEN]
    (pathogen fitness) and W [HOST] (host fitness).
    Independent variables employed in these models are shown
    above the R SQUARE value.   The TERMS column contains
    the Y intercept and any independent variable with a
    statistically significant F value.   Intercept and term
    coefficients are in the B column.   Remaining columns
    hold the standard error (SE), sum of squares (SS), F,
    and probability of significance values (PROB>F).

TABLE 44

DEVELOPMENTAL: PATHOGEN FITNESS
(W [PATHOGEN]) ON TREBI

R1 P5 P7 P13 P16 P18

R SQUARE = 0.45024903

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-0.01269729				
P18	0.01107389	0.00288417	1.46731835	14.74	0.0012

DEVELOPMENTAL: PATHOGEN FITNESS
(W [PATHOGEN]) ON ODESSA

R1 P5 P7 P13 P16 P18

R SQUARE = 0.41374006

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	0.30816765				
P5	0.29291859	0.08218490	1.99222996	12.70	0.0022

TABLE 44
(continued)

DEVELOPMENTAL: HOST FITNESS
(W [HOST]) ON TREBI

R1 P5 P7 P13 P16 P18

NO MODEL GENERATED

DEVELOPMENTAL: HOST FITNESS
(W [HOST]) ON ODESSA

R1 P5 P7 P13 P16 P18

NO MODEL GENERATED

TABLE 45.   Stepwise regression results of the DEVELOPMENTAL:
   P (PARTIALLY DISEASED PLANTS) BASED (HOST PERSPECTIVE)
   models for the dependent variables W [PATHOGEN]G
   (pathogen fitness) and W [HOST]G (host fitness).
   Independent variables employed in these models had
   significant genetic components and are shown above the R
   SQUARE value.   The TERMS column contains the Y
   intercept and any independent variable with a
   statistically significant F value.   Intercept and term
   coefficients are in the B column.   Remaining columns
   hold the standard error (SE), sum of squares (SS), F,
   and probability of significance values (PROB>F).

TABLE 45

DEVELOPMENTAL: PATHOGEN FITNESS
(W [PATHOGEN]G) ON TREBI

P5 P18

R SQUARE = 0.45024903

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-0.01269729				
P18	0.01107389	0.00288417	1.46731835	14.74	0.0012

DEVELOPMENTAL: HOST FITNESS
(W [HOST]G) ON ODESSA

R1

NO MODEL GENERATED

TABLE 45
(continued)

DEVELOPMENTAL: HOST FITNESS
(W [HOST]G) ON TREBI

P5 P18

NO MODEL GENERATED

DEVELOPMENTAL: HOST FITNESS
(W [HOST]G) ON ODESSA

R1

NO MODEL GENERATED

TABLE 46.   Stepwise regression results of the DEVELOPMENTAL:
      P (PARTIALLY DISEASED PLANTS) BASED (PATHOGEN
      PERSPECTIVE) models for the dependent variables W
      [PATHOGEN] (pathogen fitness) and W [HOST] (host
      fitness).   Independent variables employed in these
      models are shown above the R SQUARE value.   The TERMS
      column contains the Y intercept and any independent
      variable with a statistically significant F value.
      Intercept and term coefficients are in the B column.
      Remaining columns hold the standard error (SE), sum of
      squares (SS), F, and probability of significance values
      (PROB>F).

TABLE 46

DEVELOPMENTAL: PATHOGEN FITNESS
(W [PATHOGEN]) ON TREBI

R1 P5 P6 P9 P11

R SQUARE = 0.47067011

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-0.02651632				
P11	0.01719068	0.00429696	1.53386871	16.01	0.0008

DEVELOPMENTAL: PATHOGEN FITNESS
(W [PATHOGEN]) ON ODESSA

R1 P5 P6 P9 P11

R SQUARE = 0.70754410

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	0.13319956				
P6	-0.95003205	0.42169327	0.44672007	5.08	0.0387
P9	6.77381373	1.99178439	1.01796803	11.57	0.0037
P11	0.02746657	0.00932473	0.76363961	8.68	0.0095

TABLE 46
(continued)

DEVELOPMENTAL: HOST FITNESS
(W [HOST]) ON TREBI

R1 P5 P6 P9 P11

NO MODEL GENERATED

DEVELOPMENTAL: HOST FITNESS
(W [HOST]) ON ODESSA

R1 P5 P6 P9 P11

NO MODEL GENERATED

TABLE 47.   Stepwise regression results of the DEVELOPMENTAL:
       P (PARTIALLY DISEASED PLANTS) BASED (PATHOGEN
       PERSPECTIVE) models for the dependent variables W
       [PATHOGEN]G (pathogen fitness) and W [HOST]G (host
       fitness).   Independent variables employed in these
       models had significant genetic components and are shown
       above the R SQUARE value.   The TERMS column contains
       the Y intercept and any independent variable with a
       statistically significant F value.   Intercept and term
       coefficients are in the B column.   Remaining columns
       hold the standard error (SE), sum of squares (SS), F,
       and probability of significance values (PROB>F).

TABLE 47

DEVELOPMENTAL: PATHOGEN FITNESS
(W [PATHOGEN]G) ON TREBI

P5 P6 P9 P11

R SQUARE = 0.47067011

TERMS	B	SE	SS	F	PROB>F
INTERCEPT	-0.02651632				
P11	0.01719068	0.00429696	1.53386871	16.01	0.0008

DEVELOPMENTAL: PATHOGEN FITNESS
(W [PATHOGEN]G) ON ODESSA

R1

NO MODEL GENERATED

TABLE 47
(continued)

DEVELOPMENTAL: HOST FITNESS
(W [HOST]G) ON TREBI

P5 P6 P9 P11

NO MODEL GENERATED

DEVELOPMENTAL: HOST FITNESS
(W [HOST]G) ON ODESSA

R1

NO MODEL GENERATED

TABLE 48.   Spearman rank correlation coefficients (r) and associated probabilities (P) for variables ranked on varieties Trebi and Odessa.

TABLE 48

VARIABLE	r	P
R2	0.8714	0.0001
R4	0.8526	0.0001
Wp [PATHOGEN]	0.4118	0.0712
Wc [PATHOGEN]	0.7180	0.0004
W [PATHOGEN]	0.7134	0.0004
Wp [HOST]	0.0016	0.9948
Wh [HOST]	0.2692	0.2511
W [HOST]	0.2165	0.3591

TABLE 49.   Spearman rank correlation coefficients (r) and
      associated probabilities (P) for ranking of specified
      variable pairs on Trebi (T) and on Odessa (O).

TABLE 49

VARIABLES		r	P
TW [PATHOGEN]	TW [HOST]	-0.4096	0.0729
OW [PATHOGEN]	OW [HOST]	-0.2560	0.2759

<u>11.3</u> <u>APPENDIX</u> <u>C</u>

This appendix contains all figures associated with this study.

FIGURE 1

A Schematic Representation of the Life Cycle of
Ustilago hordei
(from Ebba, 1974)
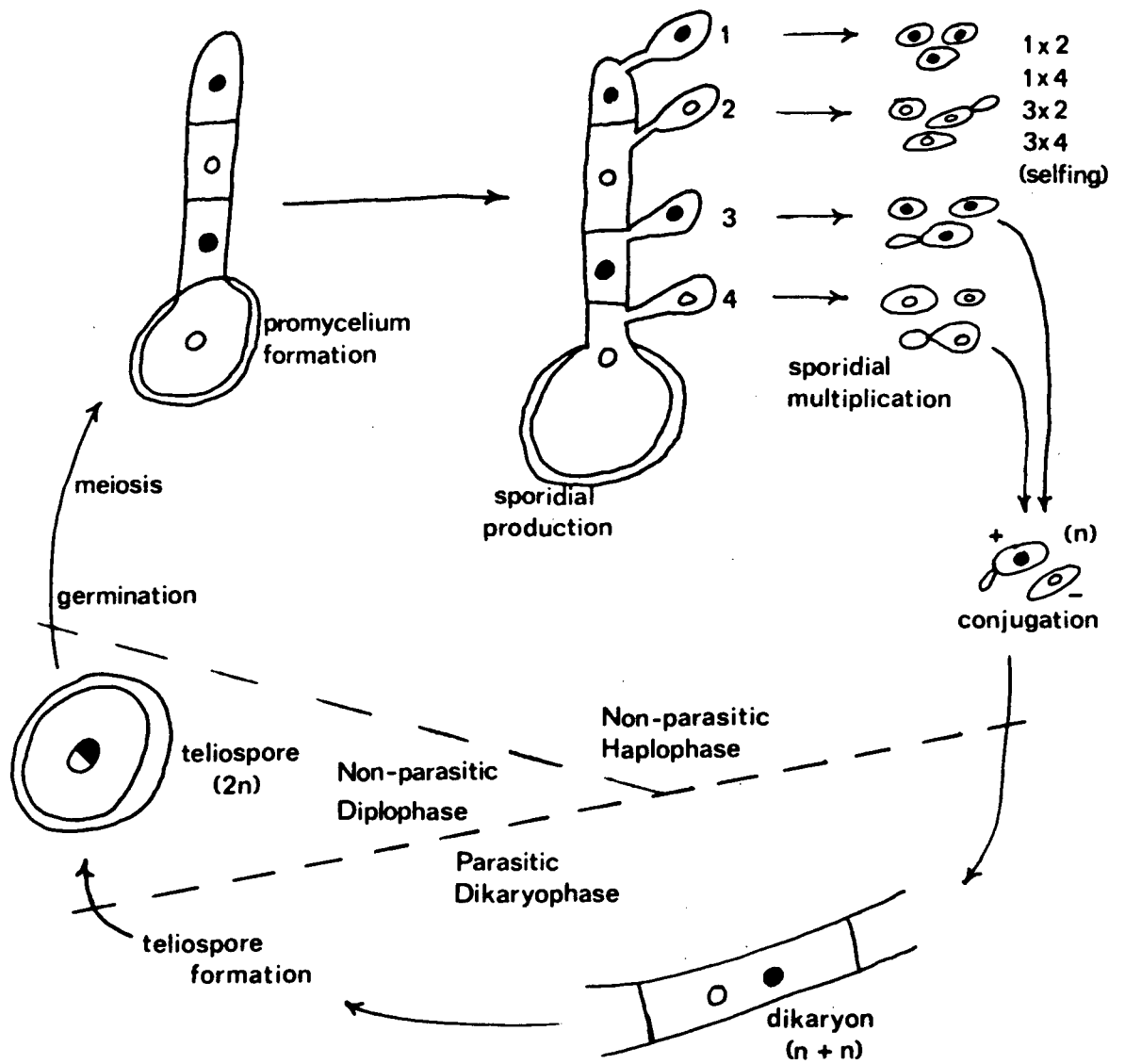
FIGURE 2.   Schematic representation of the experimental
      design.   Teliospores (2N) are represented by squares
      and sporidia (1N) are represented by circles.   Genetic
      crosses are shown by an "X".   Teliospore and sporidium
      genotype is indicated by the virulence allele symbols (V
      or v).   Parental teliospores T1 (VV) and T4 (vv) were
      crossed to produce 8 F1 dikaryotic lines.   Each F1 line
      was heterozygous for the virulence gene (Vv).   Two
      sporidia containing the dominant virulence allele (V),
      but differing in their nonspecific pathogenicity
      (Person, 1983), were isolated from the F1 population and
      crossed to produce F2 teliospores (VV).   Ten F2
      sporidia, 5 of each mating type ("+" and "-"), were
      isolated at random from the F2.   A sporidium of the "+"
      mating type was subsequently lost.   The remaining 9
      sporidia were combined in all possible ways to produce
      20 treatment dikaryons that were expected to vary for
      nonspecific pathogenicity.   Seeds of the varieties
      Trebi and Odessa were inoculated with the treatment
      dikaryons.

FIGURE 2
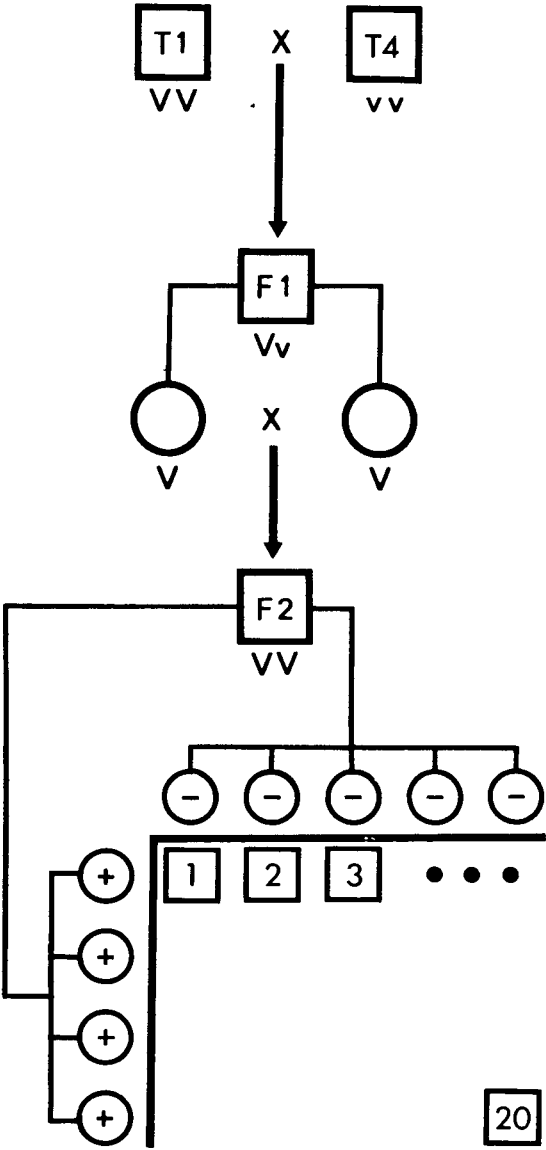
FIGURE 3.   Schematic representation of the relationship of
     the 4 subsets of variables.   Located in the center of
     the diagram is a thin vertical rectangle delimited with
     solid and broken lines.   This rectangle is a symbolic
     representation of a treatment row consisting of all
     treated plants at harvest.   The rectangle or row is
     subdivided into 2 areas.   The first area is bounded by
     solid lines and corresponds to the first 50 plants in
     the row.   The second smaller area is bounded by broken
     lines and corresponds to all plants other than the first
     50 plants.   All variables calculated from the first 50
     plants were catagorized as R subset variables (row).
     One R variable (the germination rate of the 110 treated
     seeds originally planted, R1) was calculated from all
     plants in the row.   The first area bounded by solid
     lines was further subdivided into three smaller areas by
     differential shading (unshaded, completely shaded and
     partially shaded).   The areas are labelled H, C, and P
     to correspond to variables calculated from healthy,
     completely diseased and partially diseased plants.

FIGURE 3

FIGURE 4

TELIOSPORE WEIGHT (GM)

REGRESSION: TELIOSPORE NUMBER vs TELIOSPORE WEIGHT

SLOPE=1.241e10 +/- 3.667e8

Y INTERCEPT FORCED THROUGH ORIGIN
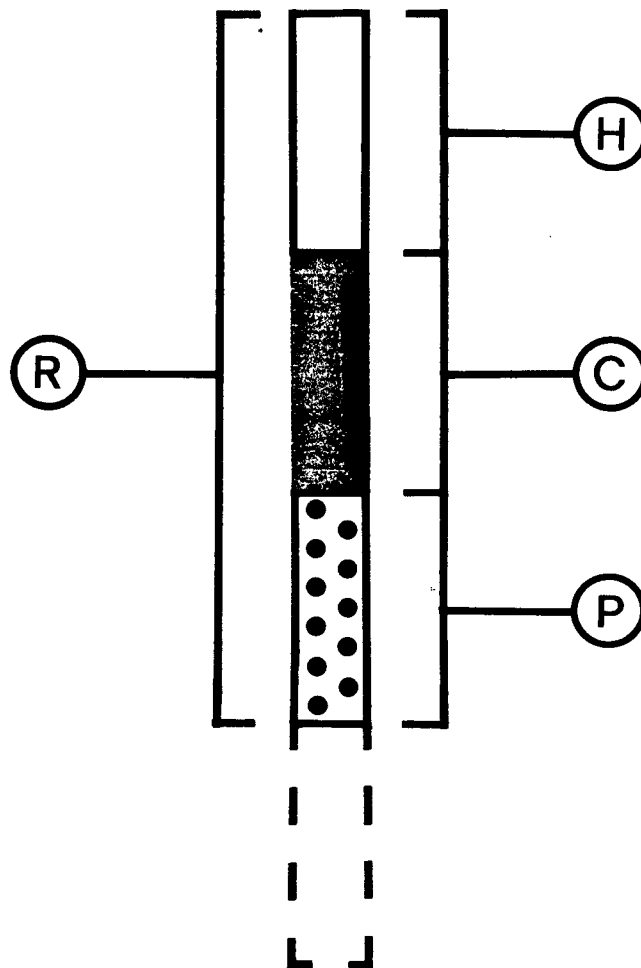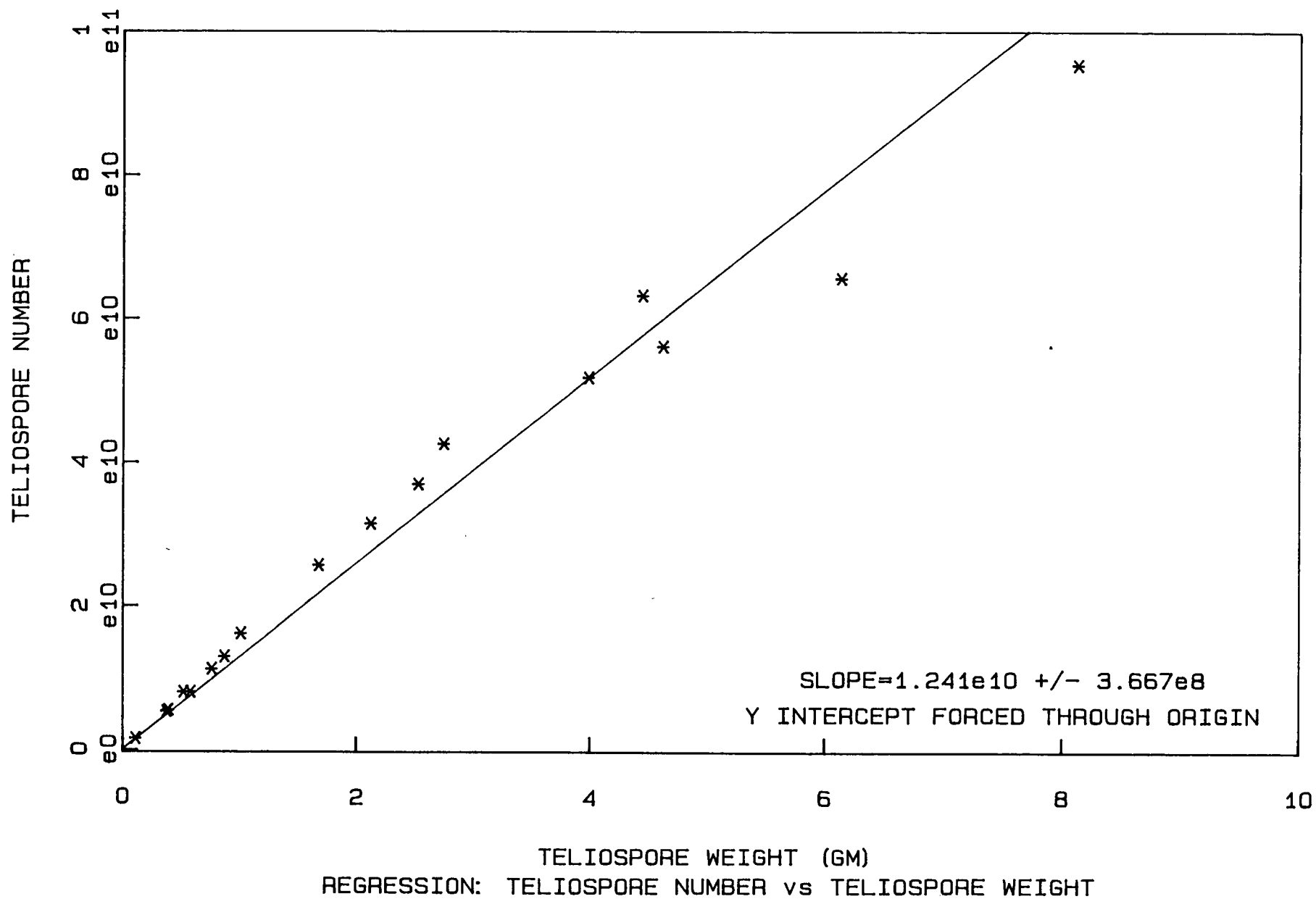
FIGURES 5 to 62.   The following graphs are frequency
      histograms for the 58 fitness related variables.
      Inverted triangles indicate variable means.

FIGURE 5

Odessa

Trebi

R1t
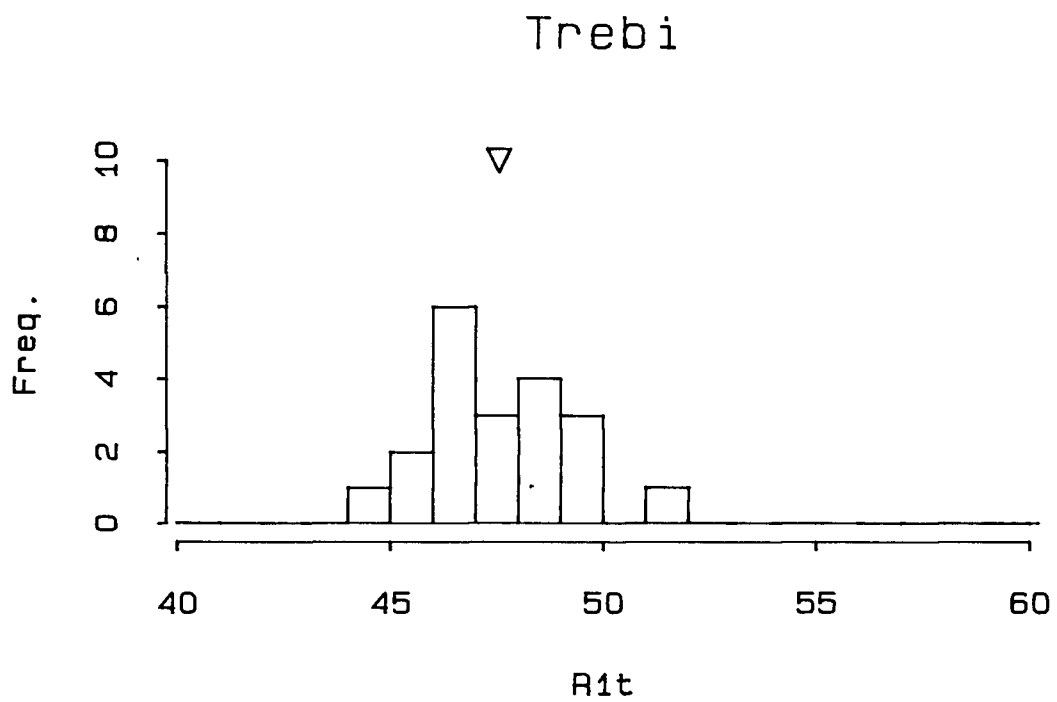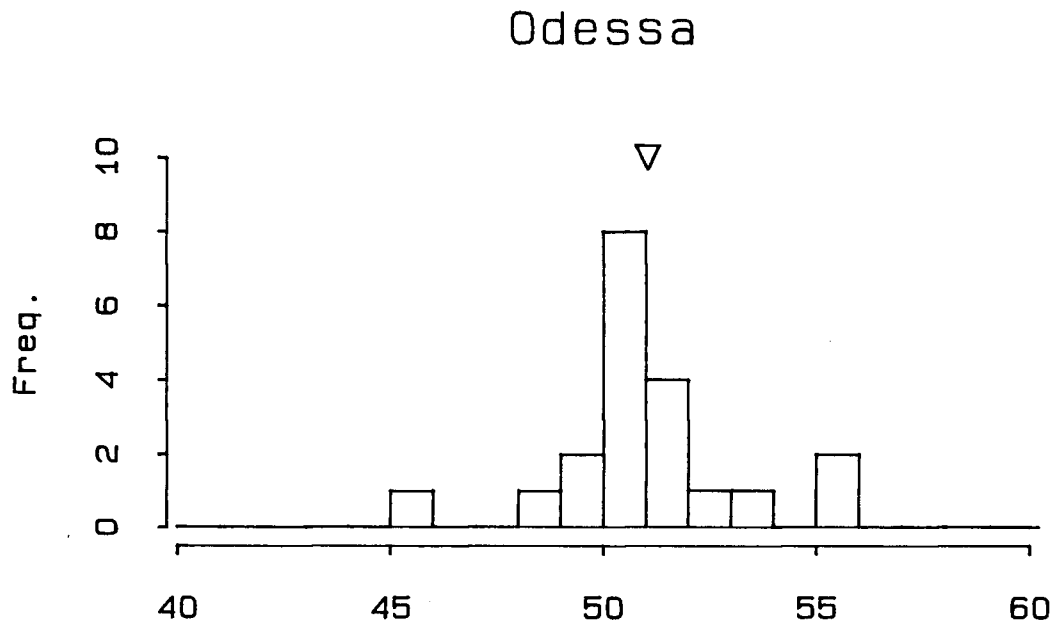
FIGURE 6

Odessa

Trebi

R2t
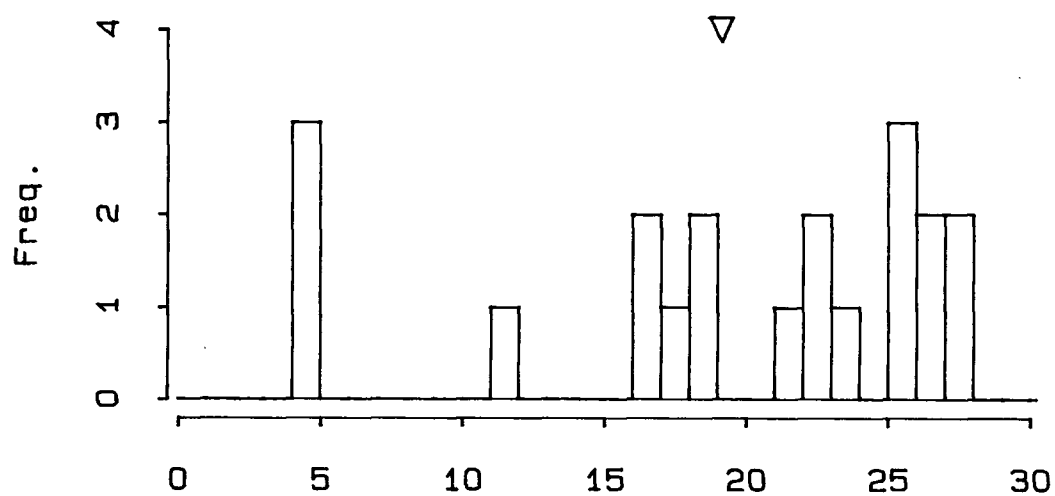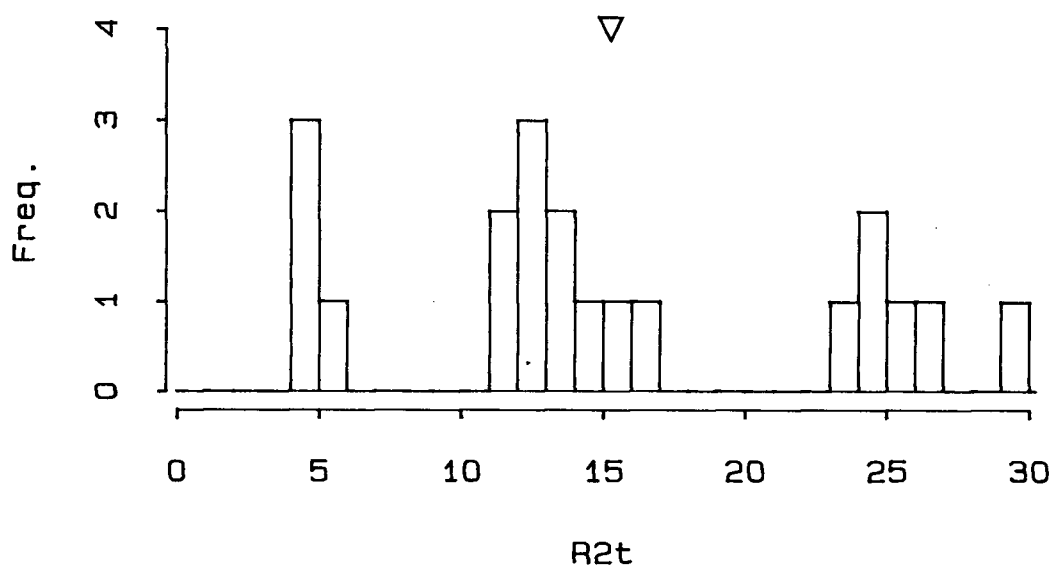
FIGURE 7

Odessa

Trebi

R3
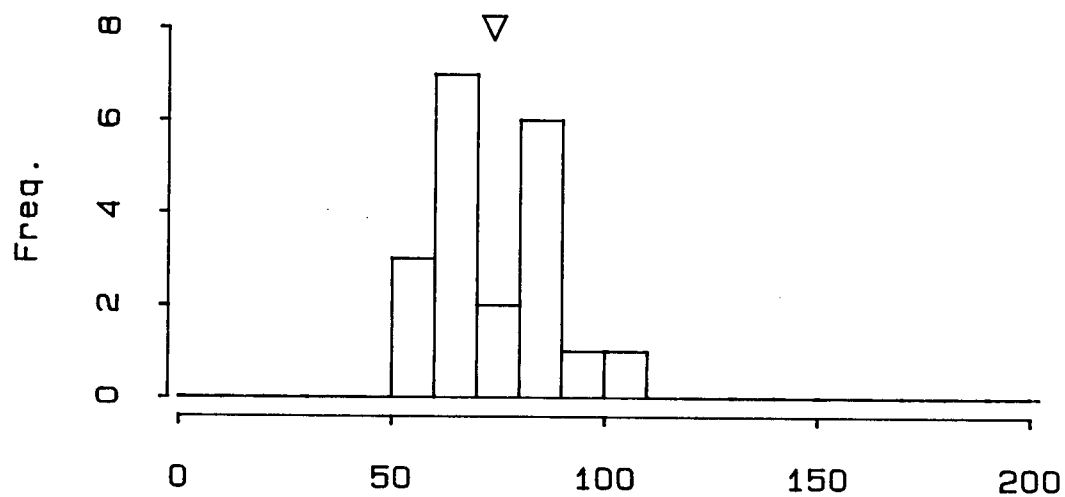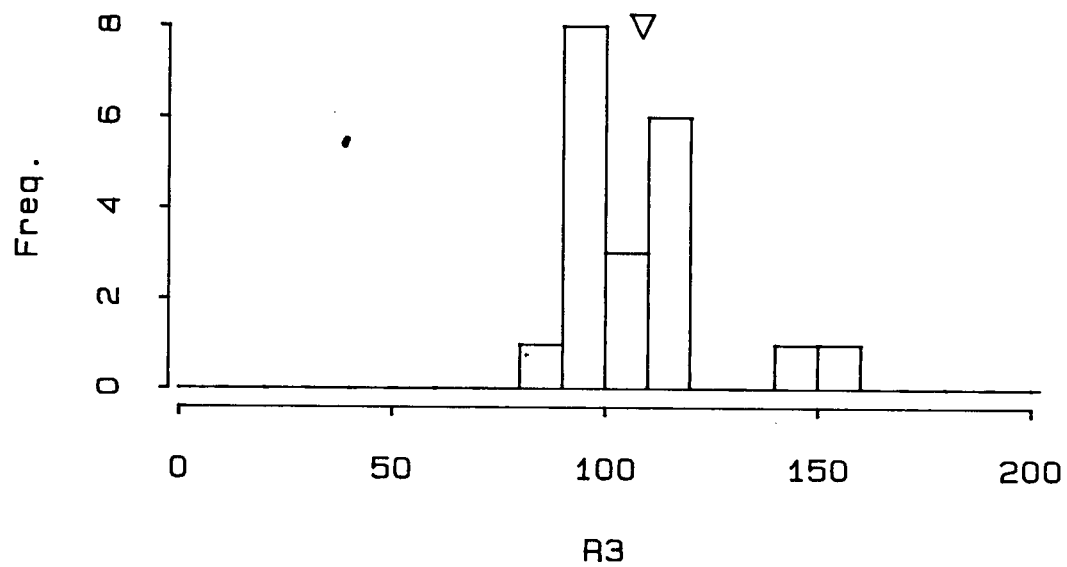
FIGURE 8

Odessa

Trebi

R4t
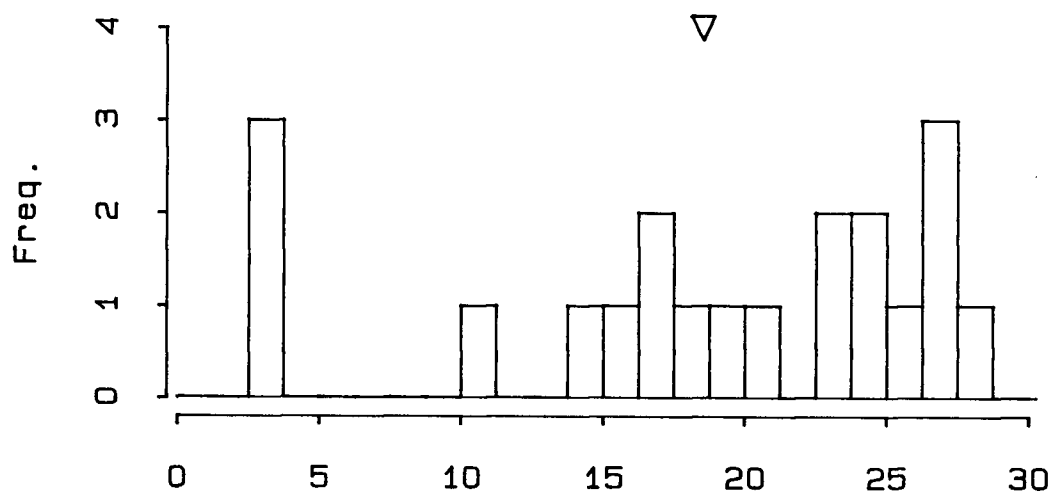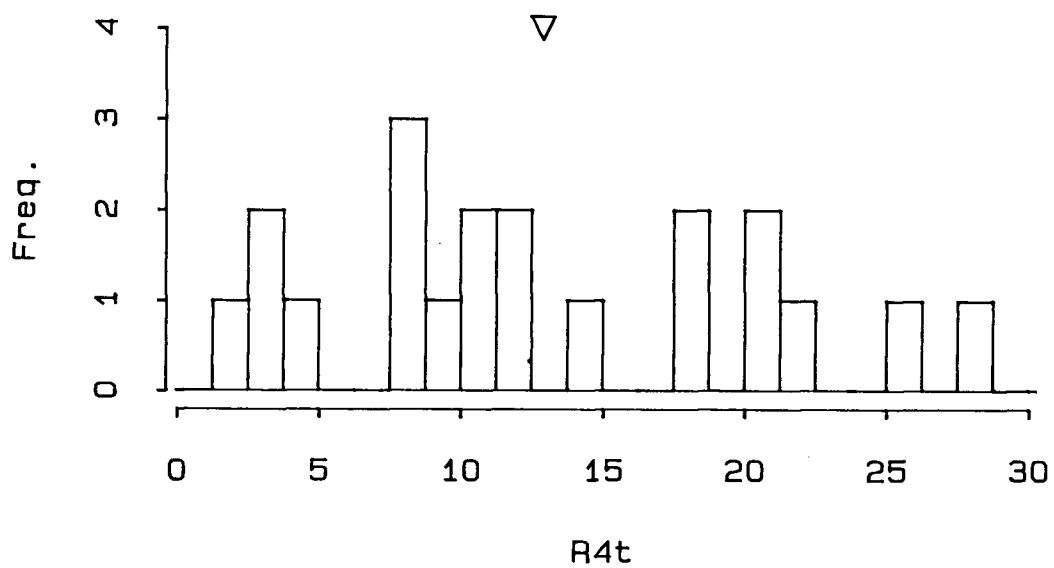
FIGURE 9

Odessa

Trebi

R5
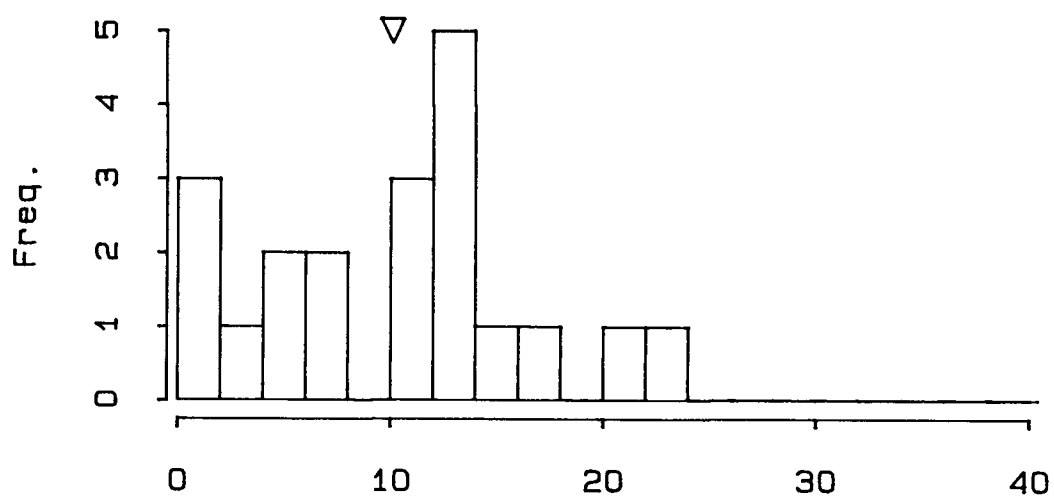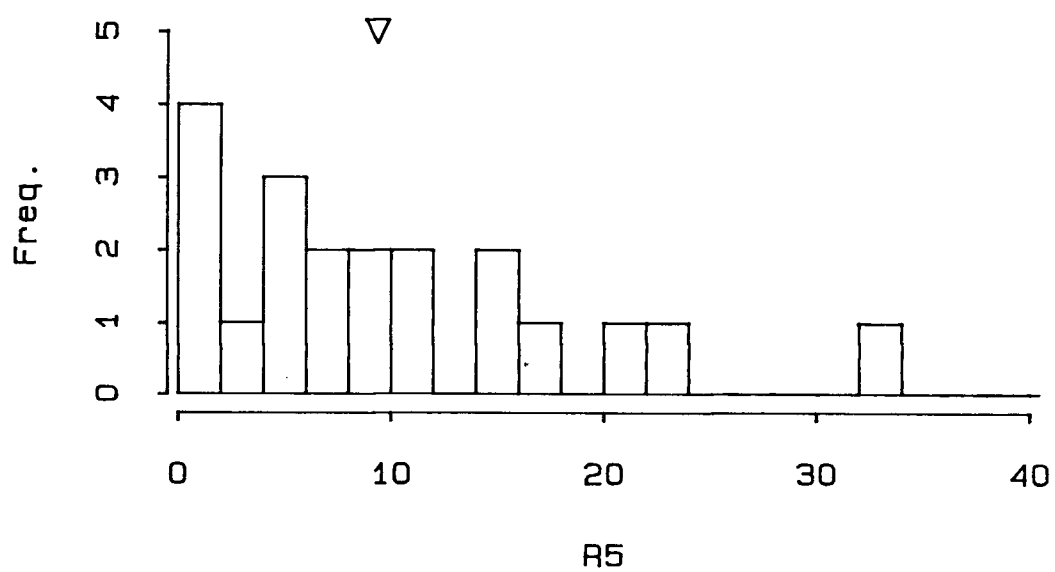
FIGURE 10

Odessa

Trebi

R6
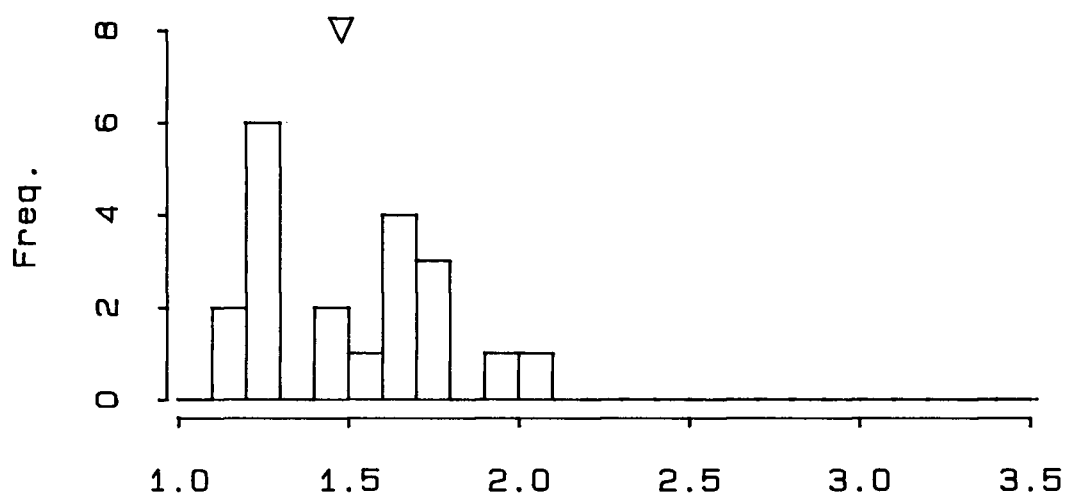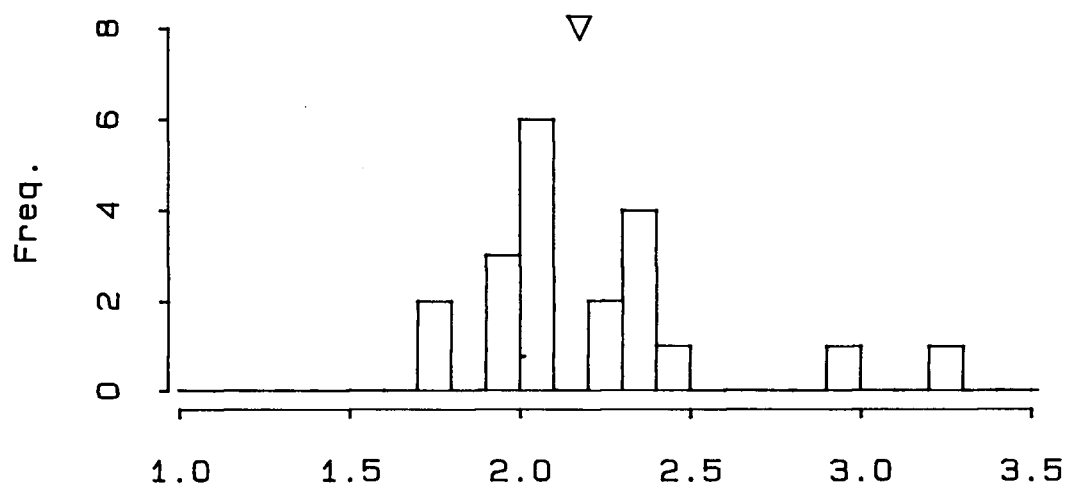
FIGURE 11
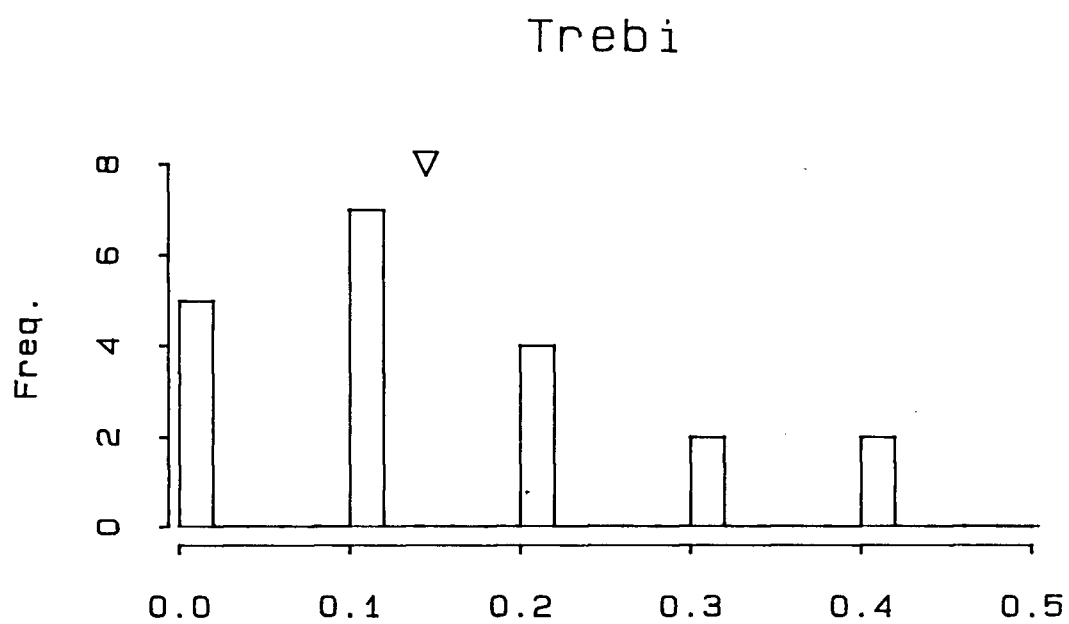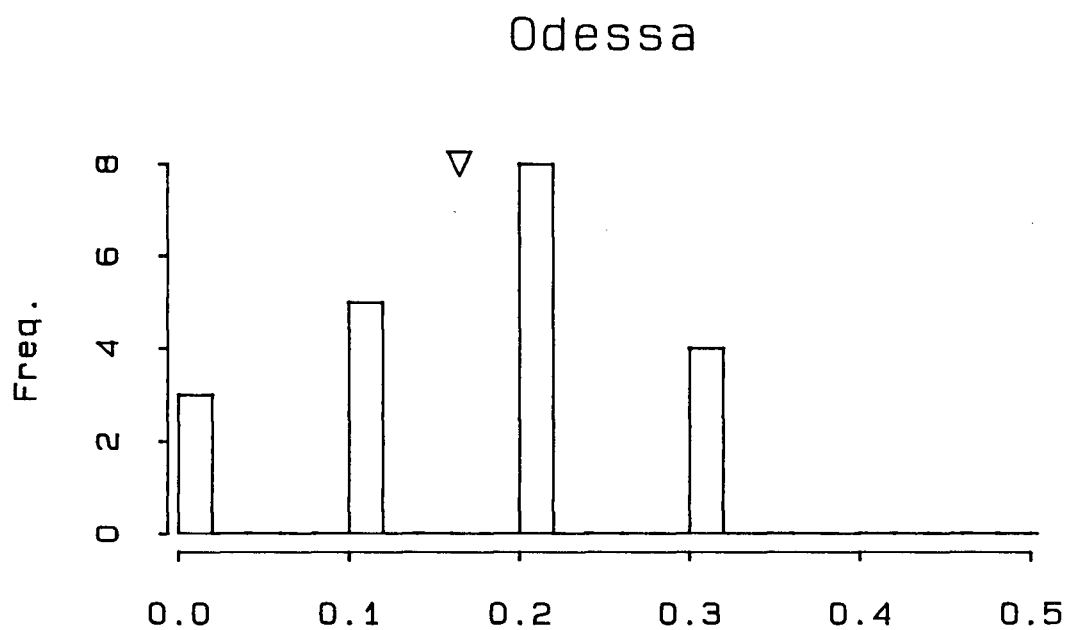
Odessa

Trebi

R7

FIGURE   12

Odessa

Trebi

R8
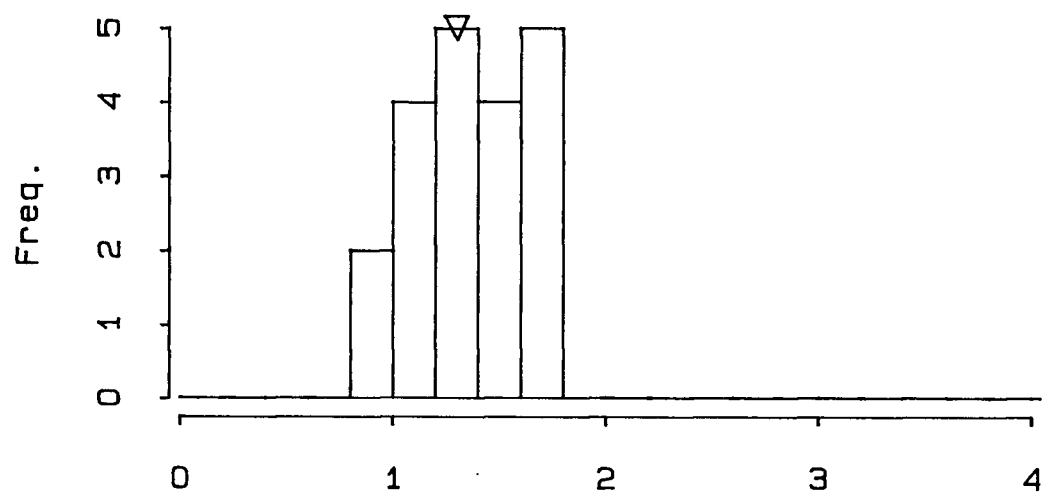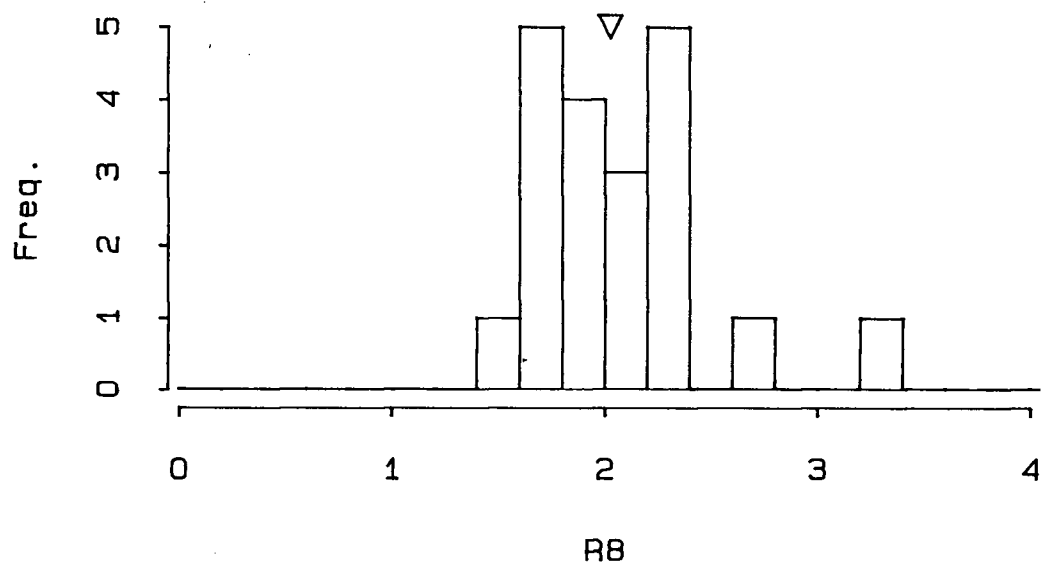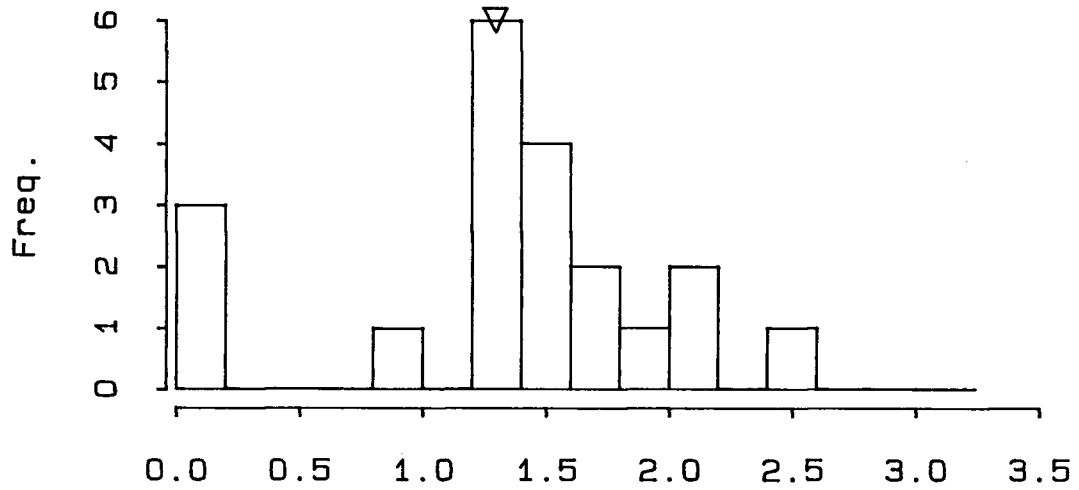
FIGURE 13

Odessa

Trebi
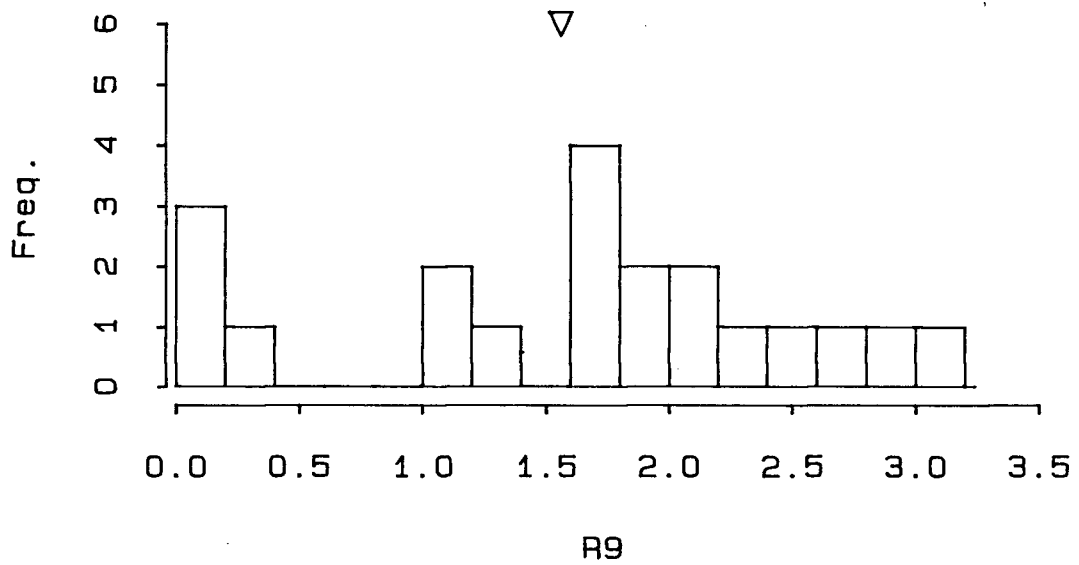
R9

FIGURE 14

Odessa

Trebi

R10
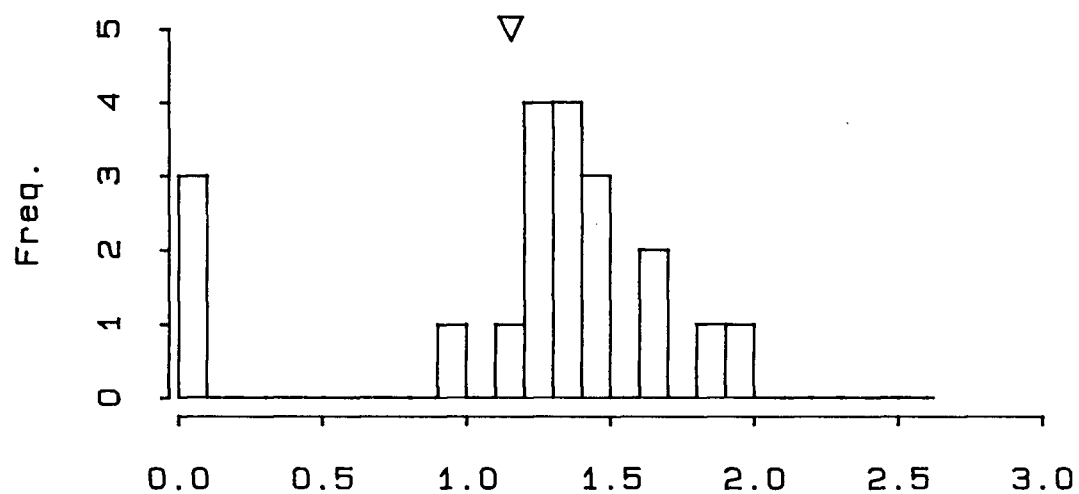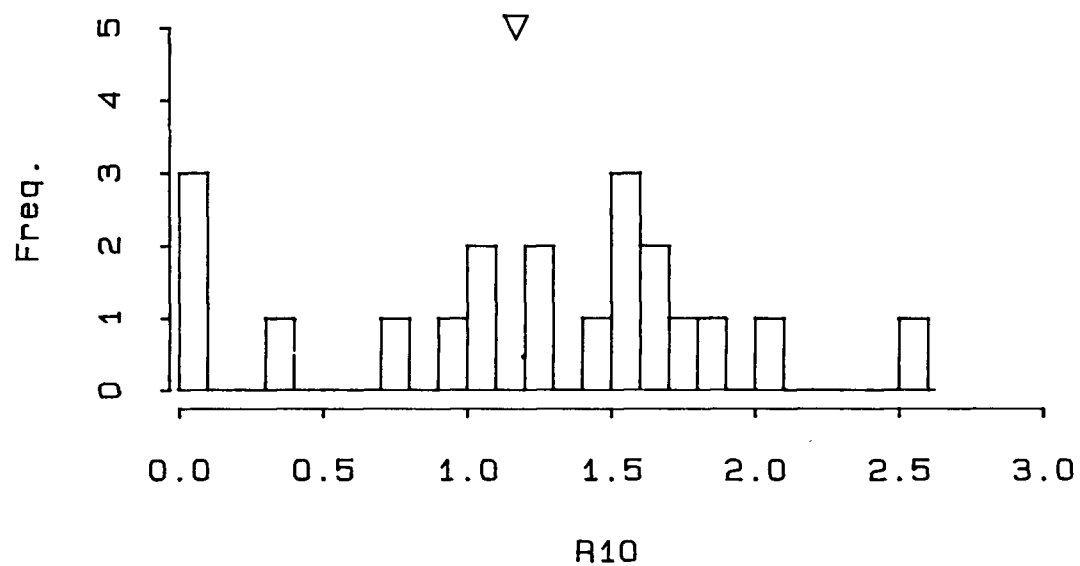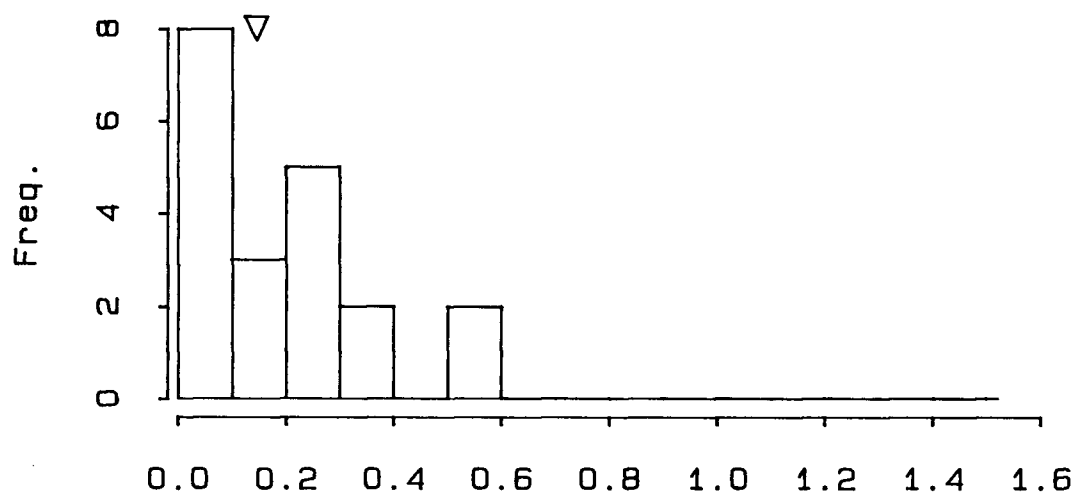
FIGURE 15

Odessa

Trebi
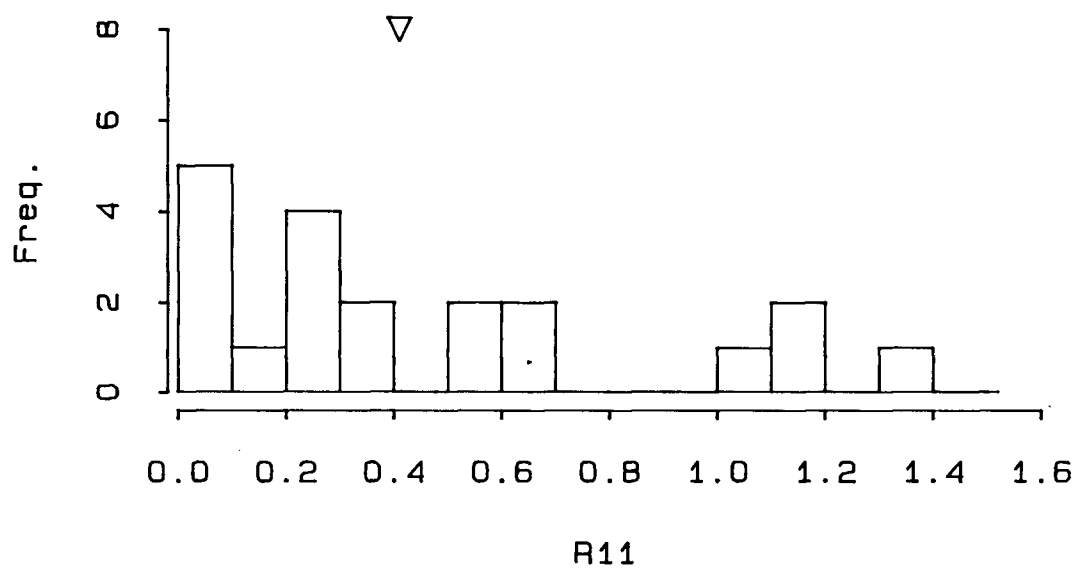
R11

FIGURE 16

Odessa

Trebi

R12
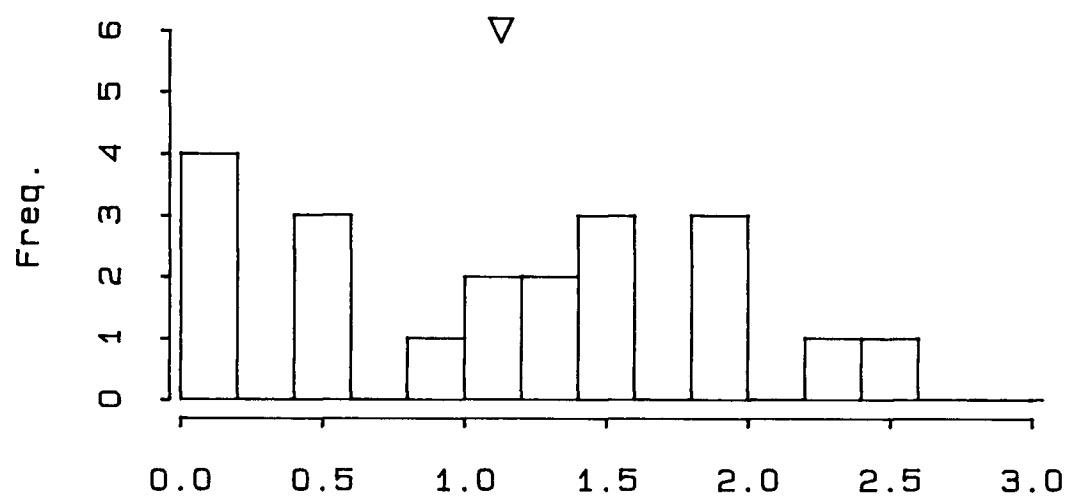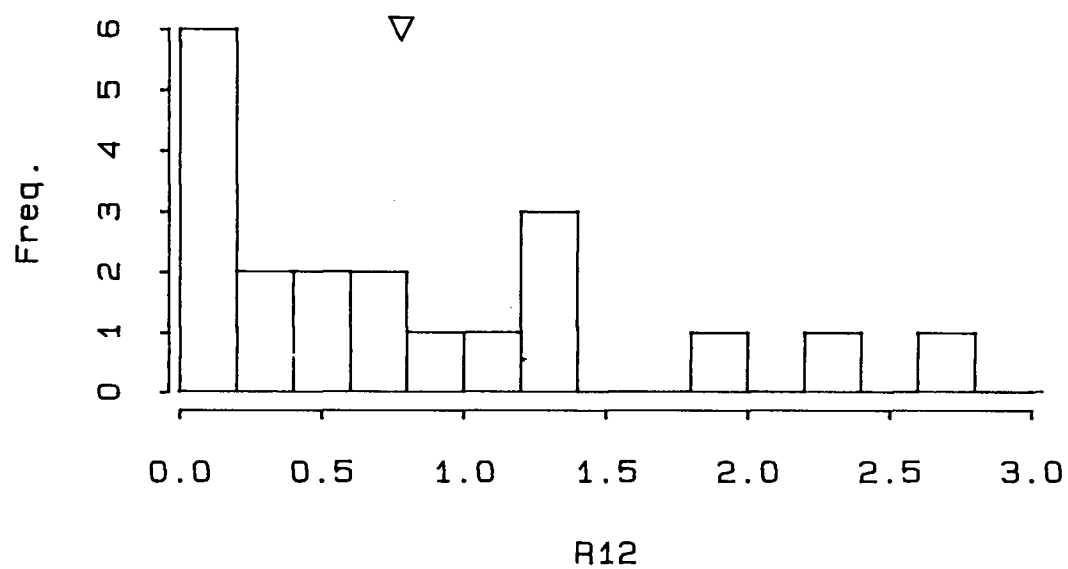
FIGURE 17

Odessa

Trebi

R13
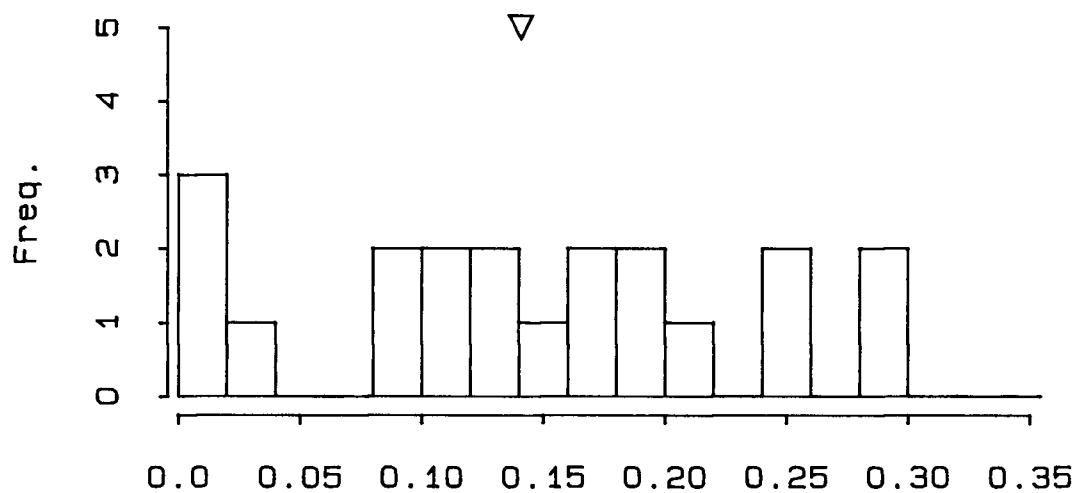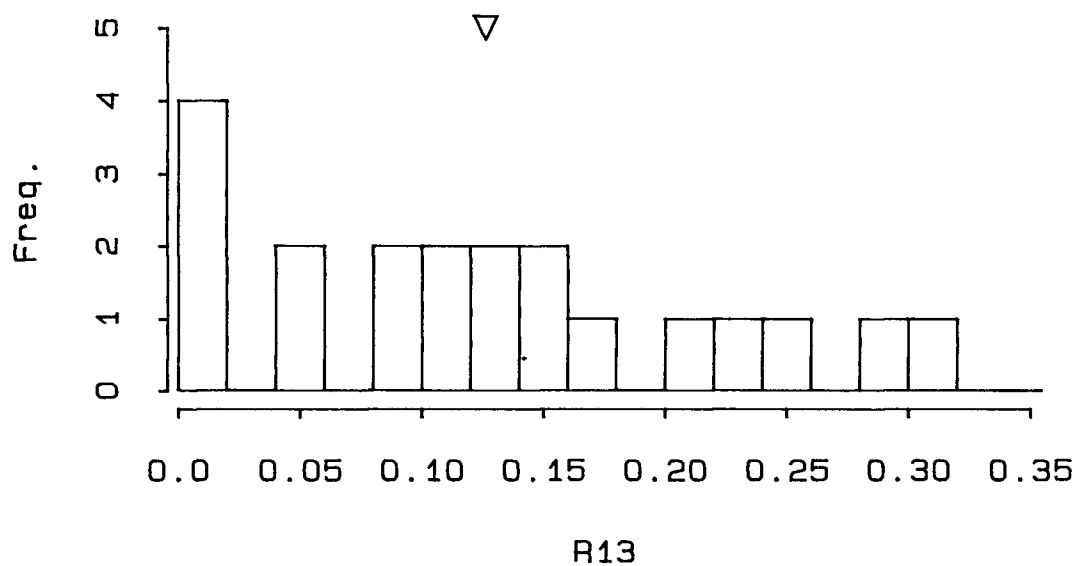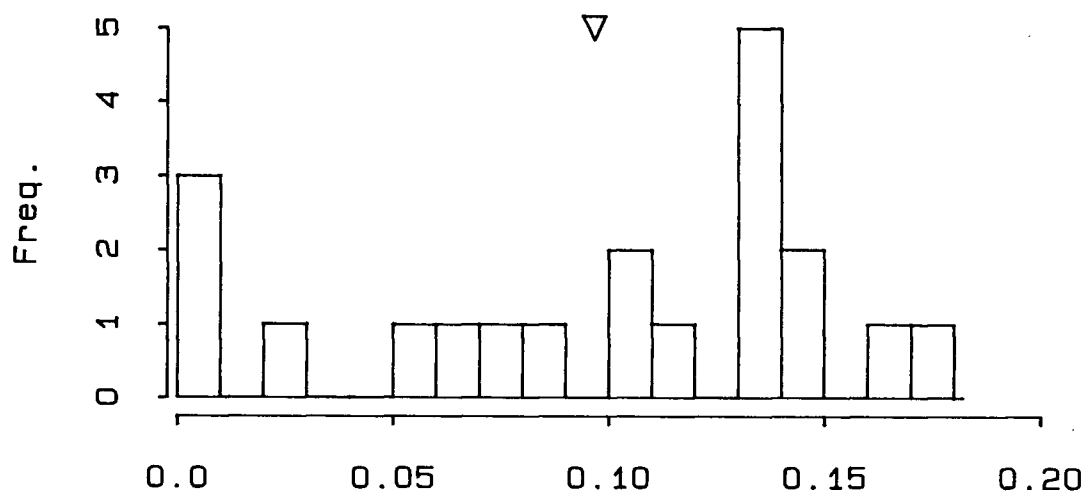
FIGURE 18

Odessa

Trebi
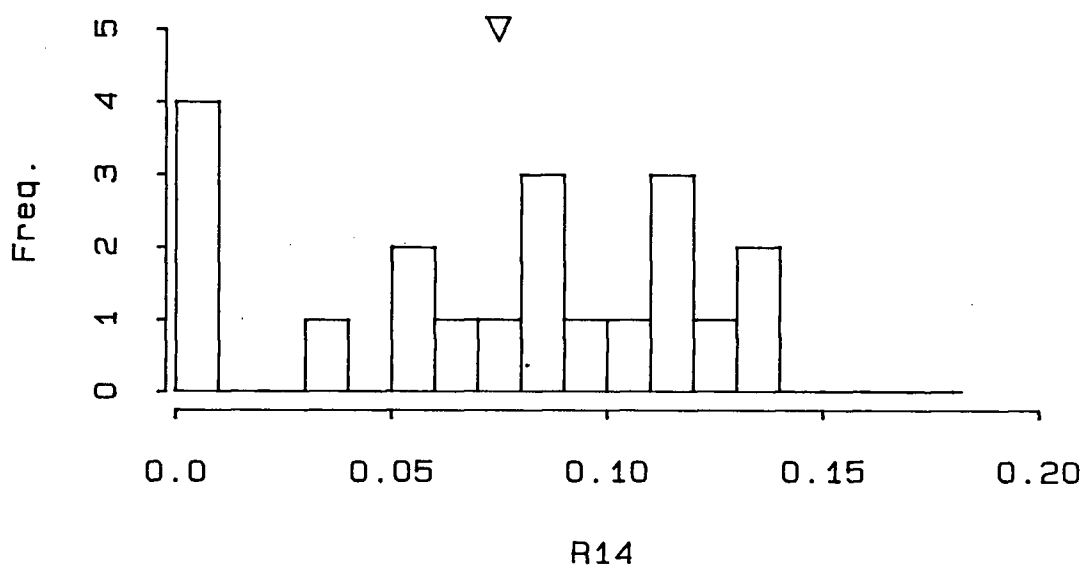
R14

FIGURE   19

Odessa

Trebi

R15t
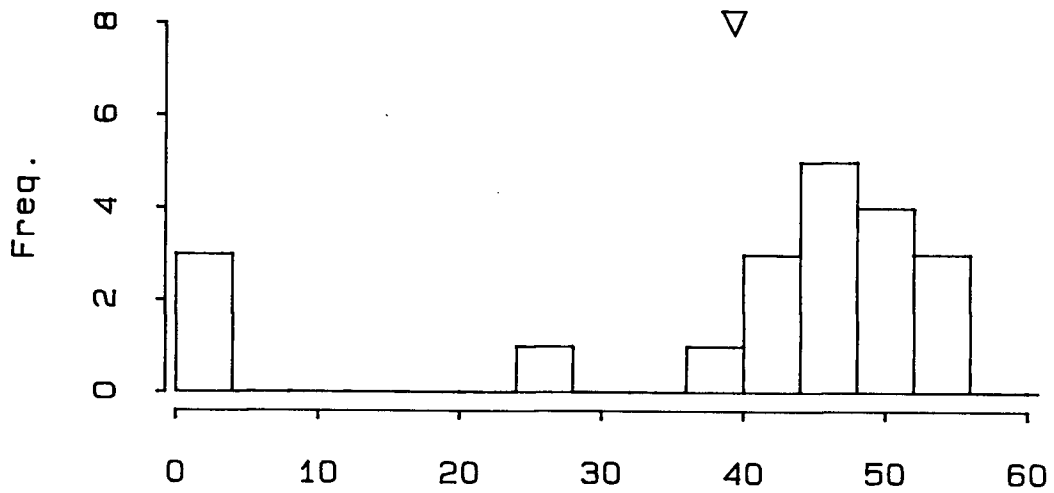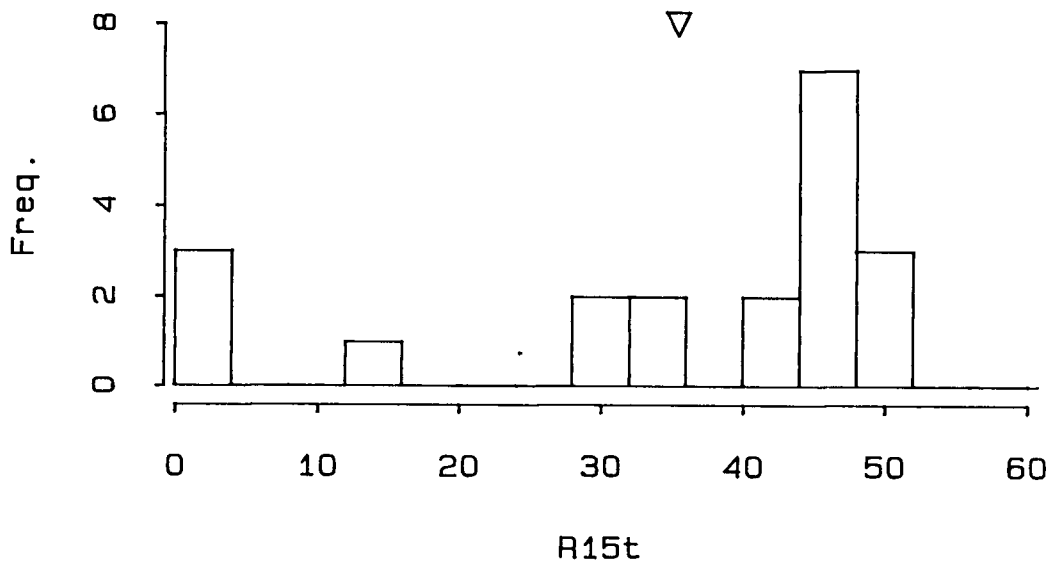
FIGURE 20

Odessa

Trebi

R16
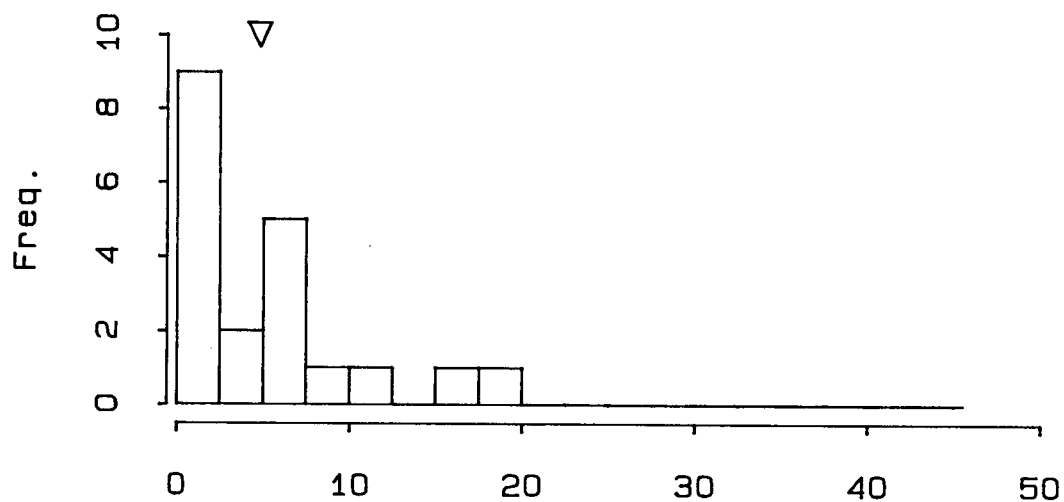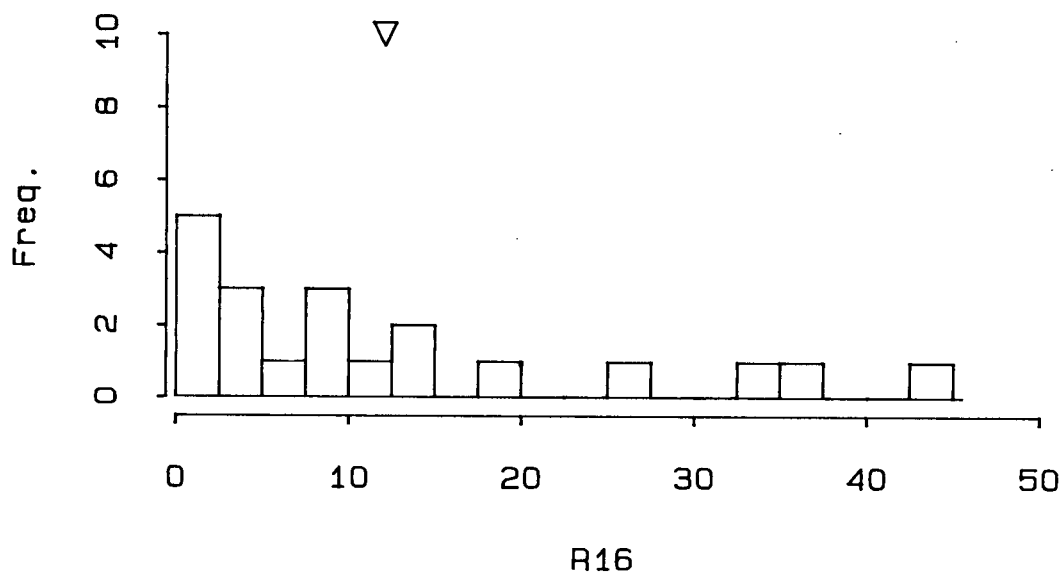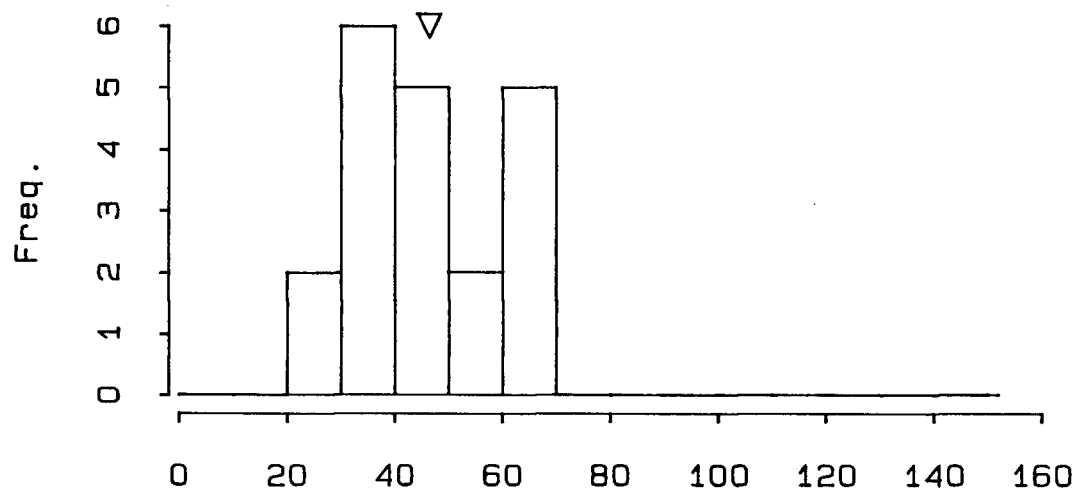
FIGURE 21

Odessa

Trebi
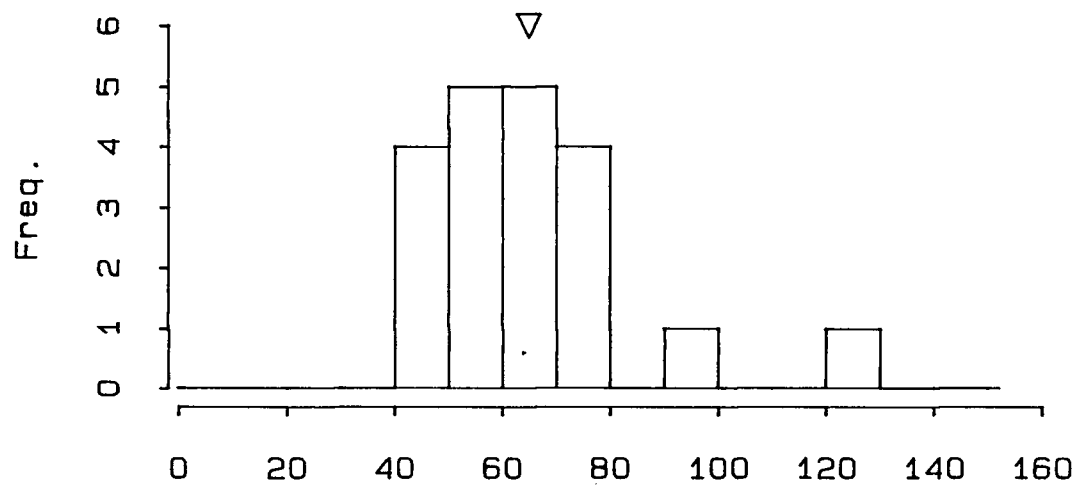
R17

FIGURE 22

Odessa

Trebi

Wp [PATHOGEN]
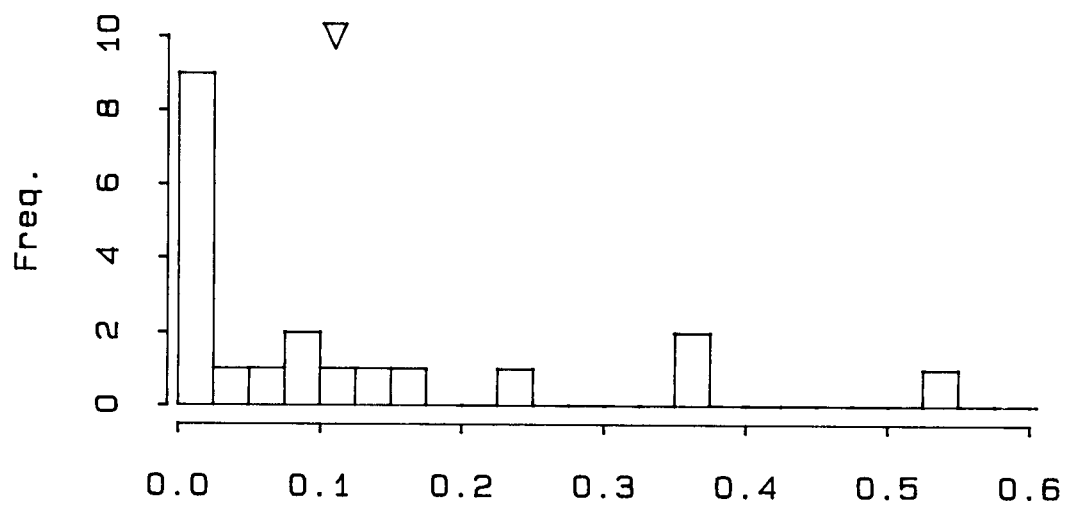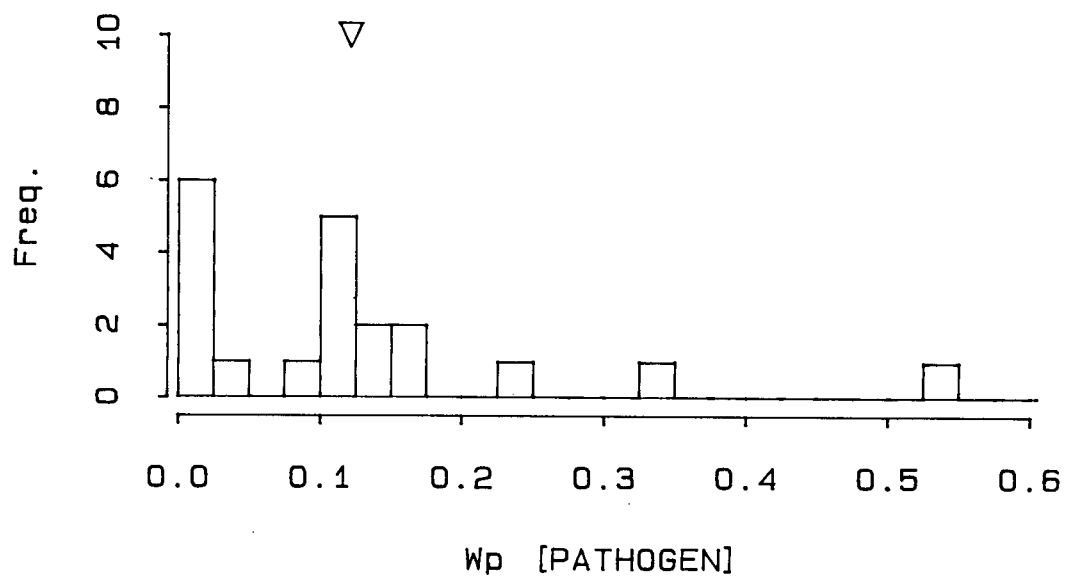
FIGURE 23

Odessa

Trebi

Wc [PATHOGEN]
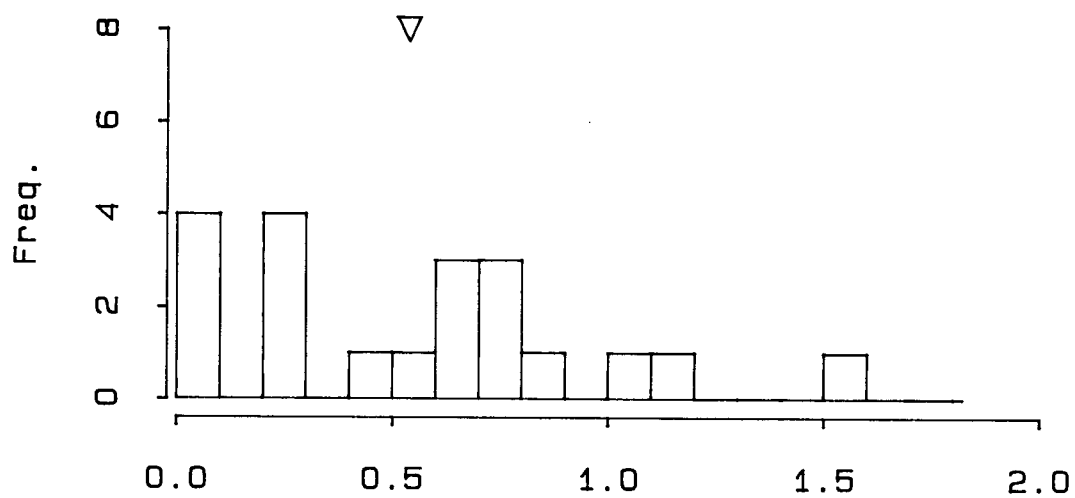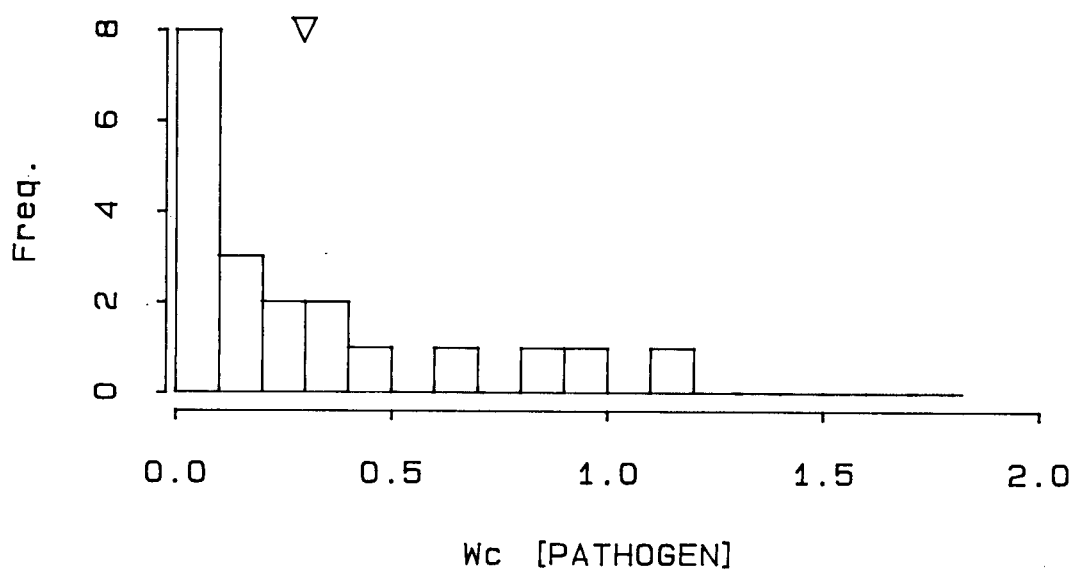
FIGURE 24

Odessa

Trebi

W [PATHOGEN]
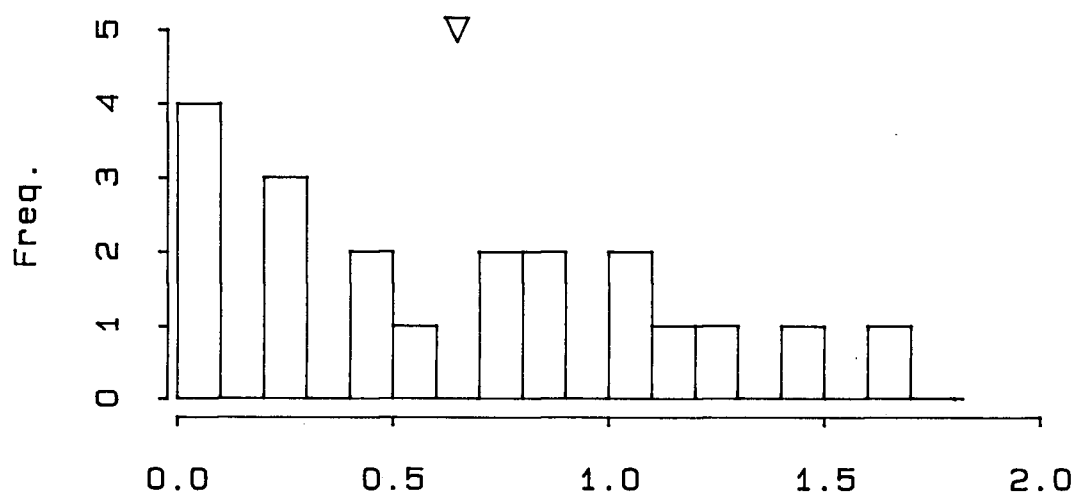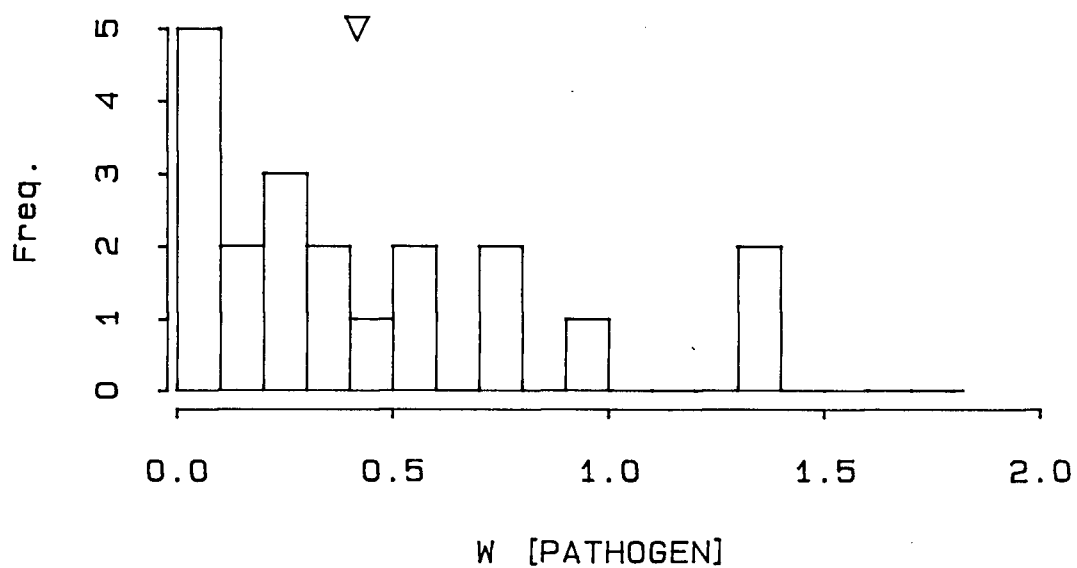
FIGURE   25

Odessa

Trebi

Wp [HOST]
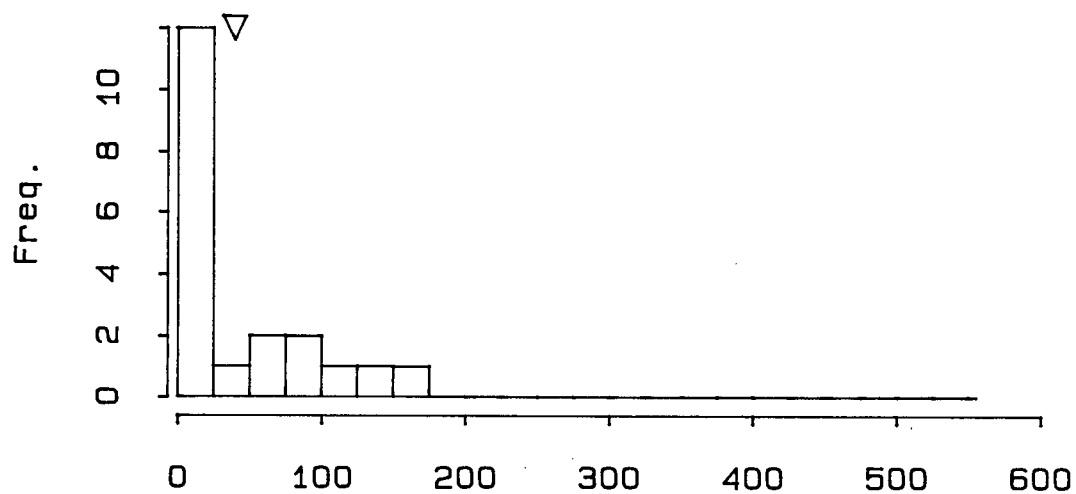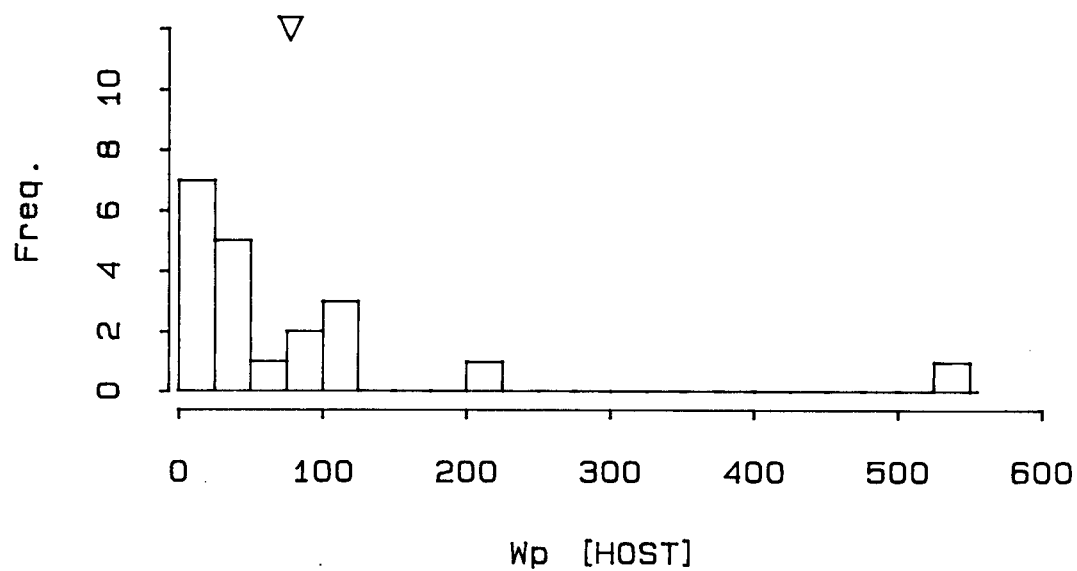
FIGURE   26

Odessa

Trebi

Wh [HOST]
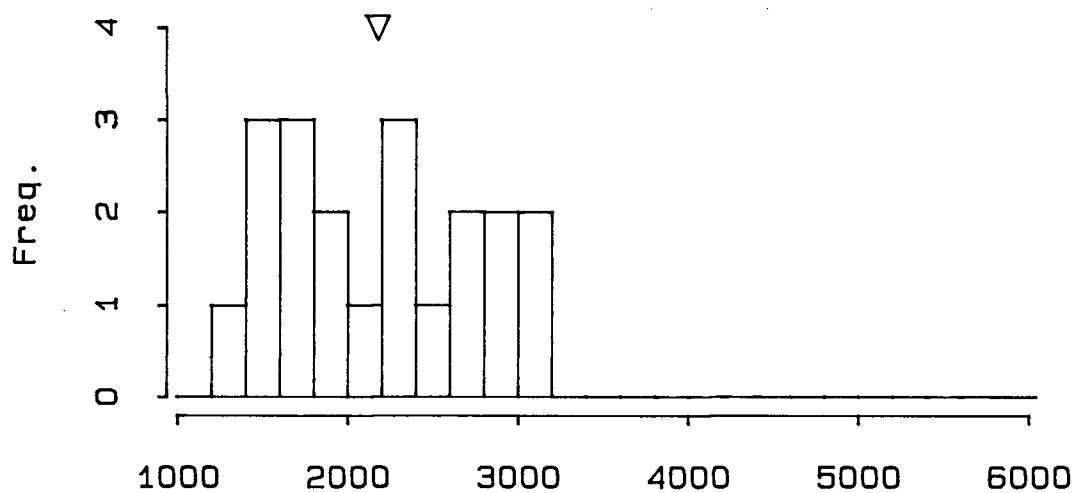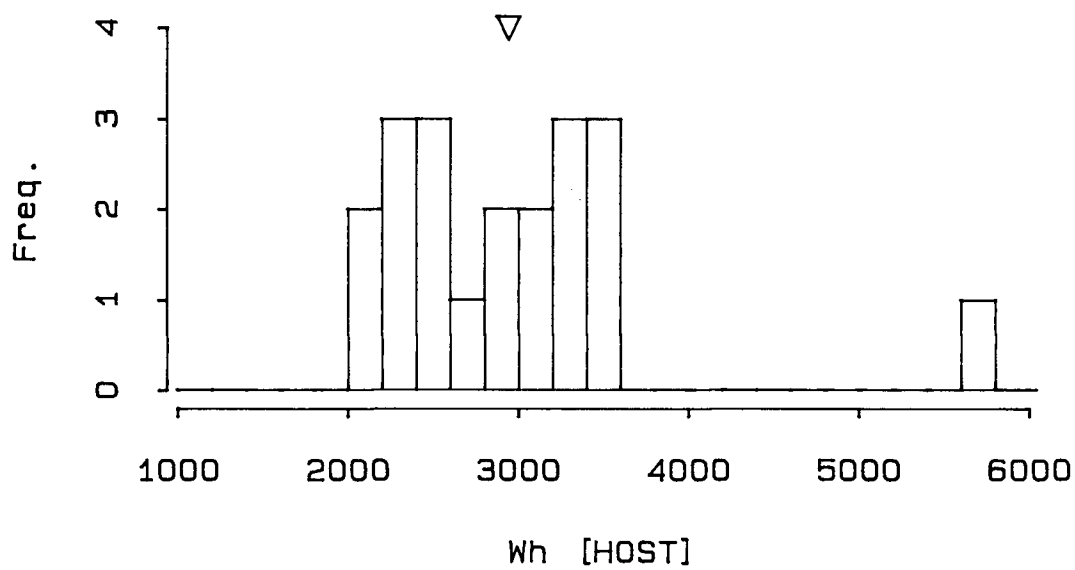
FIGURE 27

Odessa

Trebi

W [HOST]
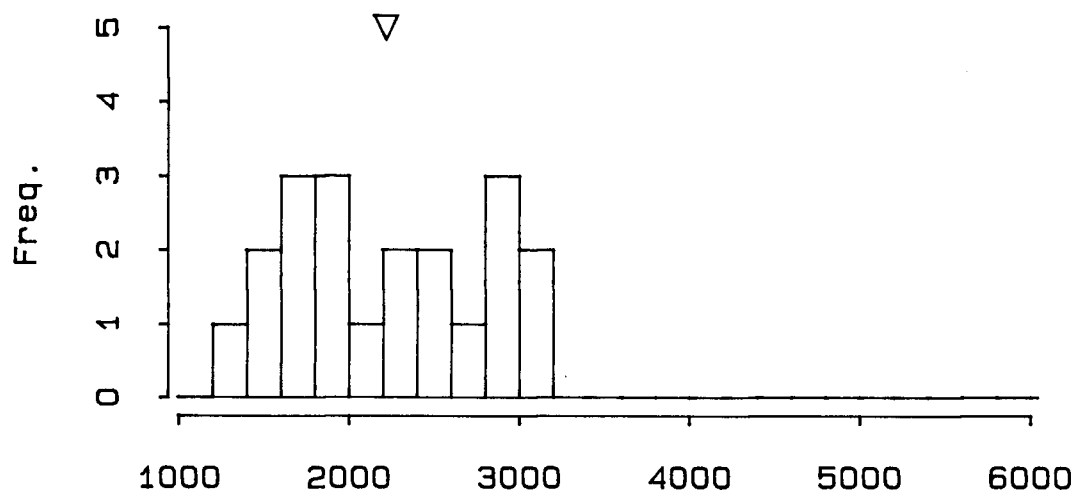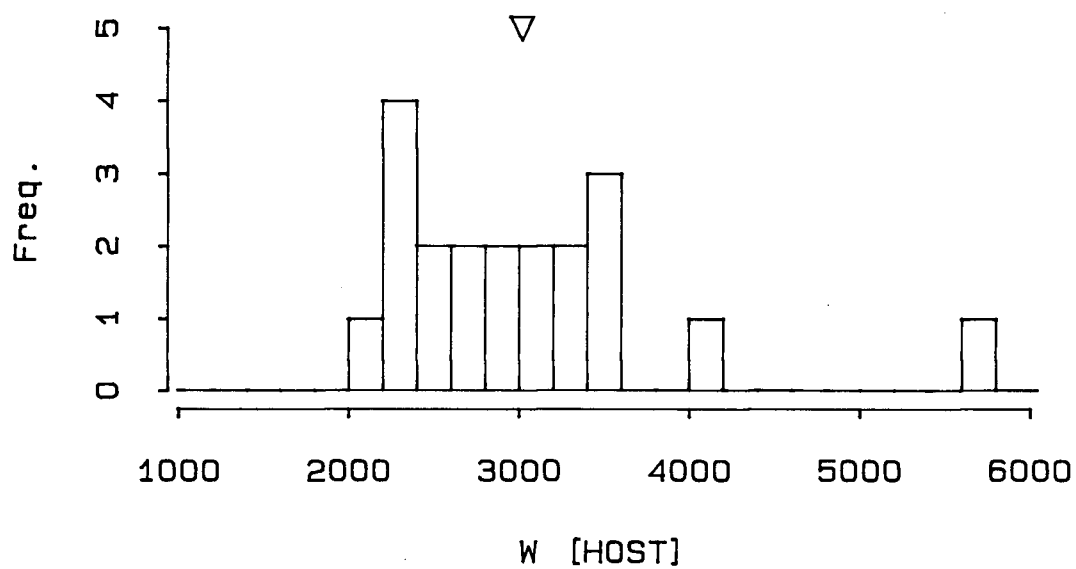
FIGURE 28

Odessa

Trebi

H1
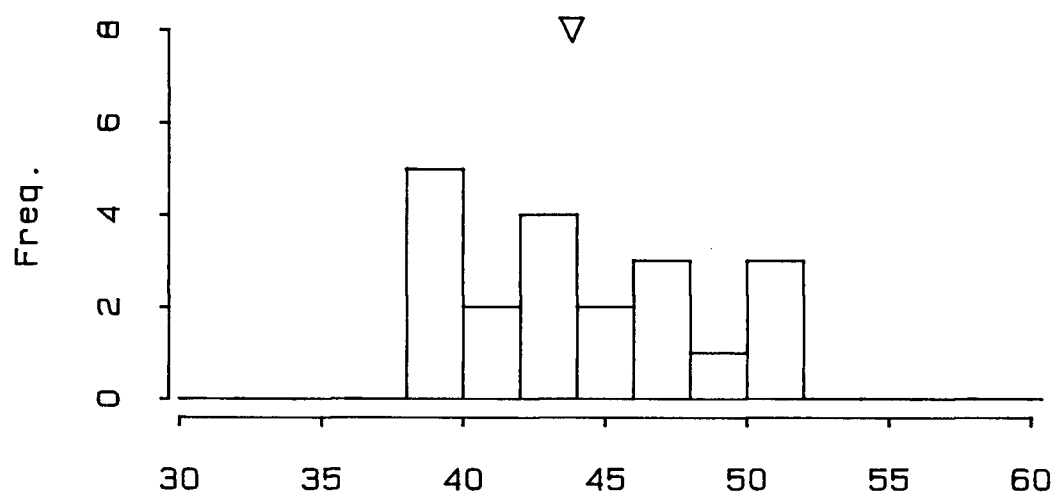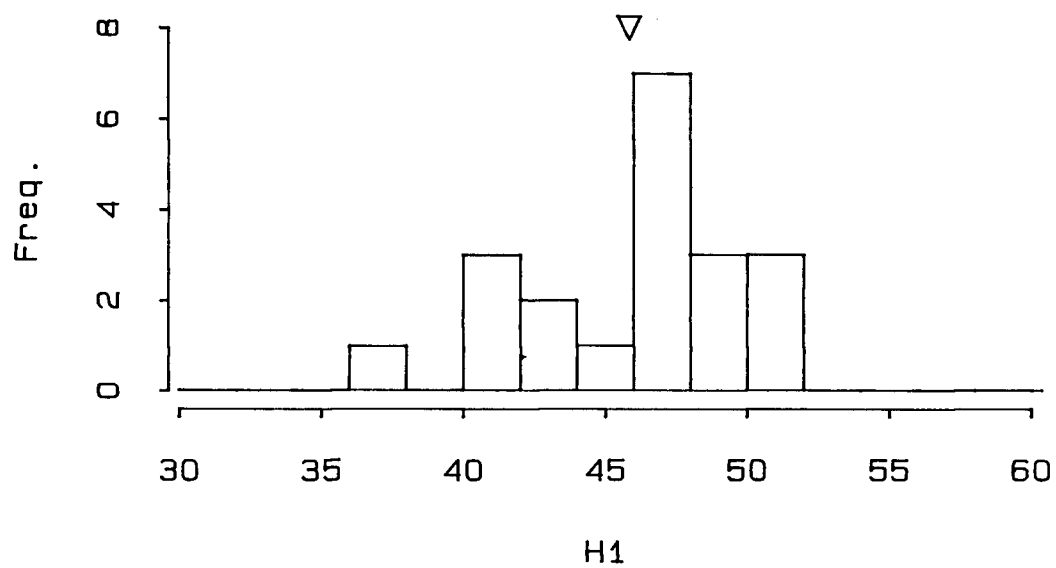
FIGURE 29

Odessa

Trebi

H2
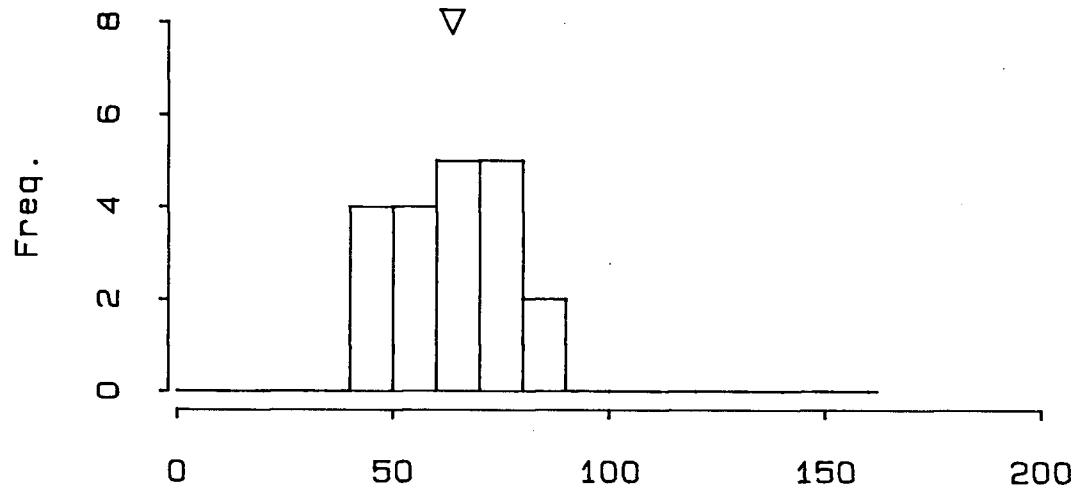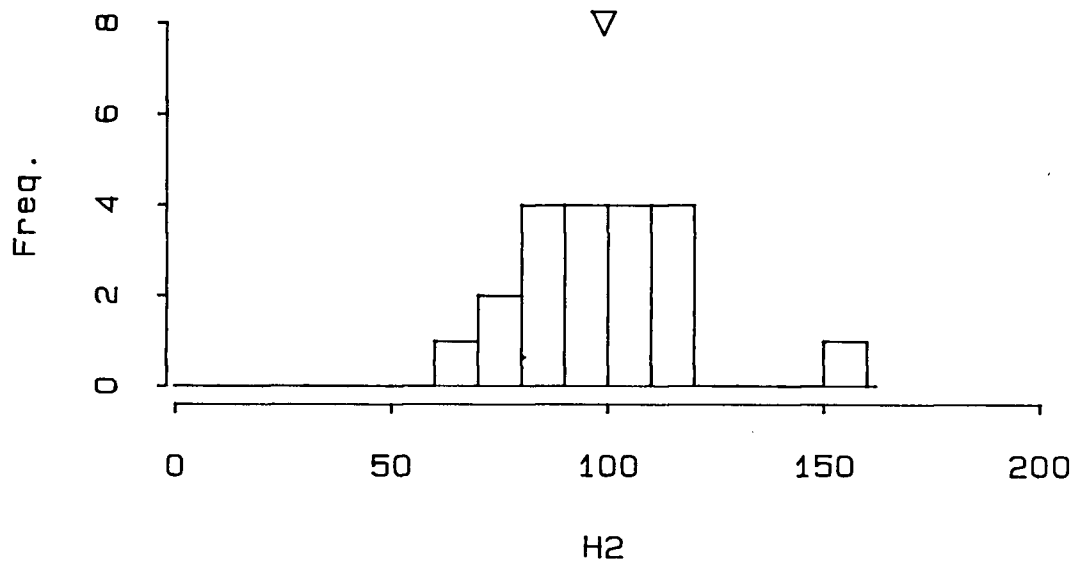
FIGURE 30

Odessa

Trebi

H3
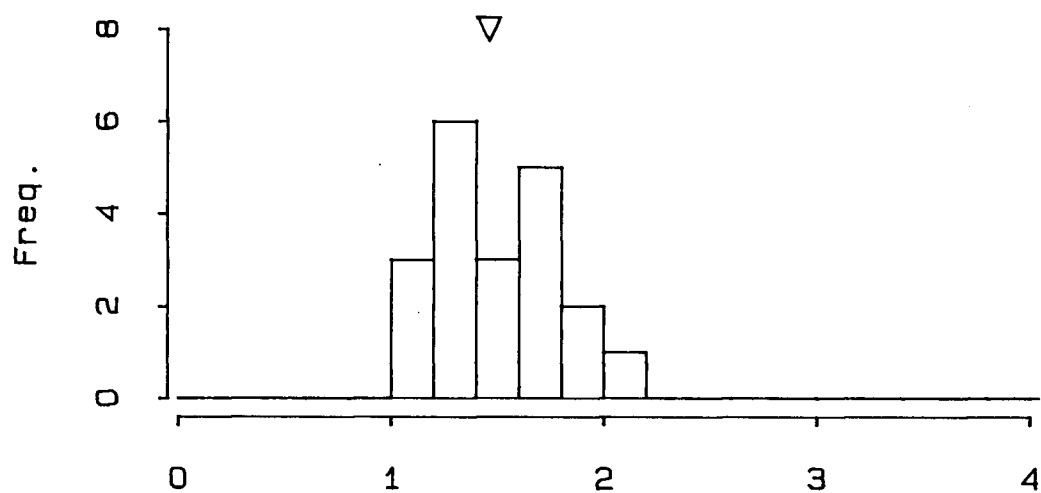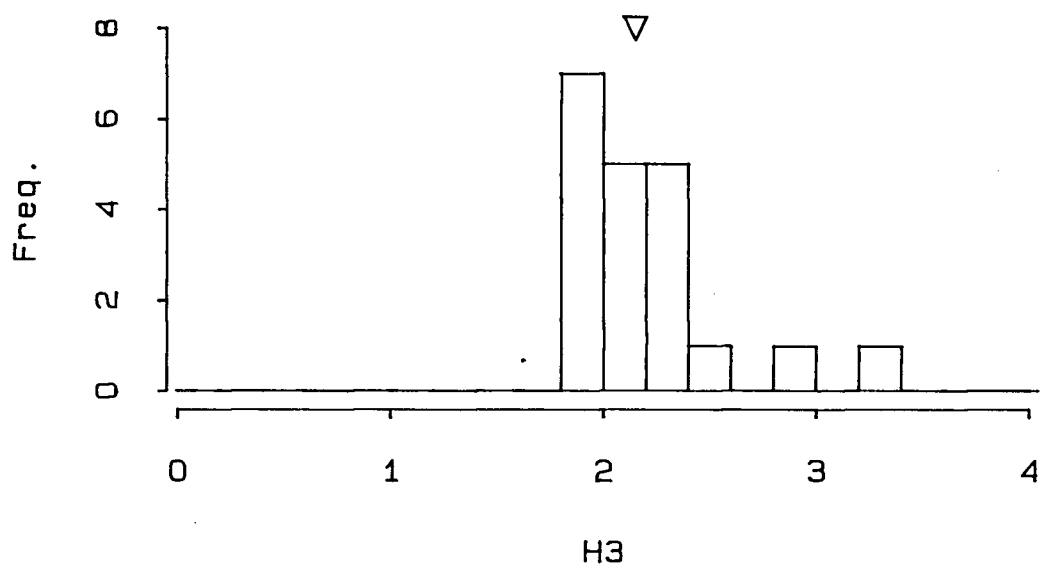
FIGURE 31

Odessa

Trebi

H4
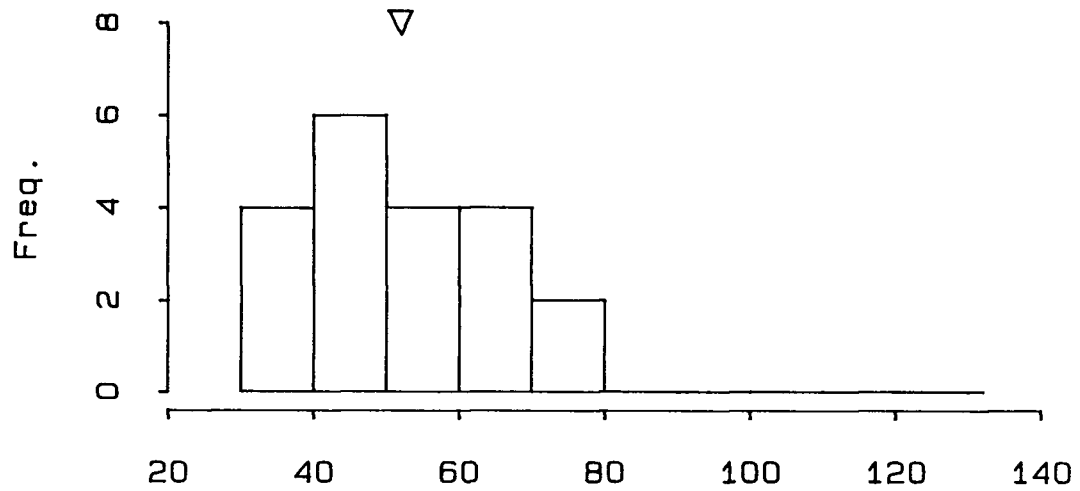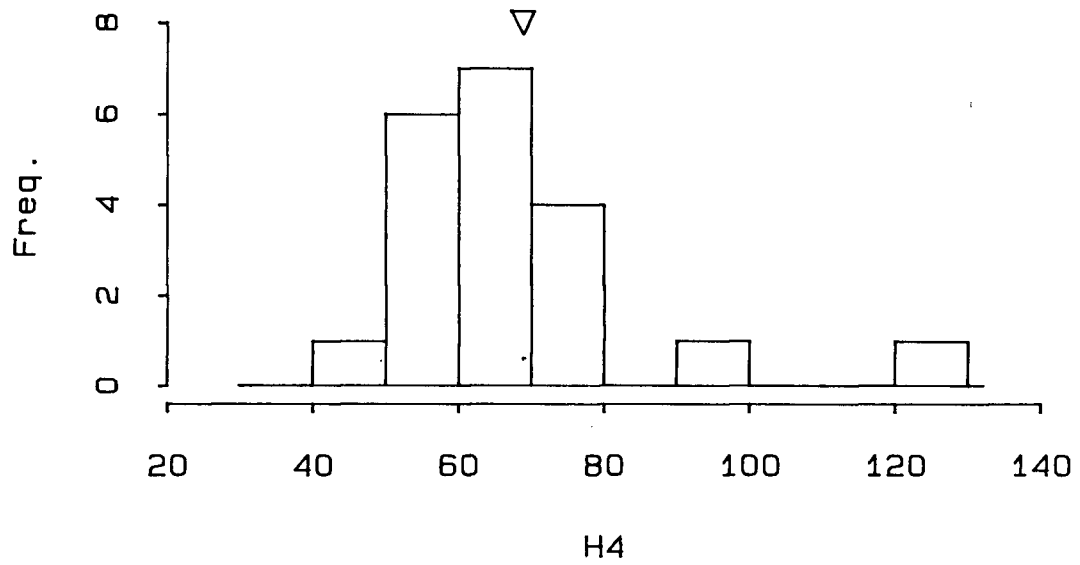
FIGURE 32

Odessa

Trebi

H5
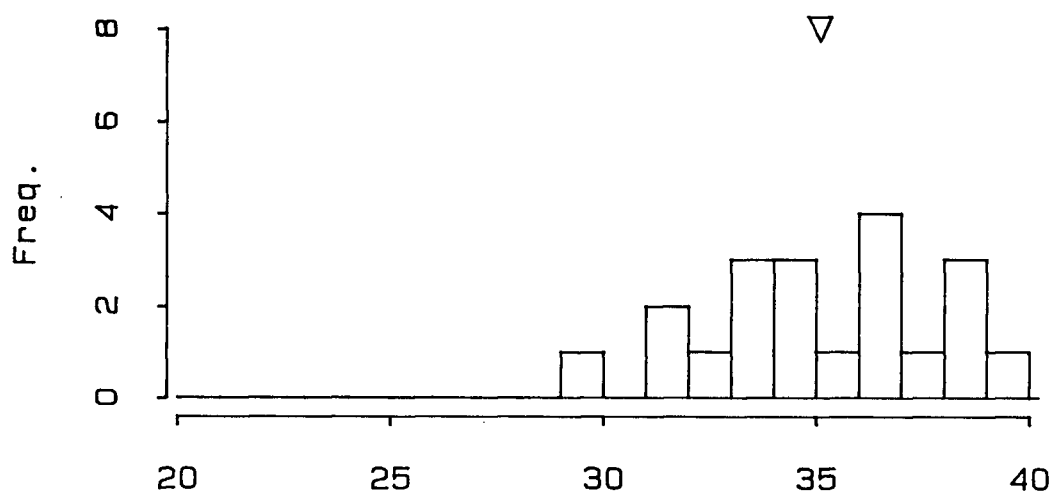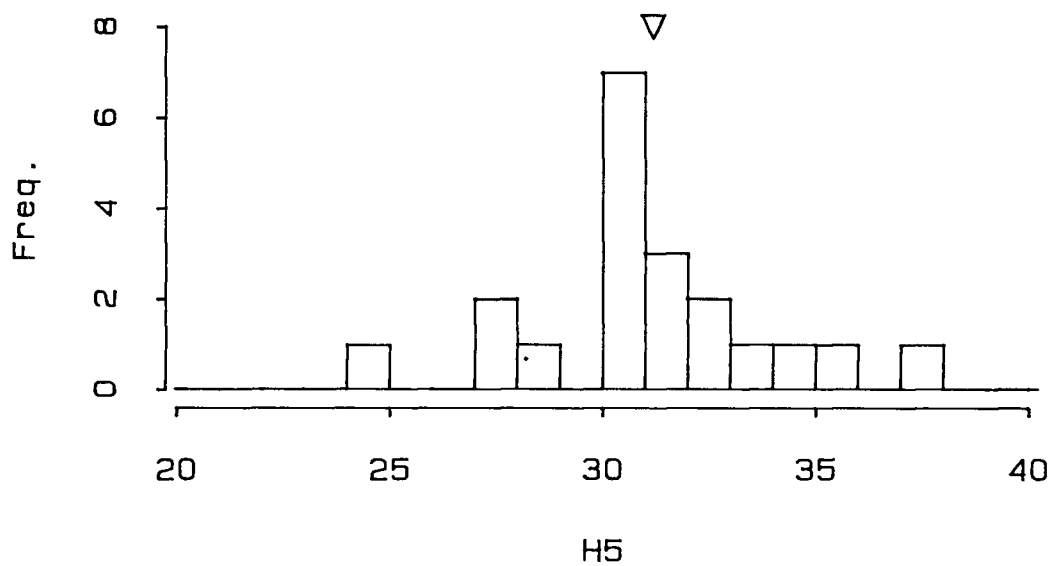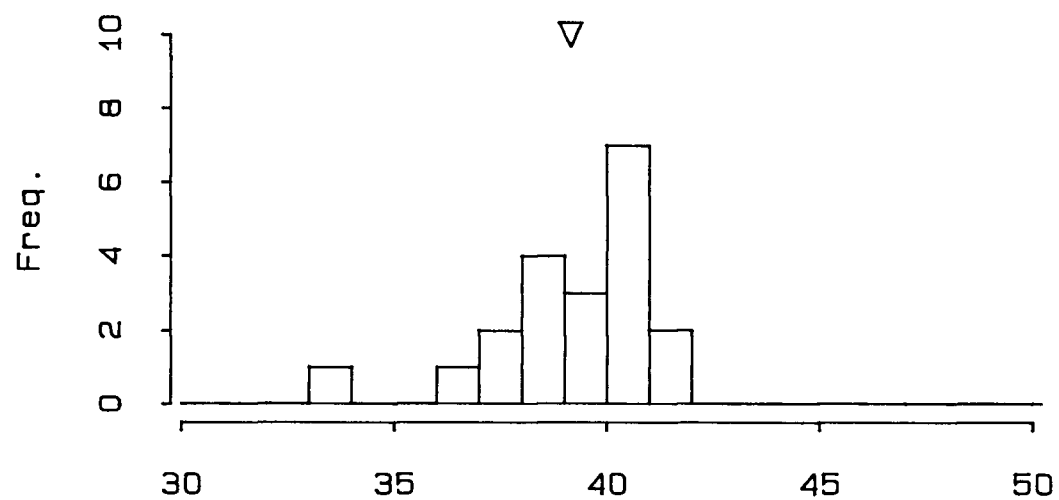
FIGURE 33

Odessa

Trebi
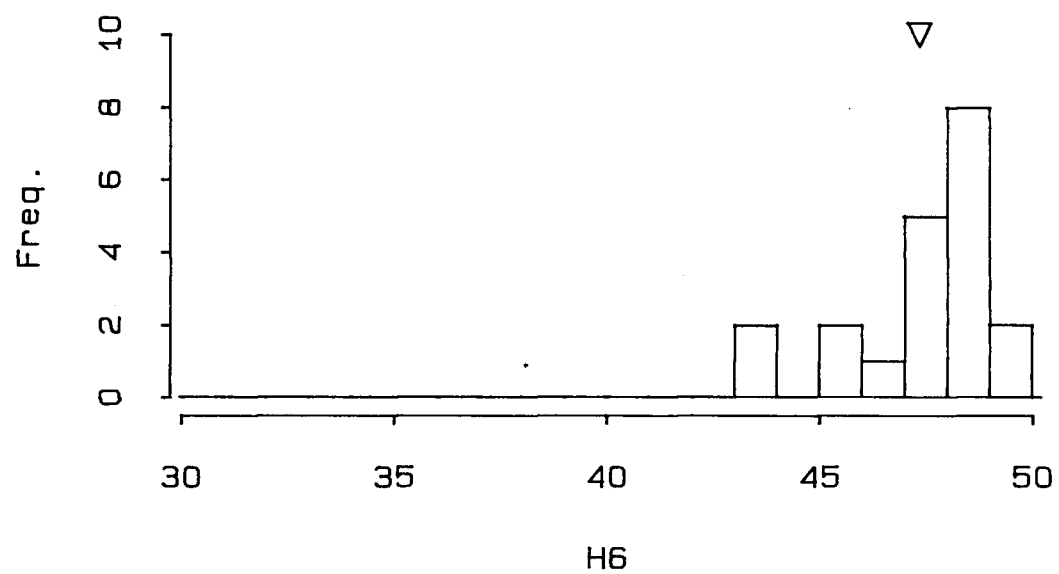
H6

FIGURE   34

Odessa

Trebi

H7
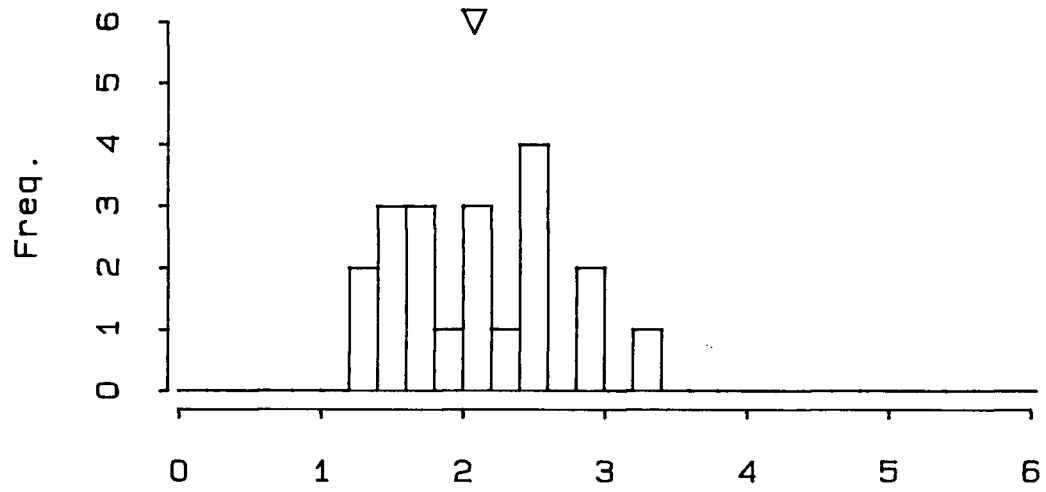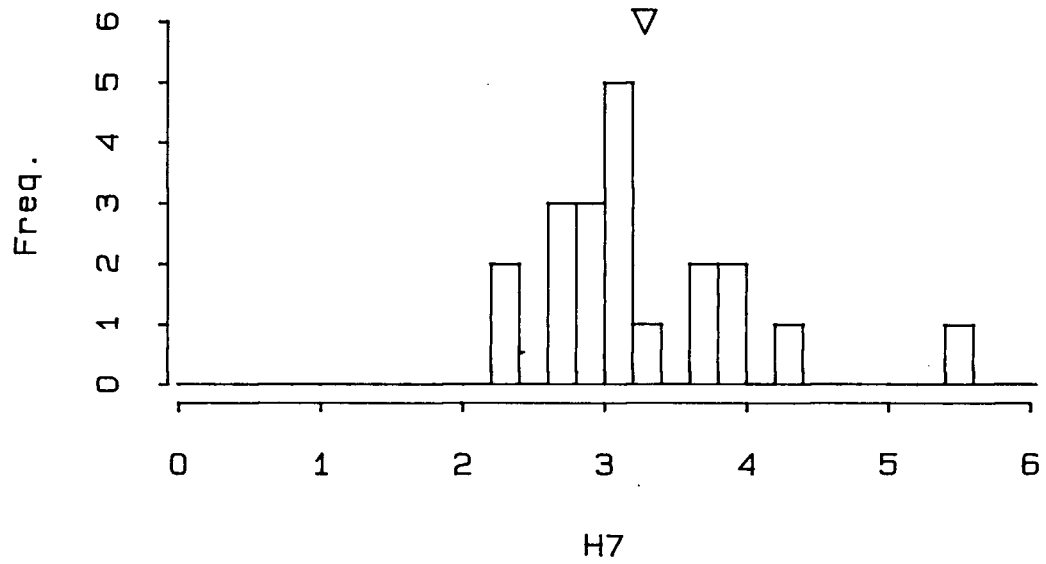
FIGURE 35

Odessa

Trebi

H8
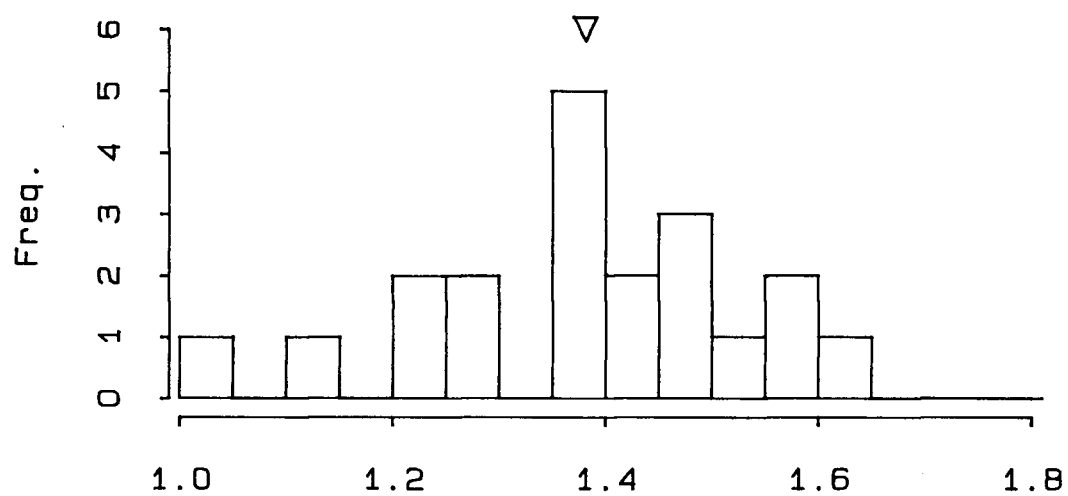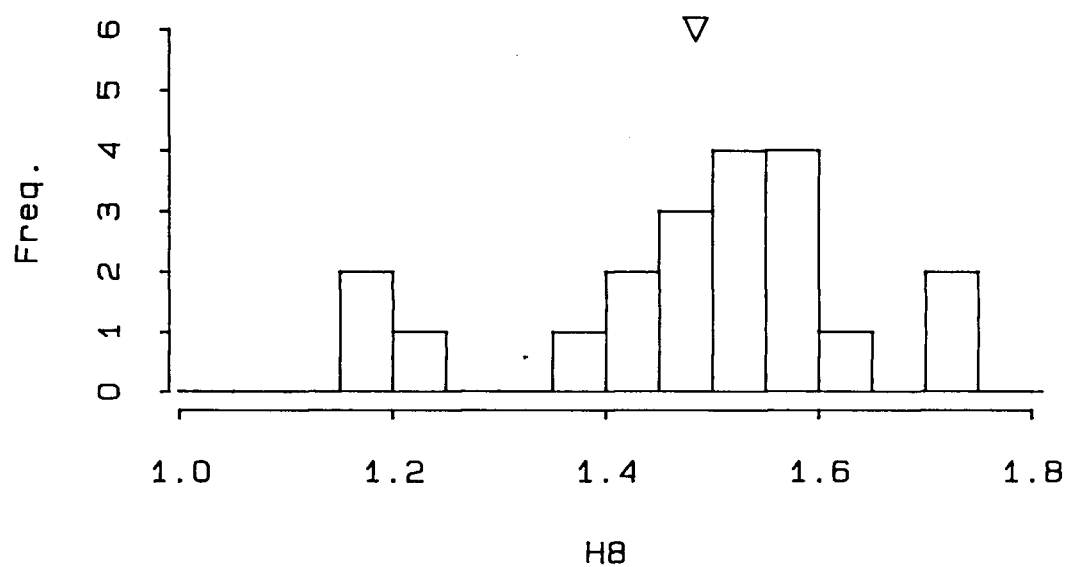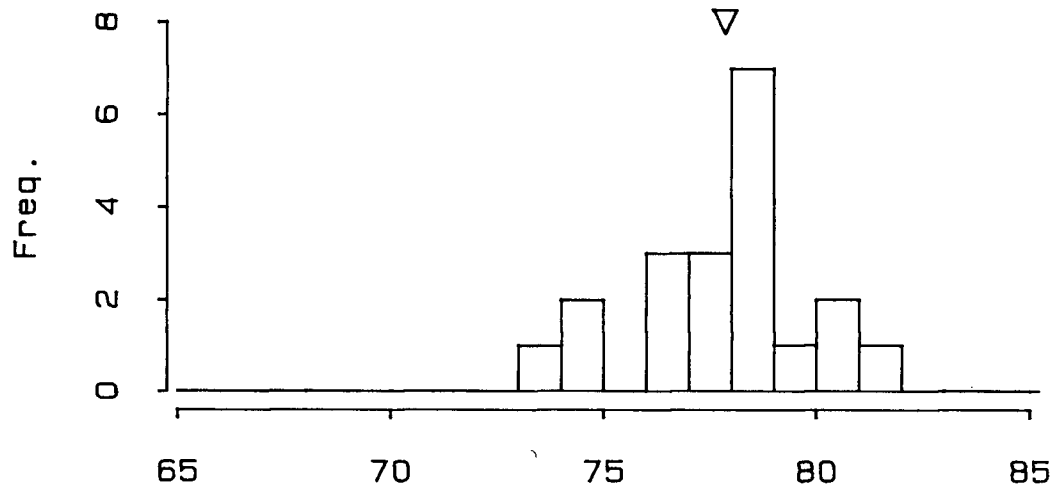
FIGURE 36

Odessa

Trebi
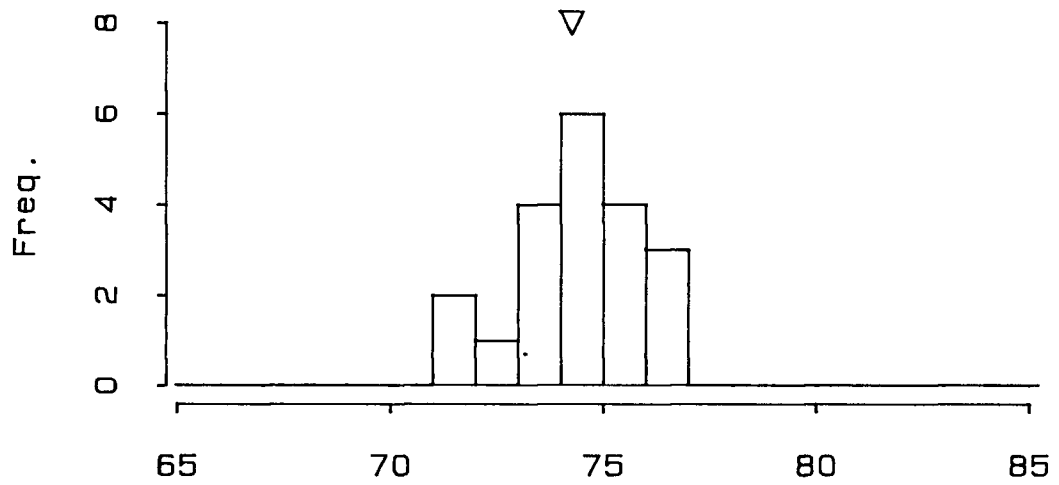
H9t

FIGURE 37

Odessa
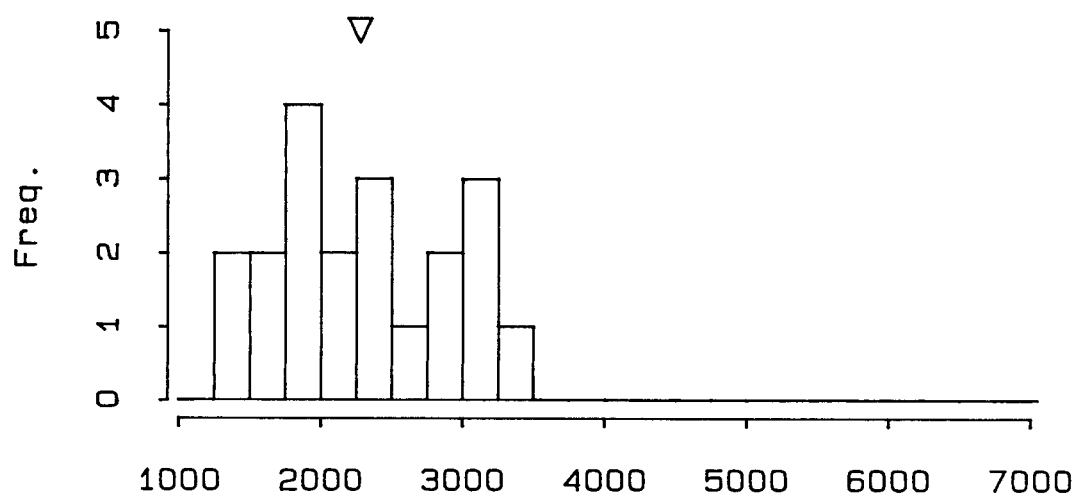
Trebi
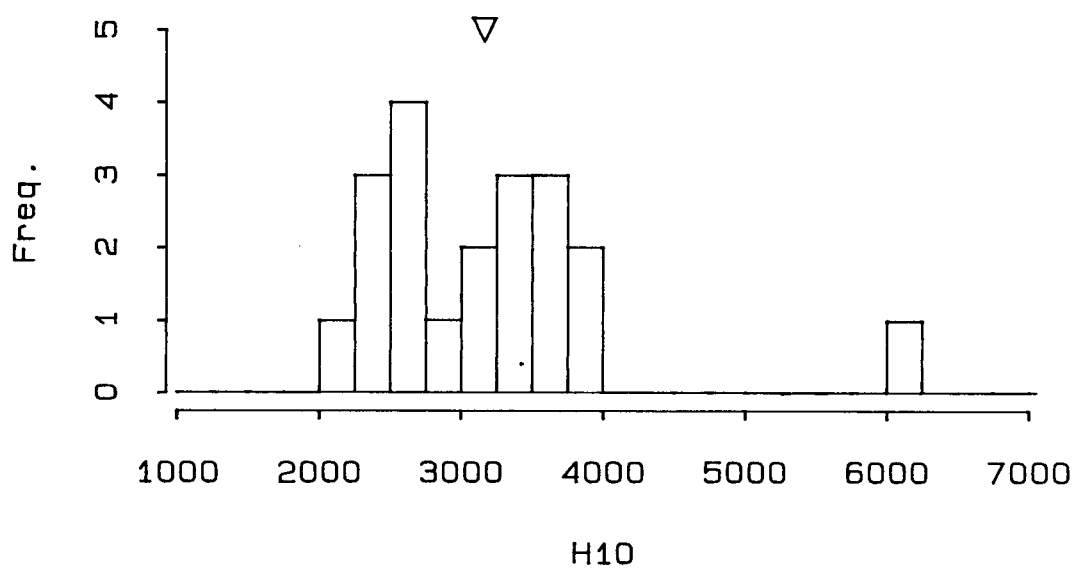
H10

FIGURE 38

Odessa

Trebi

C1
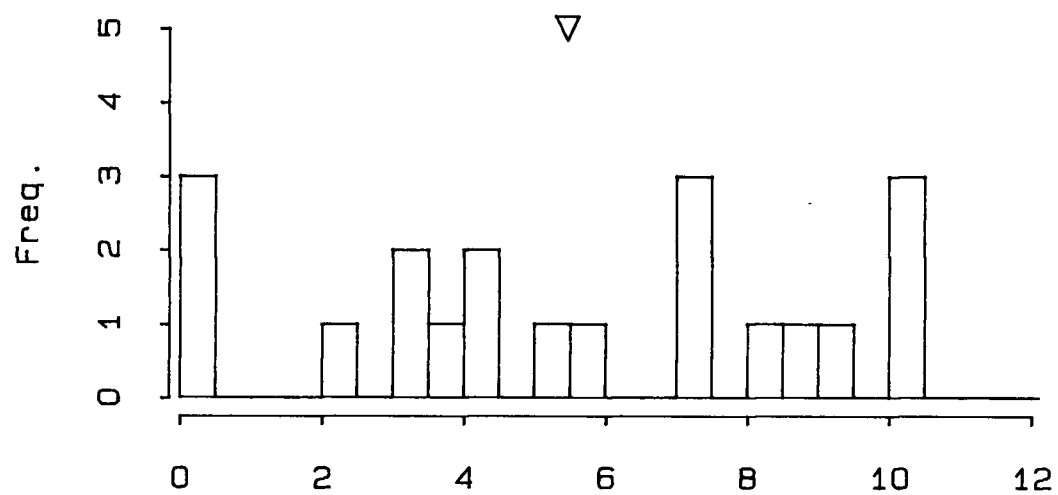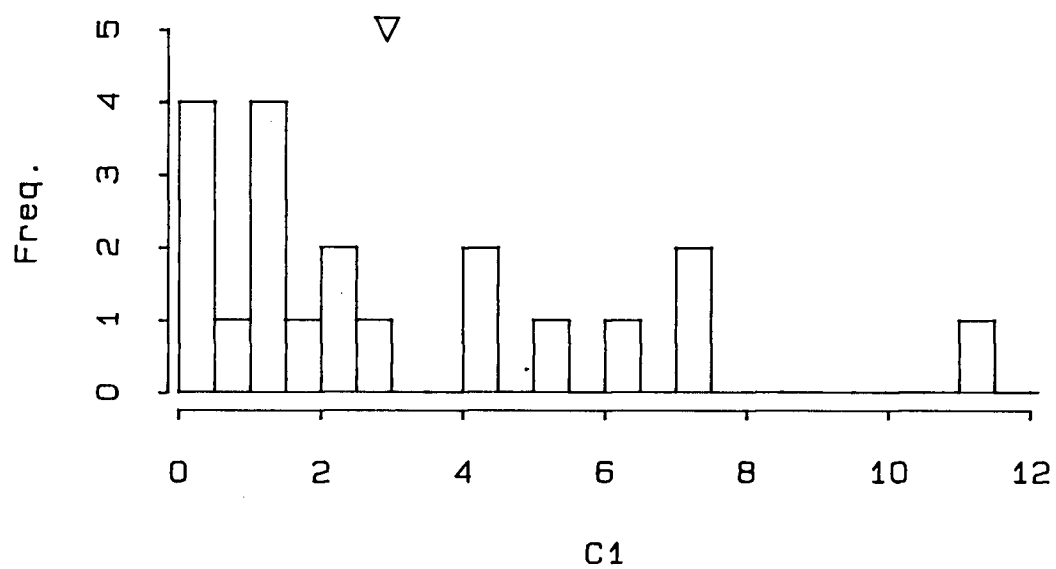
FIGURE 39

Odessa

Trebi

C2
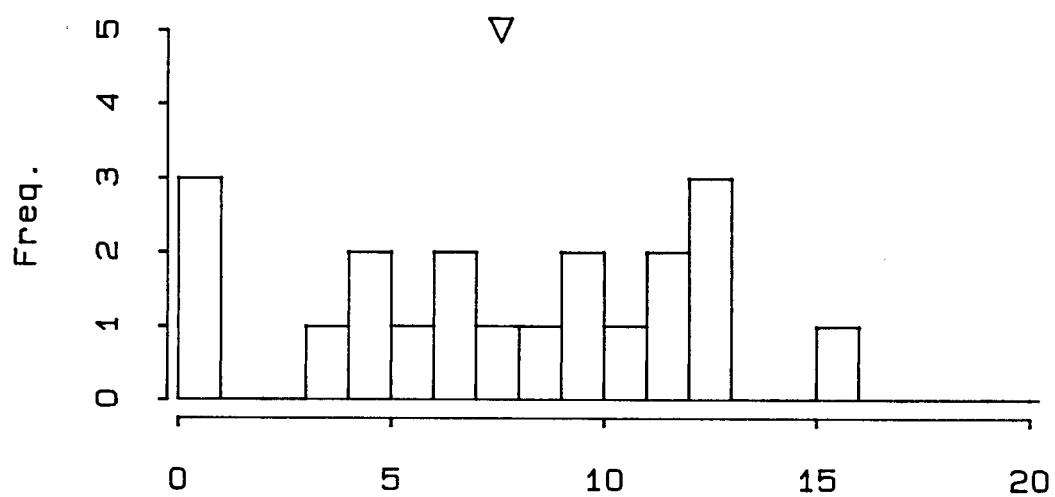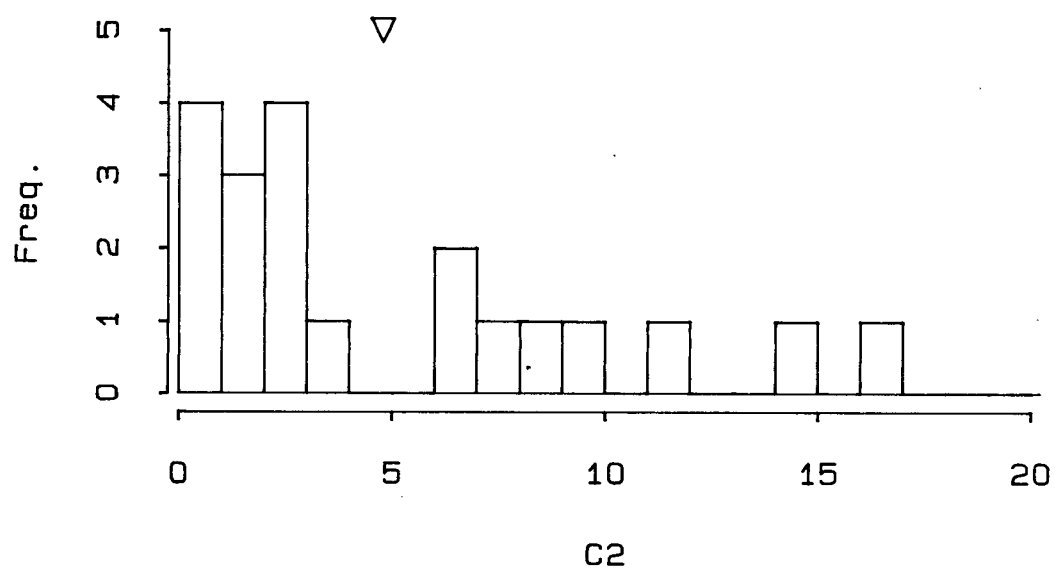
FIGURE 40

Odessa

Trebi

C3
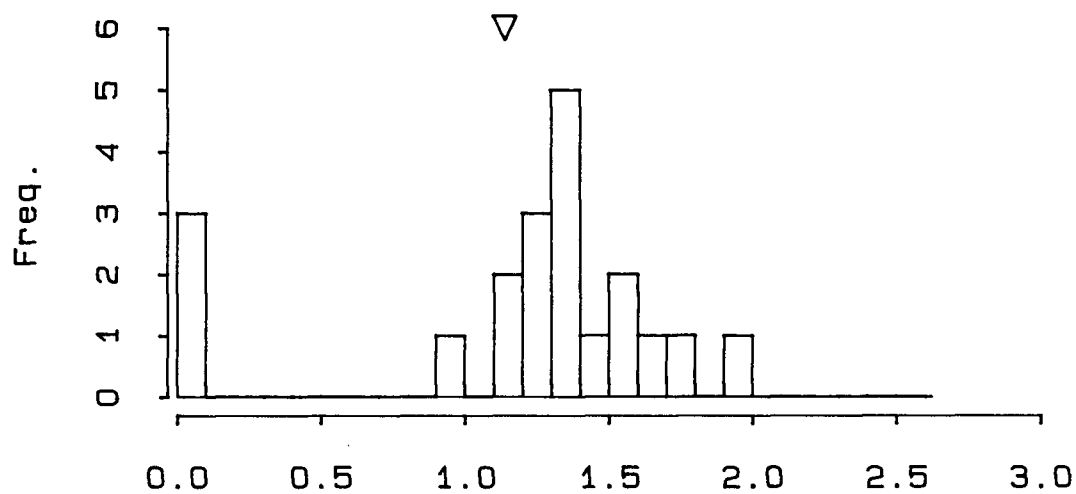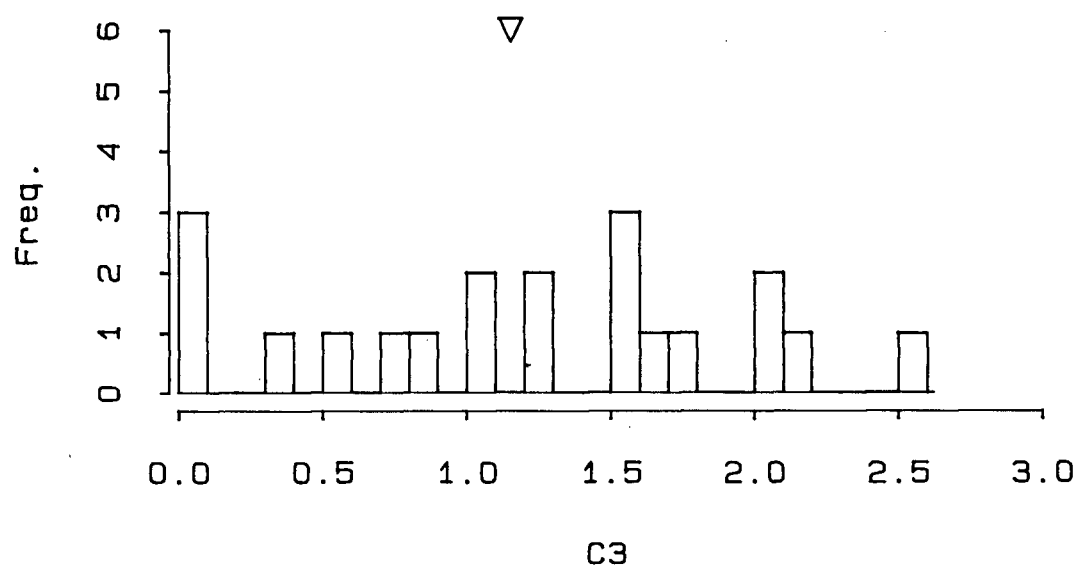
FIGURE   41

Odessa

Trebi

C4
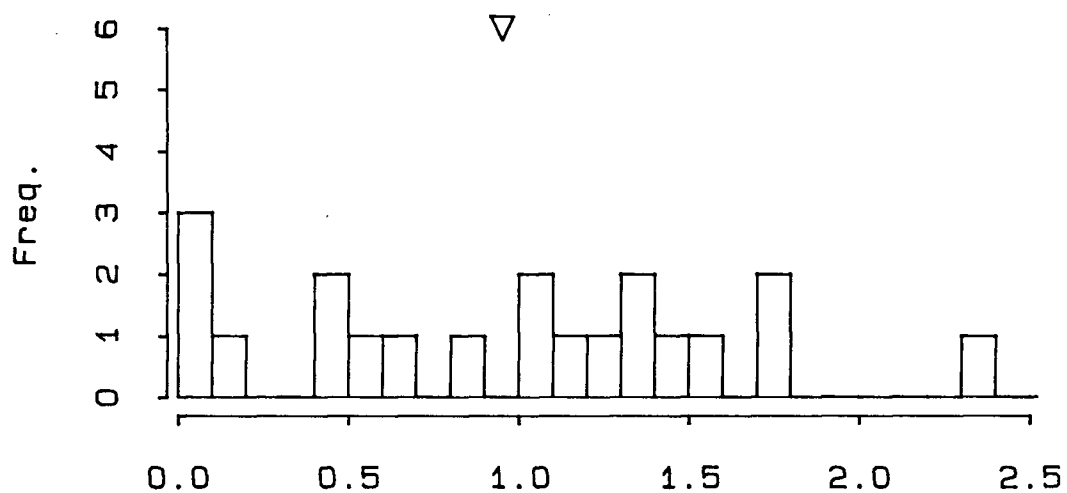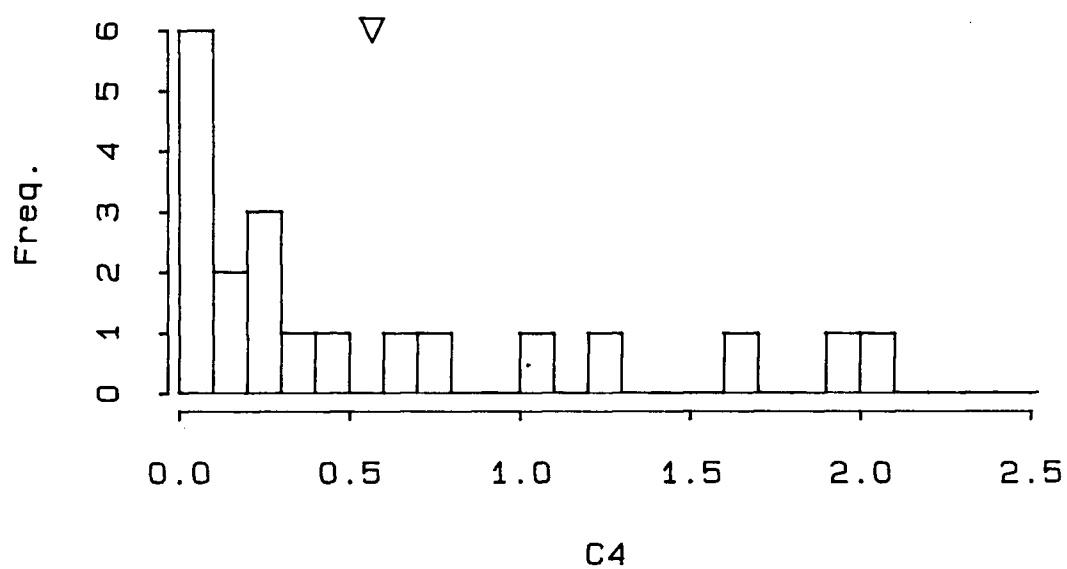
FIGURE 42

Odessa

Trebi

C5
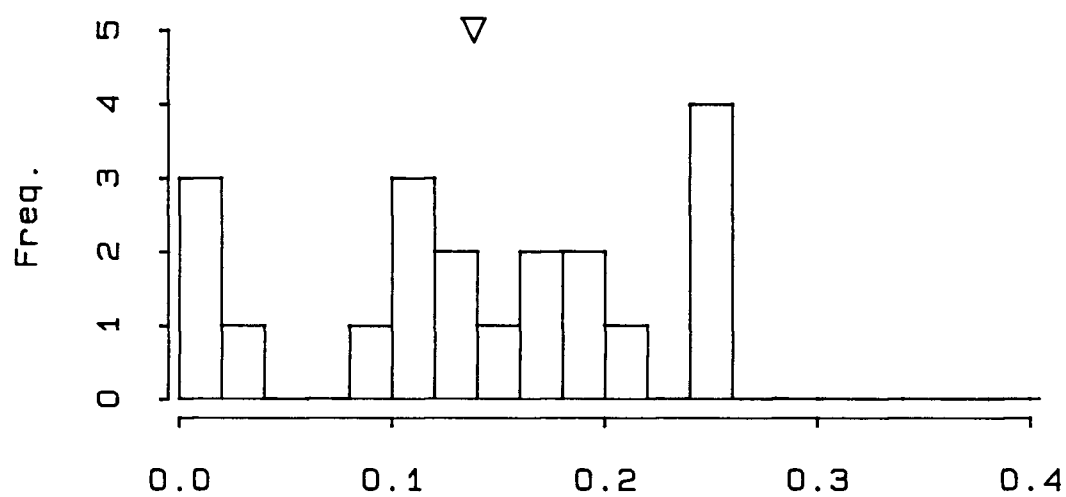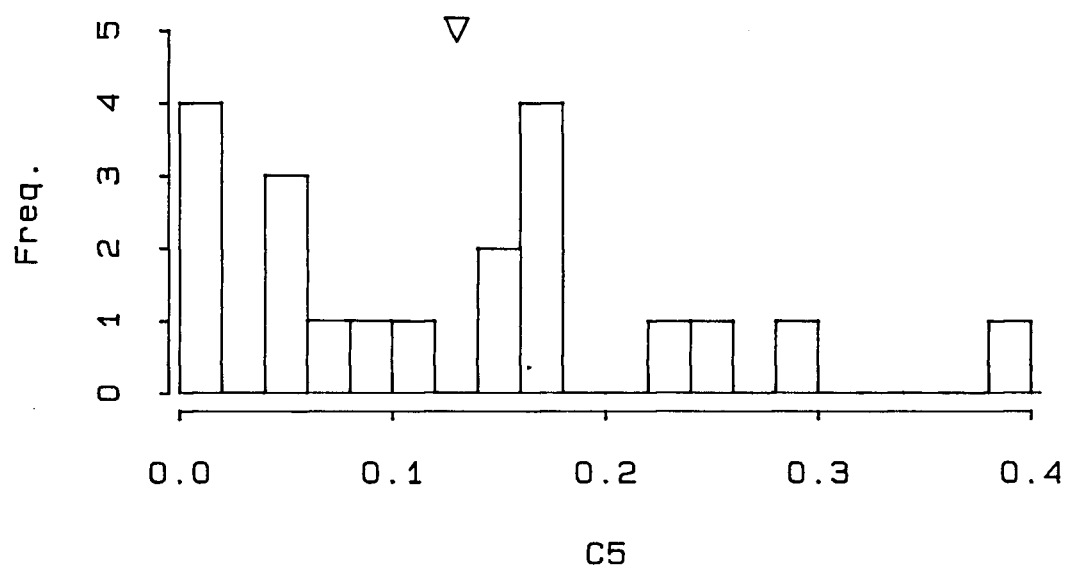
FIGURE 43

Odessa

Trebi

C6
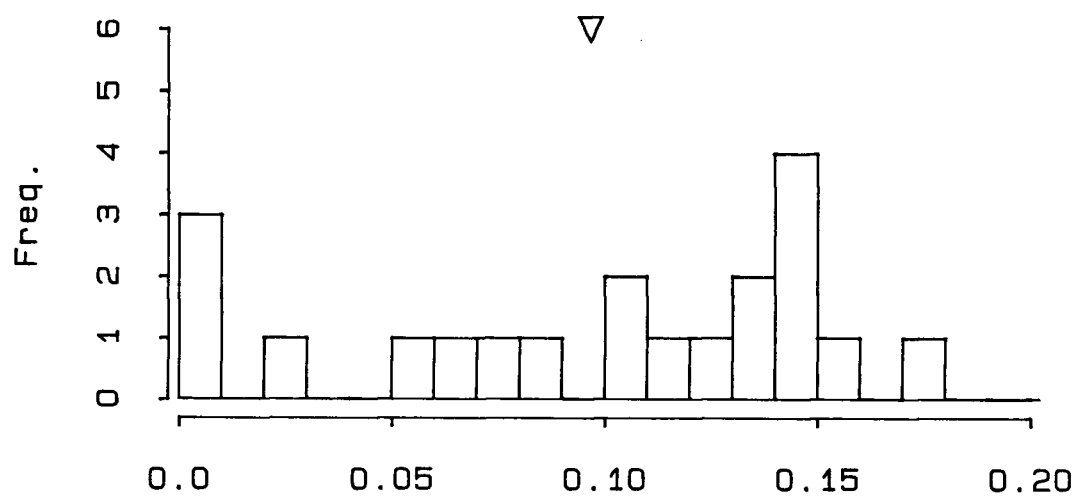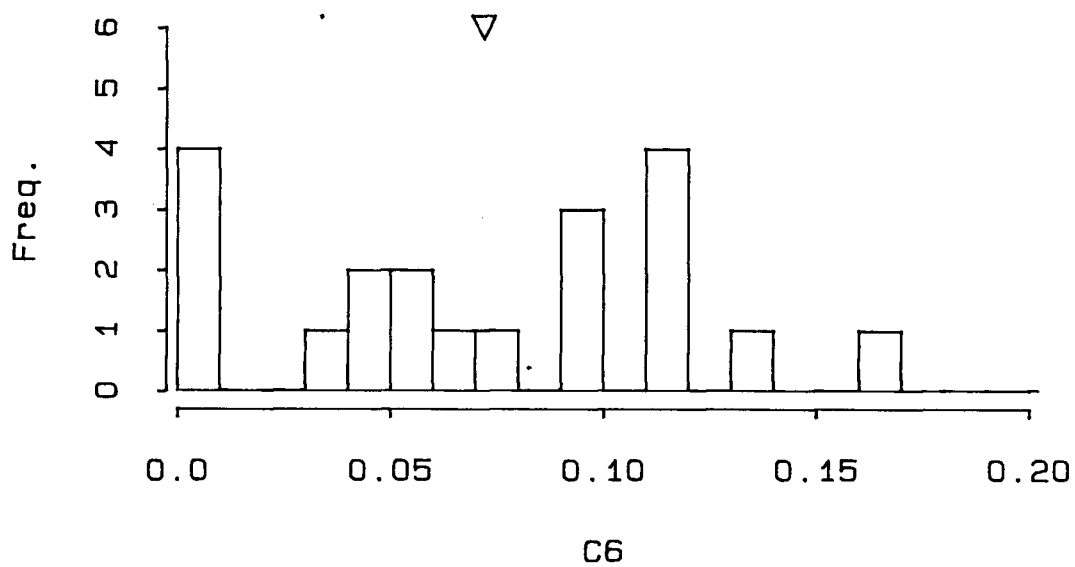
FIGURE 44

Odessa

Trebi

C7t
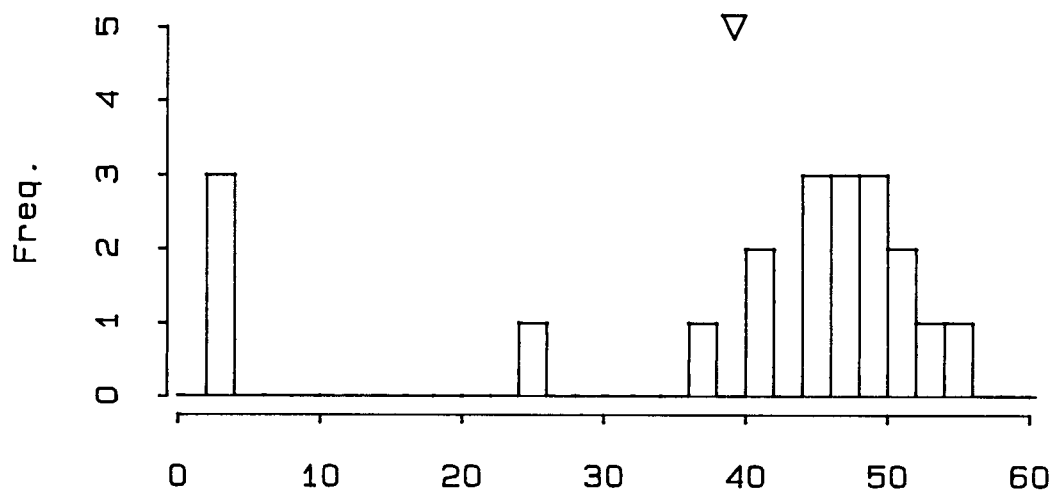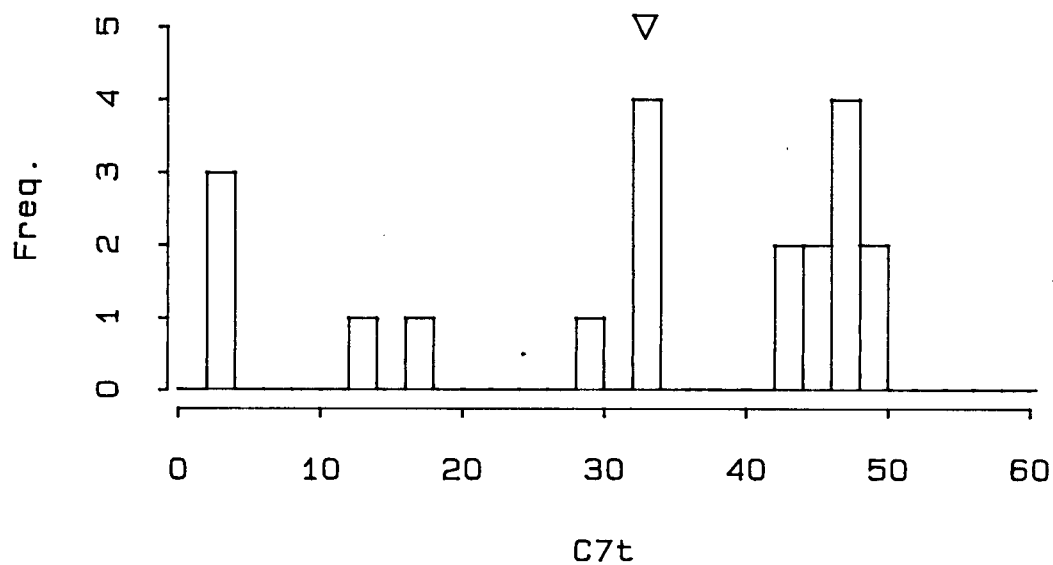
FIGURE 45

Odessa

Trebi

P1
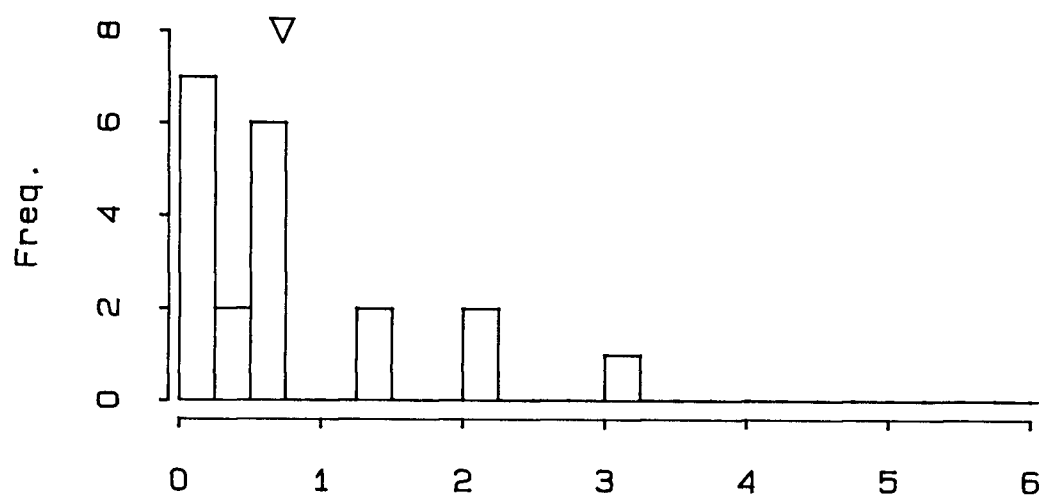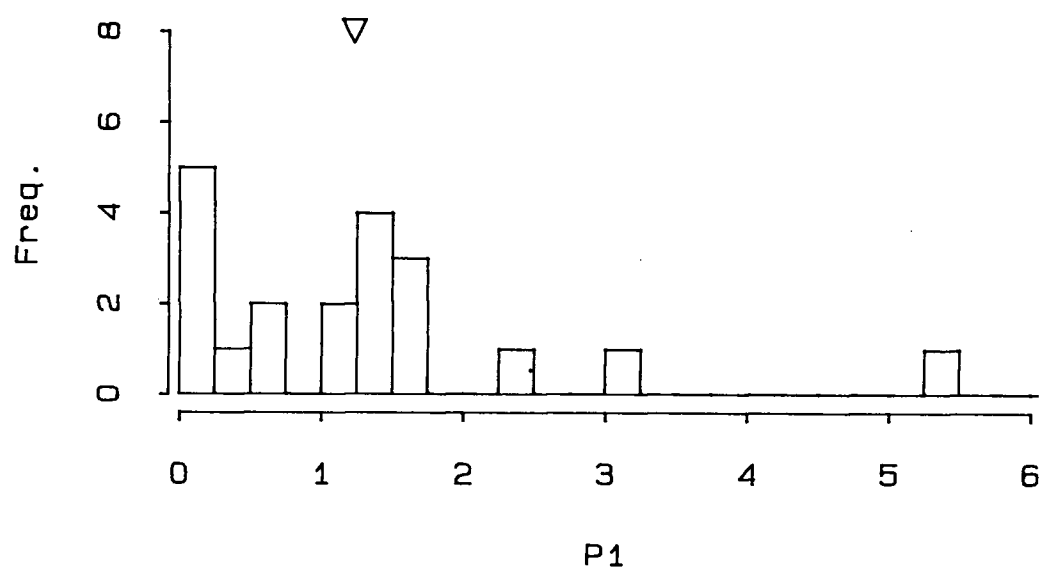
FIGURE 46

Odessa

Trebi

P2
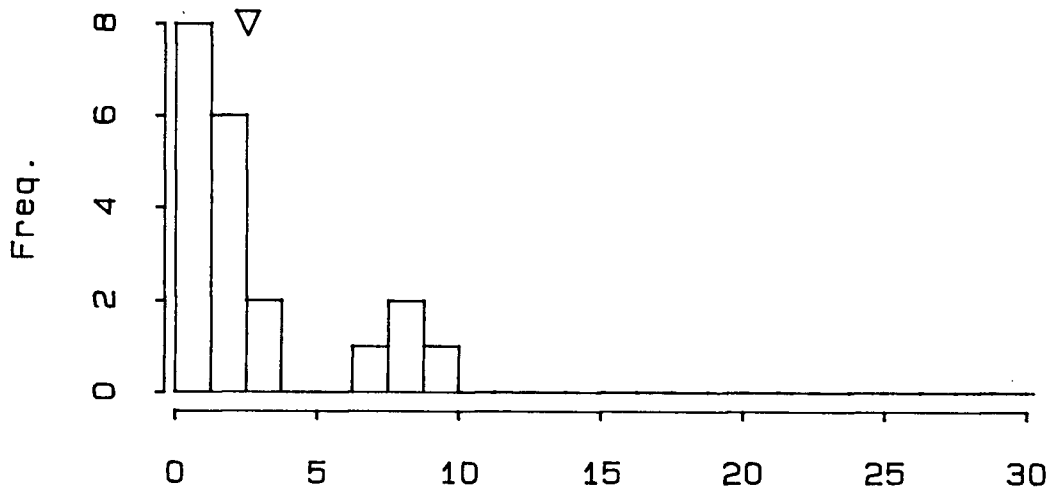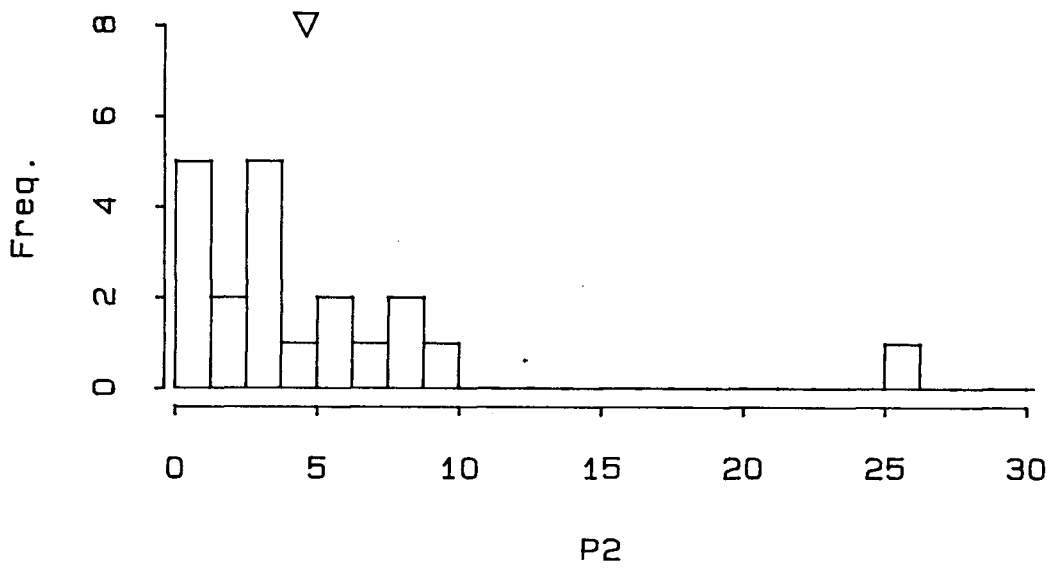
FIGURE 47

Odessa

Trebi

P3
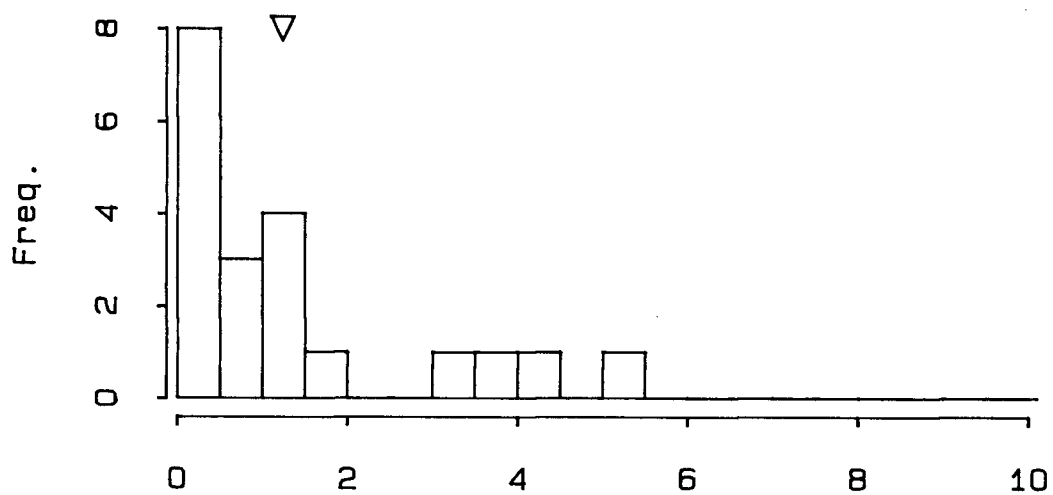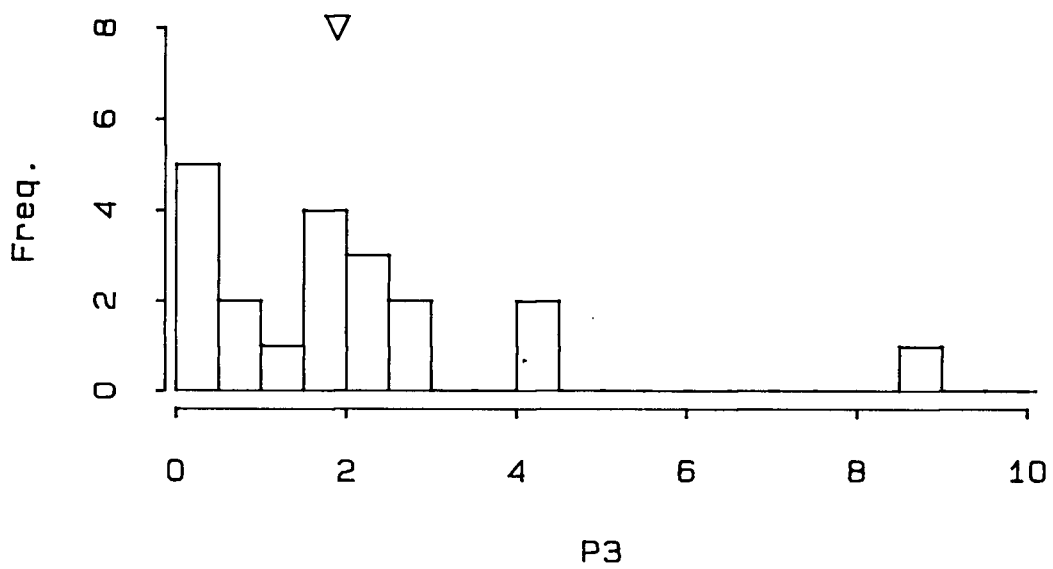
FIGURE 48

Odessa

Trebi

P4
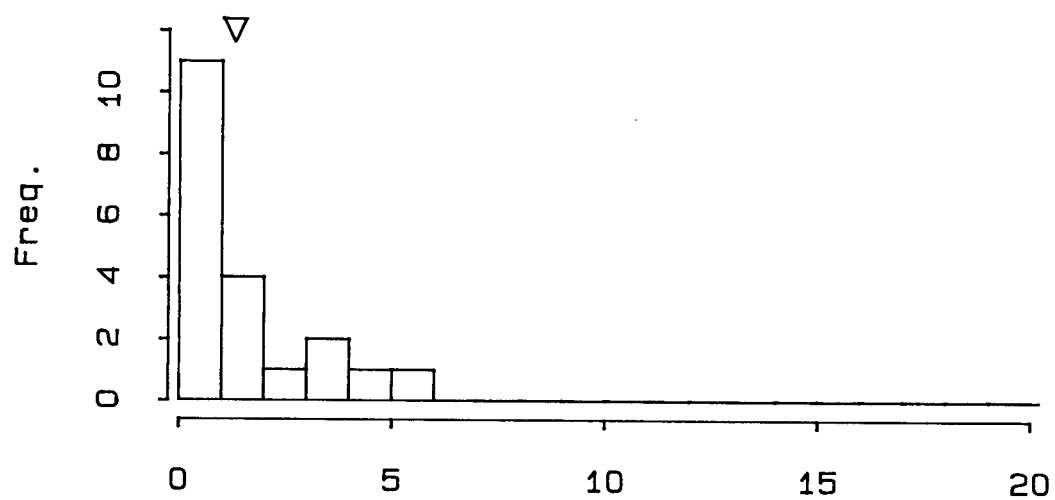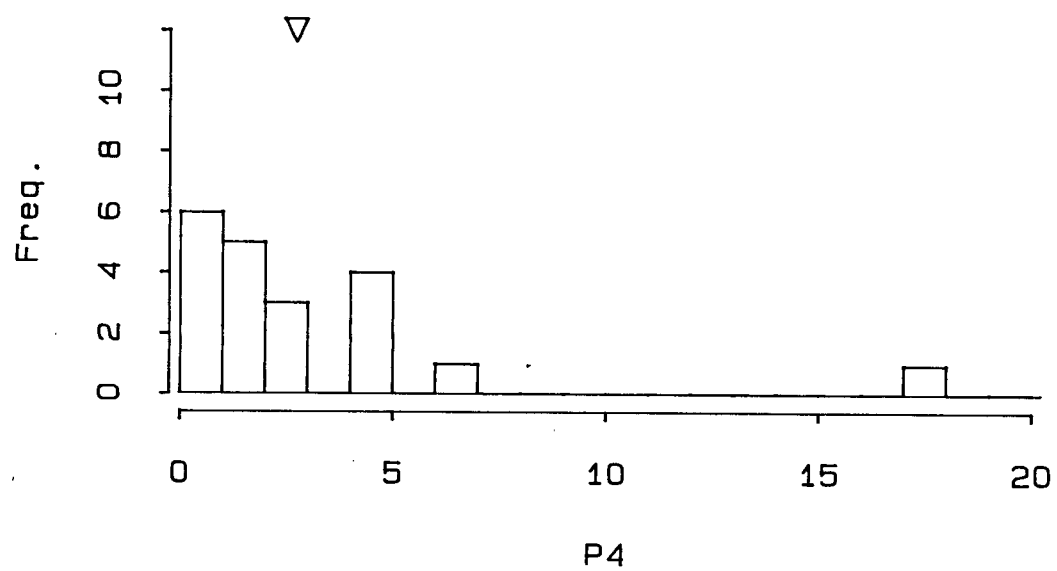
FIGURE   49

Odessa

Trebi

P5
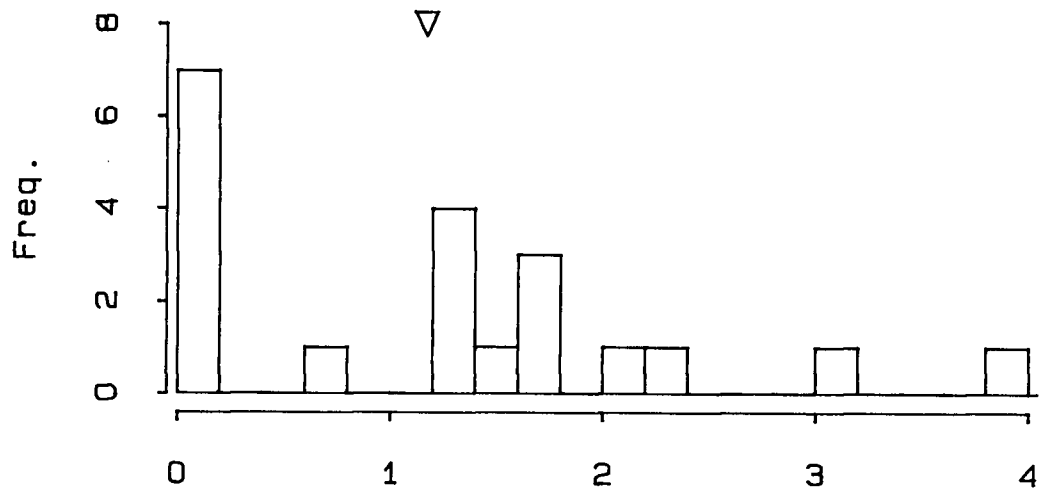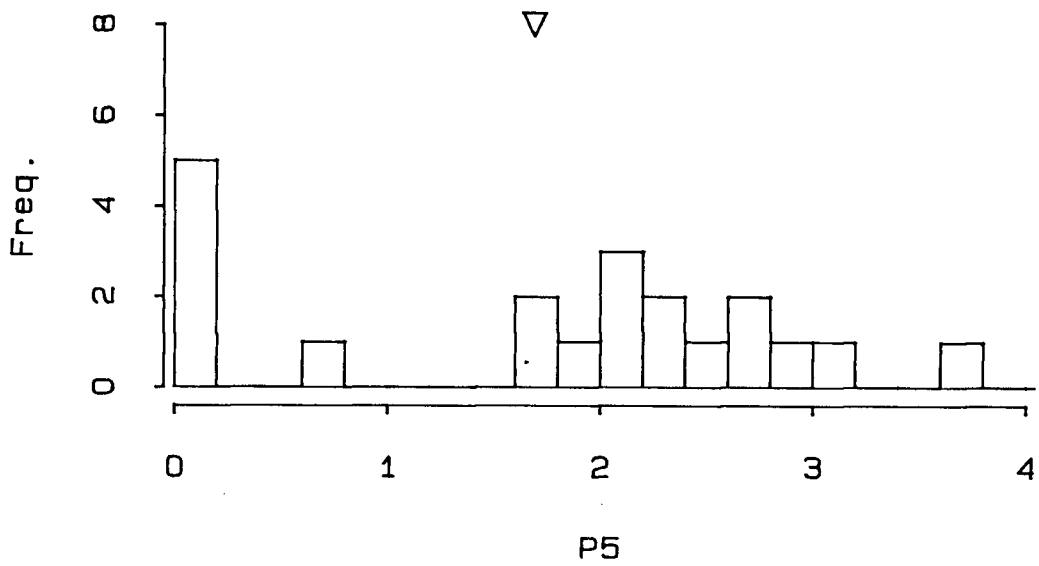
FIGURE 50

Odessa

Trebi

P6
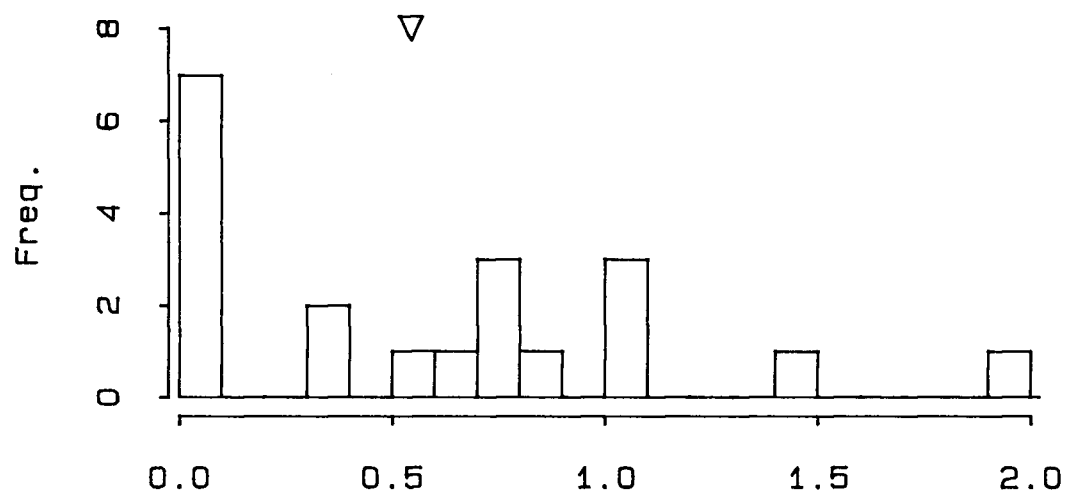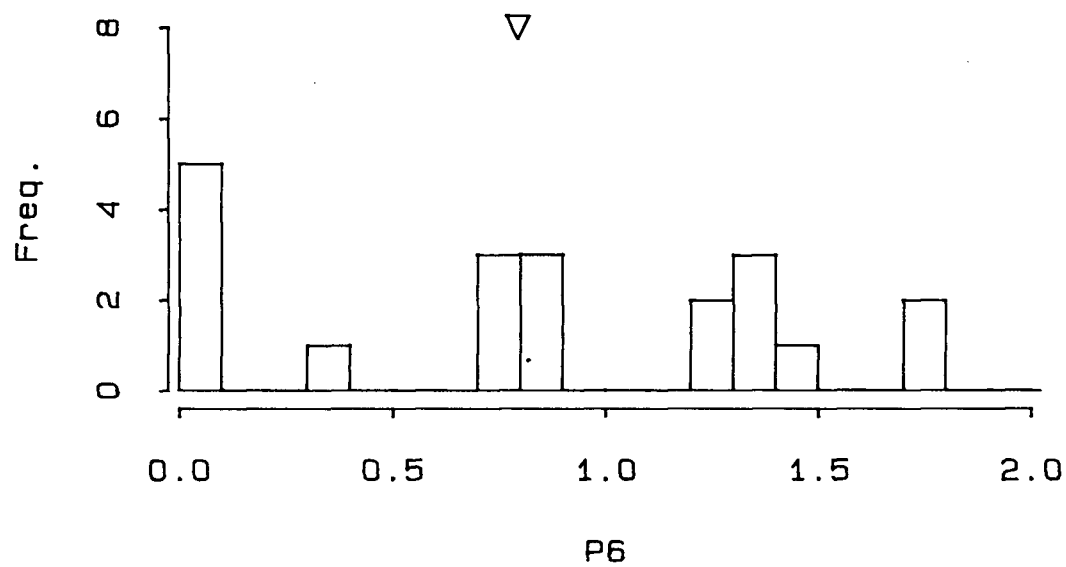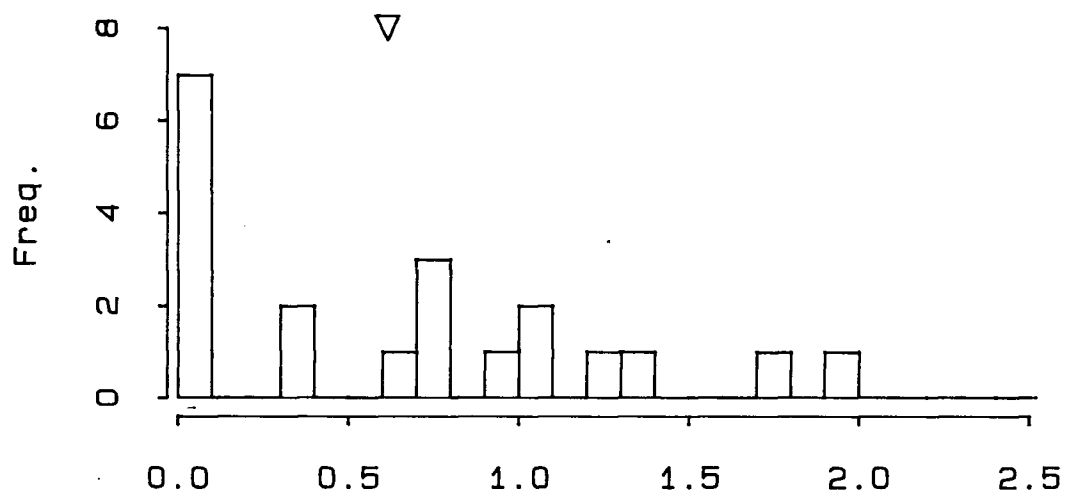
FIGURE 51

Odessa

Trebi
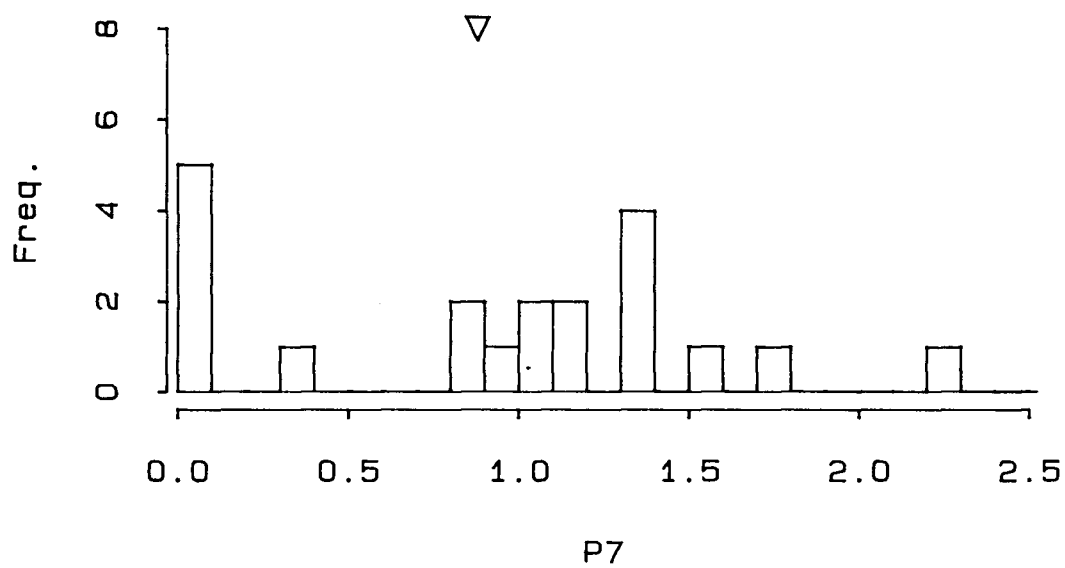
P7

FIGURE 52

Odessa

Trebi

P8
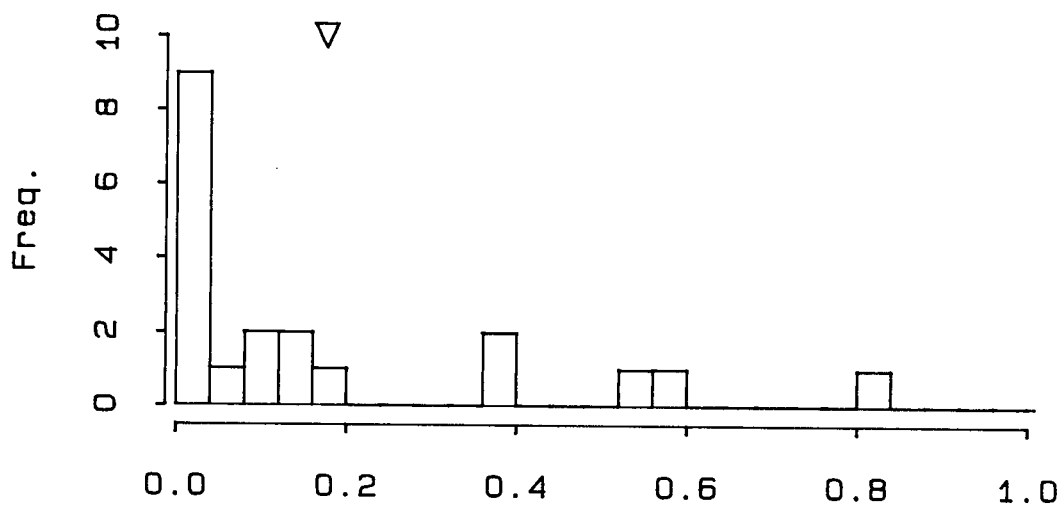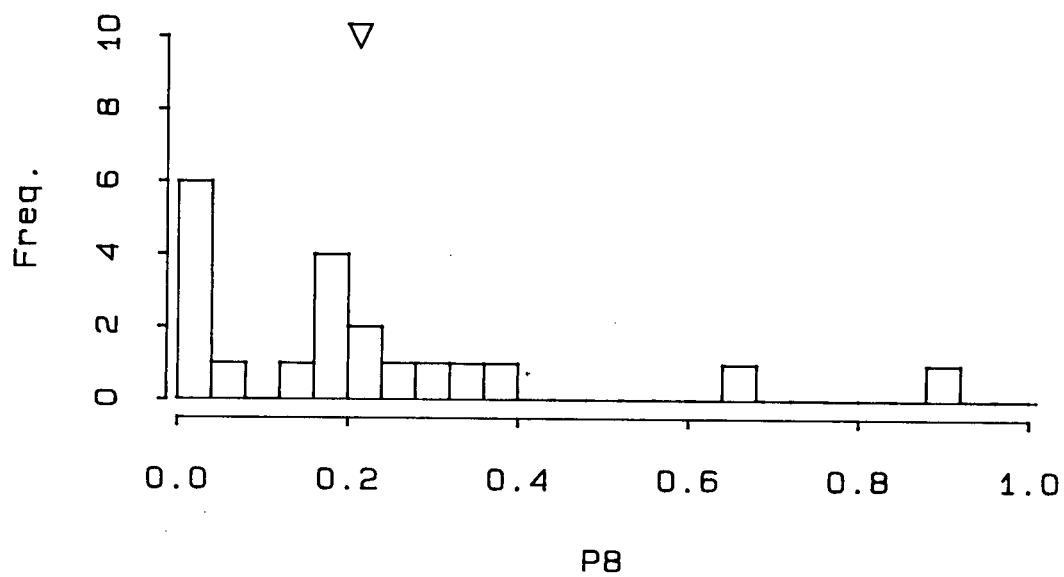
FIGURE 53

Odessa

Trebi

P9
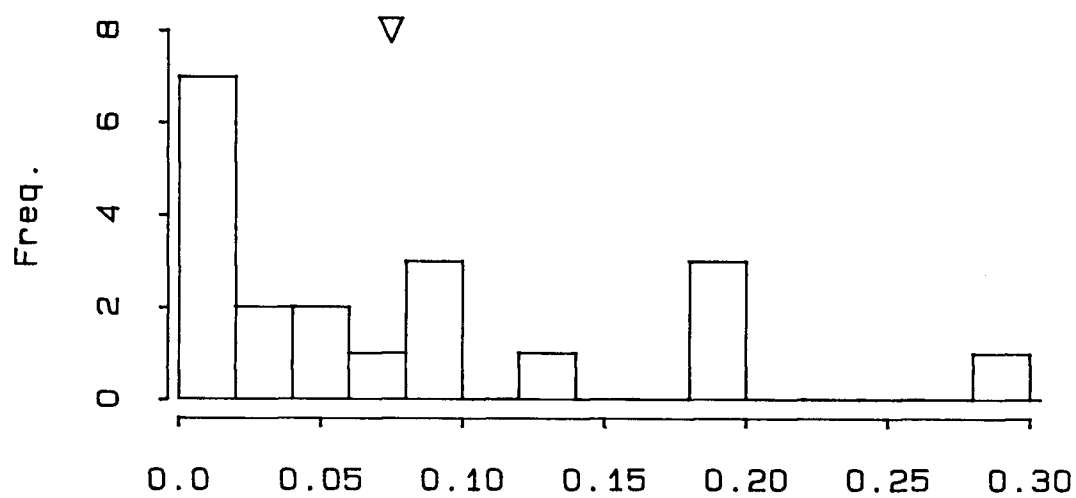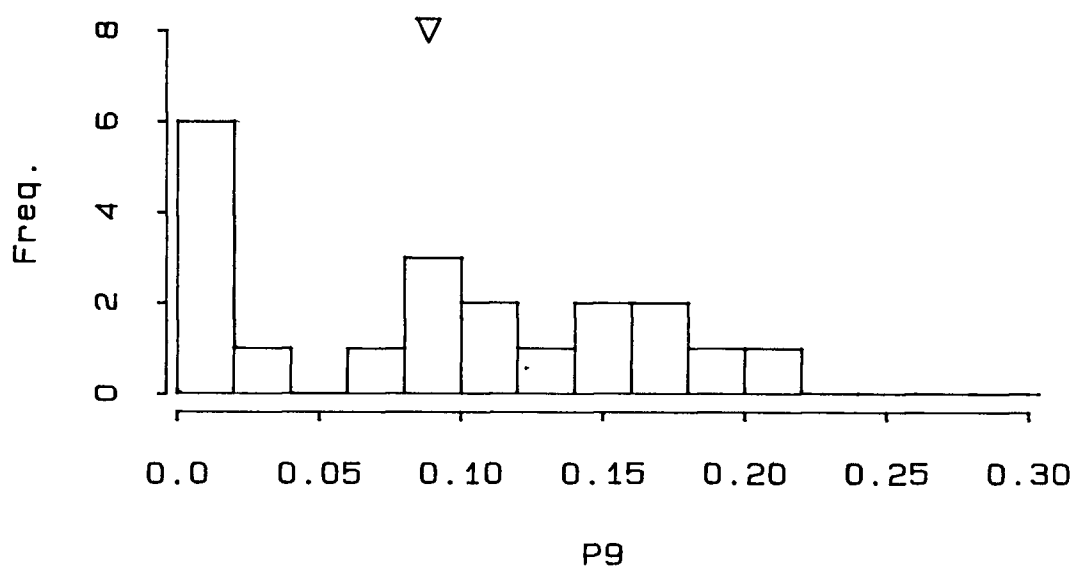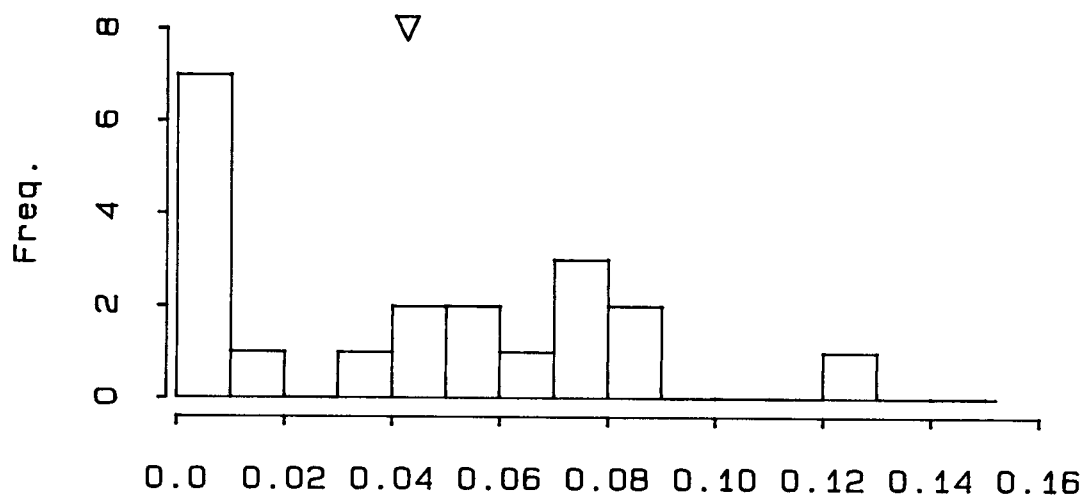
FIGURE 54

Odessa

Trebi
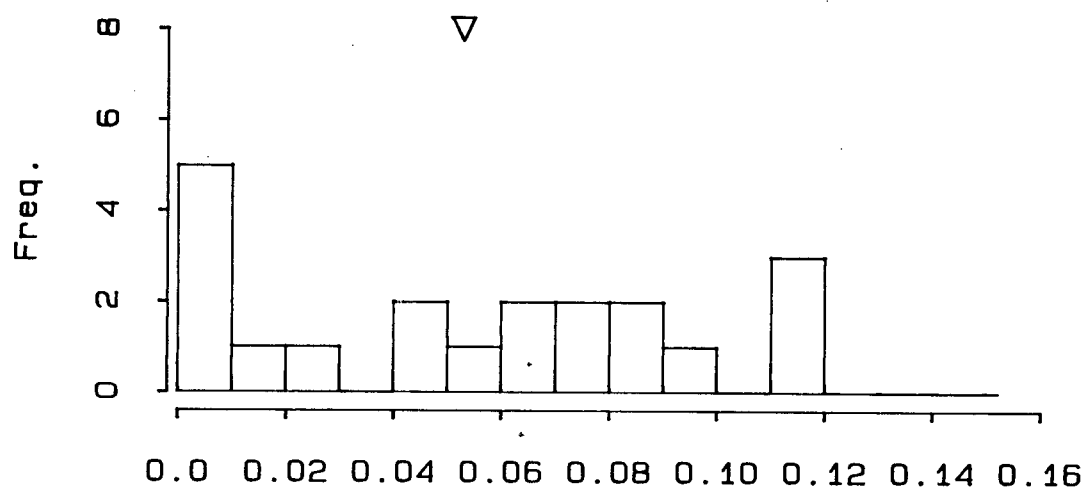
P10

FIGURE 55

Odessa

Trebi

P11t
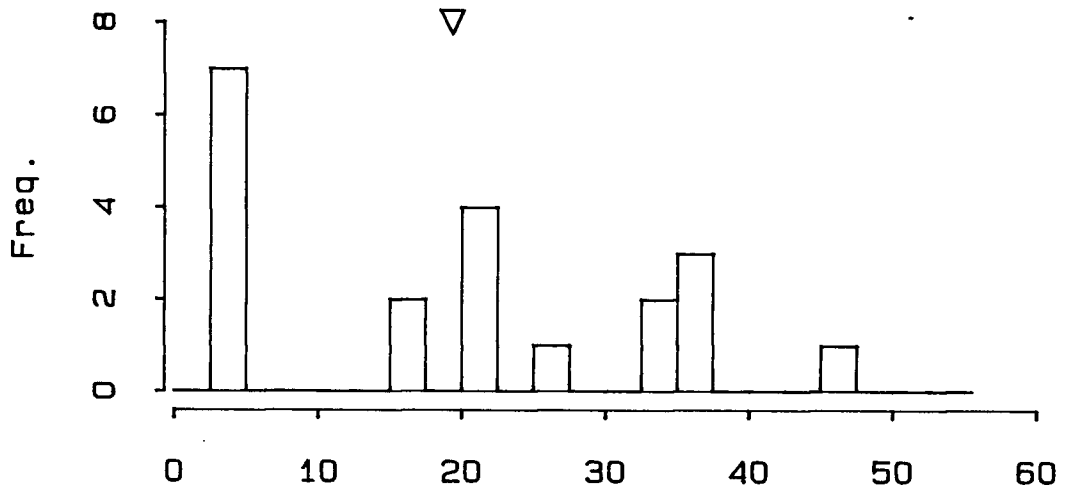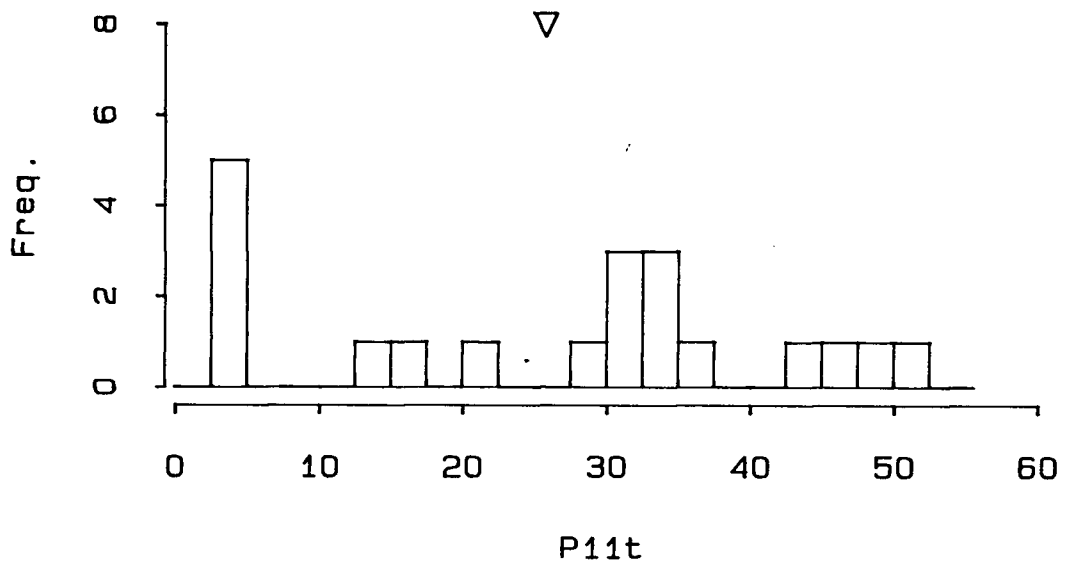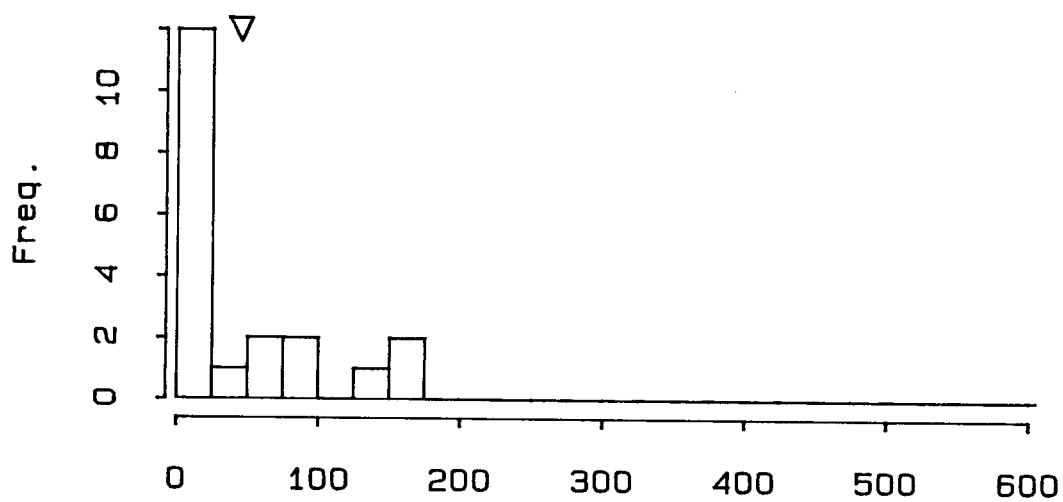
FIGURE 56

Odessa

Trebi
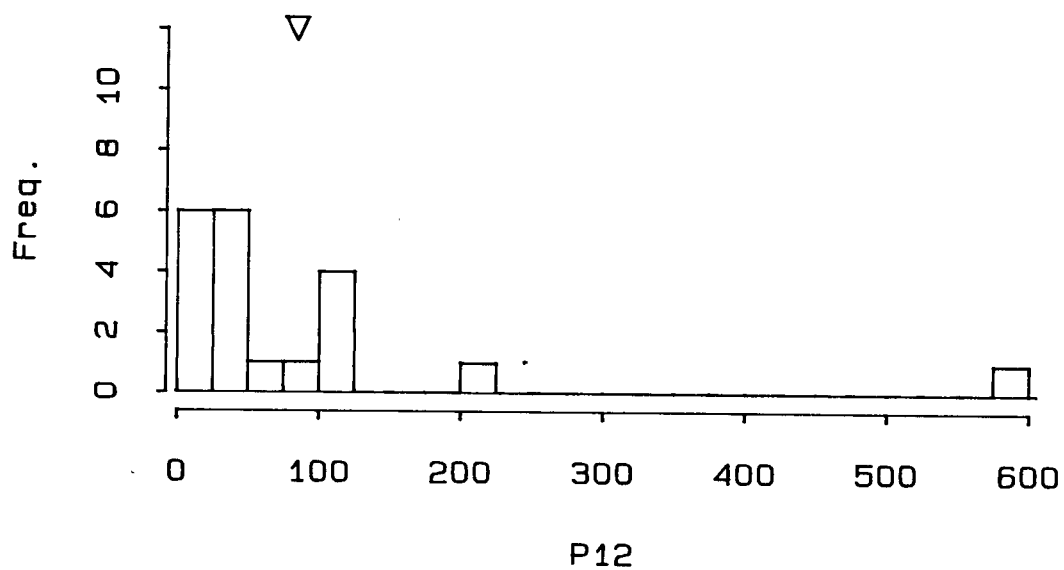
P12

FIGURE 57

Odessa
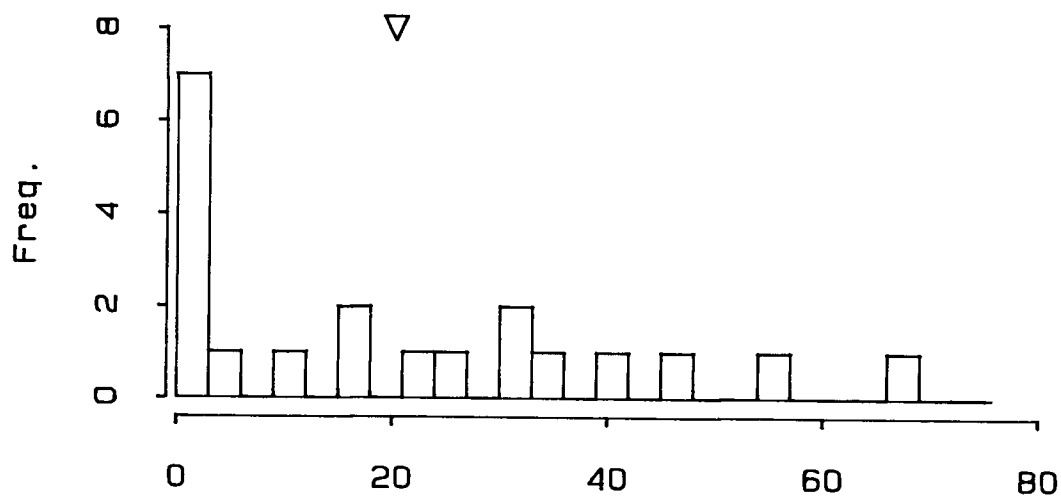
Trebi
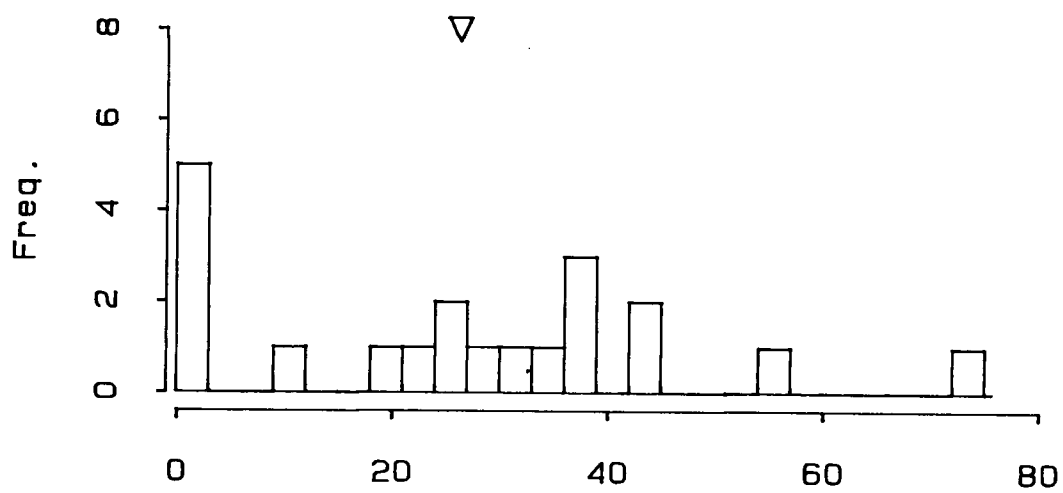
P13

FIGURE 58

Odessa
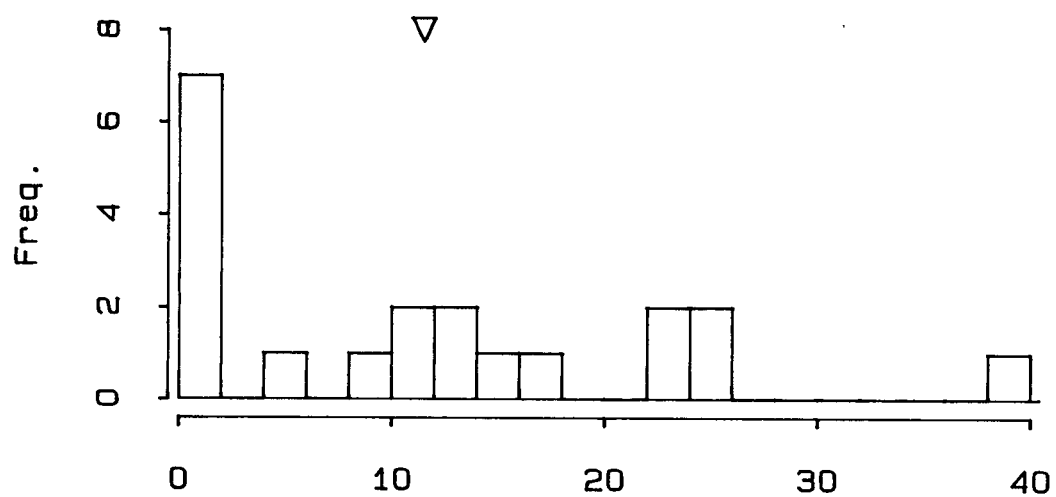
Trebi
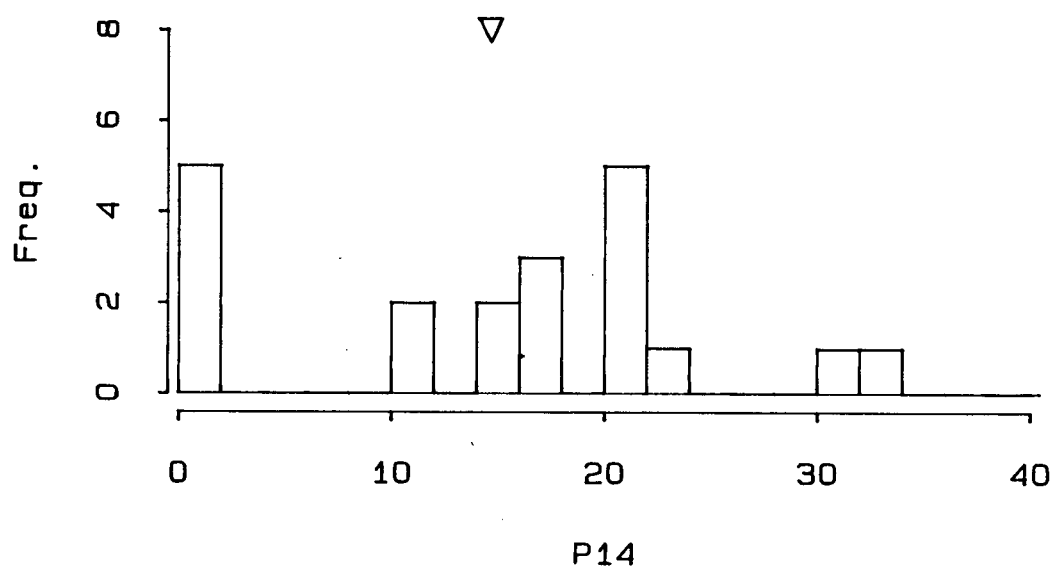
P14

FIGURE   59

Odessa
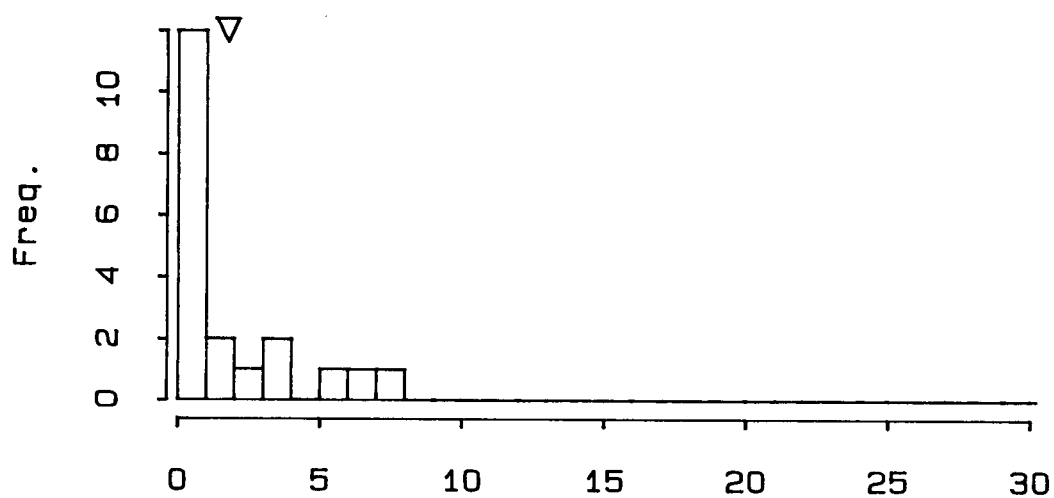
Trebi
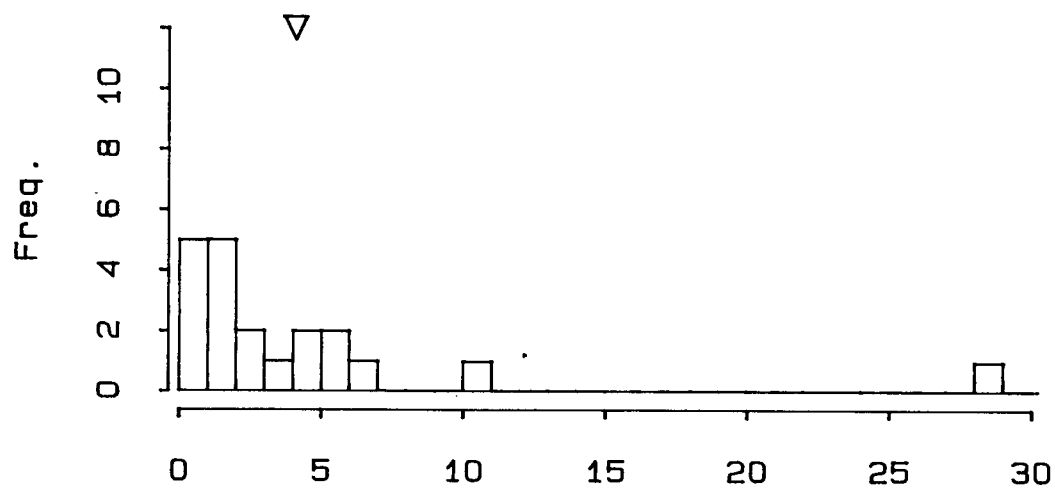
P15

FIGURE 60

Odessa
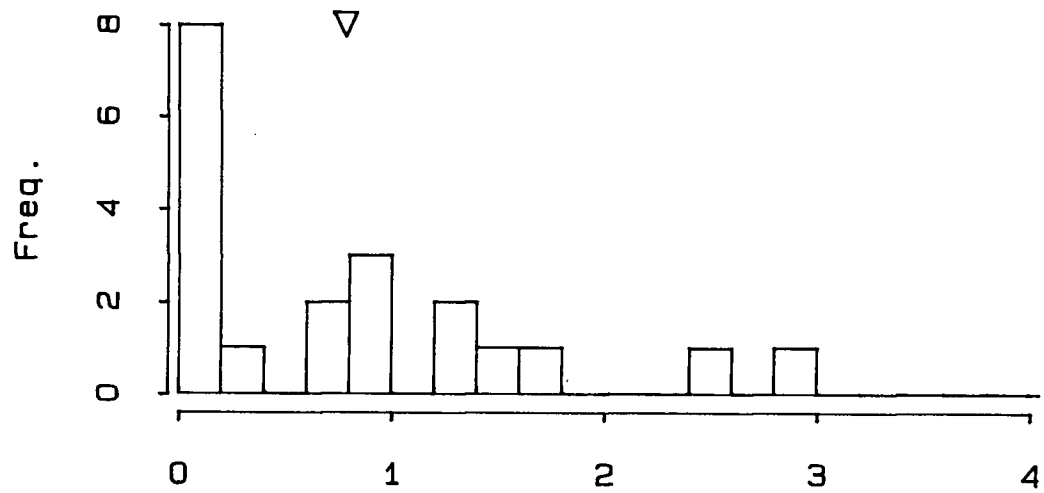
Trebi
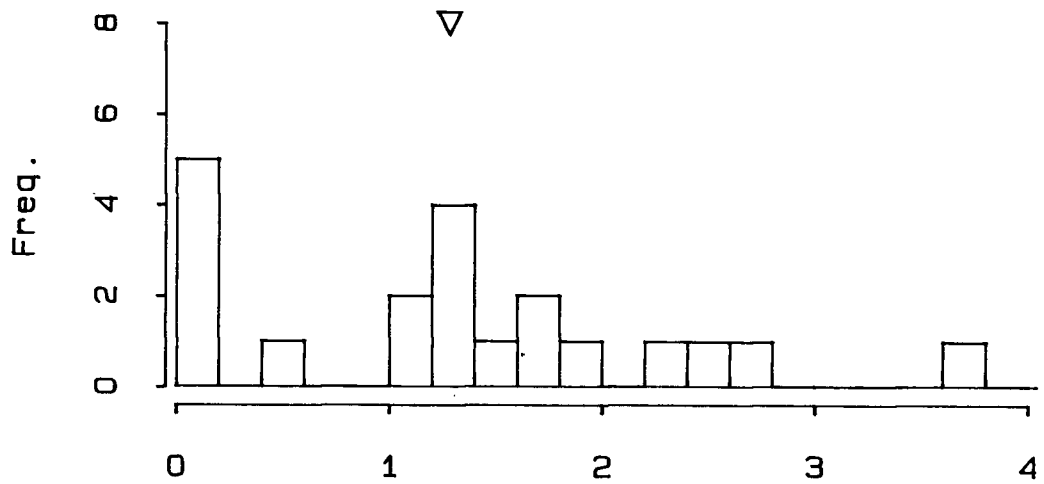
P16

FIGURE 61

Odessa

Trebi

P17

FIGURE 62

Odessa

Trebi

P18t