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Modelling the Epidemiology of Barley Yellow Dwarf Virus.

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ABSTRACT

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MODELLING THE EPIDEMIOLOGY OF
BARLEY YELLOW DWARF VIRUS.

by Derek Morgan

A model was developed simulating the population dynamics of Rhopalosiphum padi and the spread of barley yellow dwarf virus (BYDV) in barley fields during the autumn and winter.

The R. padi sub-system developed is a discrete-time, age-structured, deterministic, simulation model with temperature as the only driving variable. The BYDV sub-system is also a discrete-time, deterministic model but is driven by rainfall as well as by temperature.

Both sub-systems were developed by fitting mathematical functions to published data, where possible. Information on BYDV epidemiology, such as aphid movement, virus acquisition and transmission, was obtained from laboratory experiments, and functions were fitted to these results also.

Both sub-systems were validated against field data, collected over three years, and a sensitivity analysis was carried out.

Peak population numbers of aphids predicted by the sub-system closely fitted those observed early in the season but failed to predict their later extinction. The BYDV sub-system underestimated virus spread early in the season but overestimated the final incidence of the disease.

Sensitivity analysis indicated that the aphid sub-system was sensitive to changes in temperature, nymphal development time and survival rates, while the BYDV sub-system was sensitive to changes in the latent period of the disease in the host plant and vector density.

Both sub-systems have revealed areas of the BYDV epidemiology where further research is required. The research has highlighted the usefulness of systems analysis in the study of virus epidemiology.

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1. Introduction

Barley yellow dwarf (BYDV) is probably the most wide-spread plant virus disease in the world (Plumb, Lennon & Gutteridge, 1986). Although only members of the Gramineae are susceptible, these include a number of economically important crops, such as wheat, *Triticum* spp., barley, *Hordeum* spp., maize, *Zea* spp. and rice, *Oryza* spp. Therefore, it is important to attempt to control the detrimental effect of the virus, within economical and political constraints.

In Britain, BYDV is economically important and affects cereal production in some regions. It is spread only by infective aphids feeding on susceptible hosts. In Britain, at least 23 different species of aphids have been identified as vectors (A'Brook, 1981).

The application of insecticides is the most common method of controlling BYDV. Advice on the need for, and timing of, insecticide sprays is based on the combination of local history of the disease, monitoring the field populations of vectors, and an estimate of the number of viruliferous aphids flying. This estimate, called the infectivity index (II) (Plumb & Lennon, 1981), is the product of the numbers of each aphid species caught in the nearest Rothamsted Insect Survey (RIS) 12.2m suction trap (Taylor, 1983) and the proportion carrying virus, accumulated weekly starting from the crop sowing date (Plumb, 1983). If the II exceeds a local pre-determined threshold, 50 for the Rothamsted area (Plumb, Lennon & Gutteridge, 1986), insecticides are recommended.

There are two major disadvantages to the II. Firstly the time required to determine the proportion of flying aphids carrying virus. This is assessed by caging aphids, caught live in a suction trap, onto virus-sensitive oat seedlings and then waiting for 2-4 weeks for any visual symptoms of virus infection (Plumb, 1983). Thus, there is an inherent delay of at least 2 weeks before advice on control measures can be given. If conditions during the period are favourable for virus spread, the delay could allow a damaging level of disease to develop in the field.

The second disadvantage is that the II is a reliable estimator of the risk only from primary infection; that is, spread caused by immigrant winged aphids. It does not take into account the risk of secondary spread, caused by the dispersal of the wingless offspring. Kendall & Smith (1981) found that a high II did not necessarily result in a high incidence of virus, and vice versa. They concluded that the extent of virus incidence was dependent on the number of aphids present and the time they were present and active on the crop,

which are dependent on climatic factors such as temperature and rainfall (Kendall & Smith, 1981).

The objectives of the present study were to :
(i) construct a model simulating the epidemiology of BYDV which incorporated both primary infection and secondary spread.
(ii) develop and experiment with the model to investigate the system so that key processes were identified and further research stimulated.
(iii) assess the usefulness and limitations of systems analysis in the study of virus epidemiology.

2. Epidemiology of Aphid-vectored Viruses

The epidemiology of aphid-vectored viruses is the result of interactions between virus, vector, plant and environment. This chapter discusses the interaction between these variables, with emphasis on how they impinge on the BYDV system.

2.1 Virus Classification and Isolates

Viruses have been conveniently classified into groups based upon their similarity of characteristics. The most widely adopted approach classifies viruses according to the types of vector-virus relationships (Harrison et al., 1971).

Non-persistent viruses require very short acquisition and transmission feeding times, usually less than a minute (Harrison et al., 1971). The periods of retention by the vector are also short and so vectors require frequent acquisition feeds to remain viruliferous. Non-persistent viruses include members of the Potyviruses, examples of which are potato virus Y (PVY), beet mosaic virus (BMV), the Cucumboviruses, such as cucumber mosaic virus (CuMV), and the Caulimoviruses, such as cauliflower mosaic virus (CaMV).

Vectors of persistent viruses require long acquisition and transmission feeds, usually several days depending on temperature (Sylvester, 1965; van der Broek & Gill, 1980), to acquire and transmit viruses. Vectors remain infective for life. Examples include the Luteoviruses, such as BYDV and beet western yellows, and Rhabdoviruses, such as lettuce necrotic yellow virus (LNYV) and sowthistle yellow vein virus (SYVV).

Semi-persistent viruses are intermediate in character, their vectors require acquisition and transmission feeds of hours, rather than minutes or days, and are retained by the vector for 1 or 2 days. Examples are the Closteroviruses, such as citrus tristeza virus (CTV).

Differences in the ability of aphids to transmit viruses have enabled virologists to identify different isolates of the same virus. Five isolates have been identified for BYDV (Rochow, 1970; 1979) :-

1. RPV - transmitted most efficiently by *Rhopalosiphum padi* (L.)
2. RMV - transmitted most efficiently by *R. maidis* (F.)
3. MAV - transmitted most efficiently by *Sitobion avenae* (F.)

4. SGV - transmitted most efficiently by *Shizaphis graminum* (L.)
5. PAV - transmitted non-specifically by *R. padi* and *Sitobion avenae*

The five isolates can be divided into two groups serologically. Rochow & Carmichael (1979) found that RPV and RMV were serologically related, as were MAV, SGV and PAV. However, identification of BYDV isolates by vector specificity alone is complicated by dependent transmission (Rochow, 1982), where in mixed infections of RPV and MAV the protein of RPV sometimes encapsulates the nucleic acid of MAV, allowing both to be transmitted by *R. padi*.

2.2 Virus-Host Interactions

2.2.1 Virus Sources

It is the quantity and availability of virus sources that determines how readily aphids acquire virus. Eradication of virus sources can result in control of the disease (Zitter, 1977). There are four principal sources of plant virus diseases; infected seed, taxonomically related crops, weed hosts, and "volunteer" plants which act as bridges between successive crops.

All seed-borne viruses are of the non-persistent type (Bennett, 1969). Infected seed acts as a primary source of virus which is spread by the activity of vectors. Screening programmes to ensure virus-free seed have proved successful in controlling seed-borne viruses; lettuce mosaic virus (LMV) provides an example of this (Zitter, 1977).

Similar or related crops sown near to susceptible hosts can act as sources of virus. A classic example is that of mangold clamps serving as sources of suger beet yellowing virus (BYV), suger beet mild yellowing virus (BMYV) and suger beet mosaic virus (BMV) (Broadbent et al., 1949). A decline in the incidence of these viruses was noted from the mid-1960's to mid-1970's as a result of a decrease in the acreage of mangolds (Heathcote & Byford, 1975).

Weed hosts are more important as sources for non-persistent than for either semi- or persistent viruses. Non-persistent viruses tend to have wide plant host ranges thereby increasing the likelihood of an aphid feeding on an infectious host plant. A typical example is alfalfa mosaic virus (AMV) which occurs naturally in 47 species from 12 families of plants (Hull, 1969).

In Britain, perennial grasses are the predominant source of BYDV for infection of autumn-sown cereals (Plumb, 1983). Aphids, especially *R. padi*, feed and develop on grasses, acquiring the virus from infected plants which they then transmit to newly emerging cereal crops. Virus incidence in grasses can be high, for example Doodson & Saunders (1967) found that 93% of *Lolium perenne* sampled contained virus, even though symptoms were rarely apparent. Although grasses may be potential sources of virus, they need not necessarily be efficient ones. In acquisition and transmission tests *L. perenne* is a poorer source of virus than cereals (Plumb, 1983). Also, Coon (1959) found large differences in the bionomics and virus acquisition of cereal aphids kept on different grasses. Small proprtions of aphids acquired the virus from plants on which there were low numbers of aphids.

Volunteer plants, growing from shed seed and subsequently infected, and host regrowth, can be important sources of virus because of their proximity to new crops. Several important viruses are associated with volunteers, including BMV, beet western yellowing virus (BWYV), peanut mottle virus (PMV) and BYDV (Zitter, 1977).

2.2.2 Plant-Host Response

The susceptibility of a plant to virus infection and the subsequent availability of virus particles to new aphid vectors are critical to the spread of the disease (Zitter, 1977). Factors which can influence the susceptibility of plants are genetic differences, plant age, and leaf age at the time of infection (Swenson, 1969).

Barley varieties differ in their susceptibility to BYDV when tested by aphid inoculations (Oswald & Houston, 1953; Rasmusson & Schaller, 1959). Similar results have been found with other virus-host interactions, for example potato virus Y (Bagnall & Bradley, 1958).

The older a plant becomes the less prone it is to inoculation by aphids (Zitter, 1977). For example infection of potato with leafroll decreased with increased plant age (Knutson & Bishop, 1964). Similarily, Bagnall & Bradley (1958) found that older potato leaves were less susceptible than younger ones to infection with potato virus Y, as were cucumbers with CuMV (Stimman & Swenson, 1967).

The availability of virus in the plant for aphids is dependent on a number of factors, including virus titre, phloem accessibility and age of leaf. Gill (1969) found that the titre of BYDV did not remain constant in a plant as there were cycles in infectivity during

the time that the plant was actively growing. The proportion of aphids transmitting the virus was less if they had fed on the plant during a trough than at a peak in the cycle.

Any delay in the vector acquiring the virus caused by lengthening the time for the aphid's stylets to reach the phloem of the host plant inhibits spread of the disease. For example Haniotakis & Lange (1974) correlated decreased phloem contact by *Myzus persicae* (S) with resistance to BYV in sugar beet. Scheller & Shukle (1986) proposed that inhibition of BYDV transmission, and hence resistance to the virus, could be achieved by achieved by disrupting aphid contact with the phloem of the host plant.

The age of leaves from which aphids had acquired BYDV affect the efficiency of transmission of the disease. Aphids that had fed on older leaves transmitted the virus less efficiently than those that had fed on younger leaves (Foxe & Rochow, 1975),

2.2.3 Virus Isolates and Mixed Infections

Mixtures of isolates, or strains, of a single virus in a single plant can be transmitted independently by aphids (Watson, 1967). Inoculation of cereals with a mild strain of BYDV can prevent infection of plants by more virulent strains (Jedlinski & Brown, 1965), although Plumb (1983) argued that cross protection (one strain interfering with another) is uncommon and unlikely to influence BYDV epidemiology greatly.

In plants infected with two unrelated strains of BYDV new strains of the virus may result (Rochow & Jedlinski, 1970). However, plants infected with two or more strains of persistent virus are uncommon, they are more likely to be infected with different non-persistent viruses (Zitter, 1977).

2.3 Aphid-Virus-Host Interactions

2.3.1 Source of Aphids

The complex life cycle of *R. padi* ensures that populations of it are maintained throughout the year.

R. padi is the most aphid in S.E. England in the autumn (Taylor, 1977). It is heteroecious, with bird cherry, *Prunus padus* (L.), as its primary host. It overwinters either as eggs on *P. padus* or viviparously on Gramineae (Gair, 1953; Leather, 1980). Eggs on *P. padus* eggs hatch between February and April and a number of apterous

generations occur before alate aphids are induced by overcrowding and declining host quality (Dixon & Glen, 1971). The alatae colonize cereals on which a number of generations occur parthenogenetically. In the autumn and winter, short day lengths and low temperatures induce the production of winged gynoparae and males, which fly to the primary host. The gynoparae produce oviparae which mate with the males and then lay the overwintering eggs (Dixon & Dewar, 1974).

Primary infection of autumn-sown cereal crops is the result of infective alate exules carrying virus from a previous cereal crop, infected volunteers or graminaceous weeds(Plumb, 1983).

2.3.2 Aphid-Virus Interactions

BYDV infection not only affects the host plant but also the behaviour and demography of its aphid vectors. Alate aphids when offered the choice are more likely to alight on infected plants than on healthy ones (Ajayi, 1981). Gildow (1980) found that more alate *R. padi* offspring were produced on BYDV-infected than on healthy oats. Feeding on BYDV-infected plants was found to increase the fecundity of *R. padi*, but not that of *S. avenae* and *M. dirhodum* (Markkula & Laurema, 1964). Whether BYDV infection increases the longevity of *S. avenae* is open to conjecture. (Miller & Coon, 1964) found that it increased longevity whereas Elamin (1975) found that it was reduced, while Markkula & Laurema (1964) detected no significant difference between aphids kept on BYDV infected and healthy plants.

2.2.3 Aphid Population Dynamics

Zitter (1977) considered that the number of aphid vectors present in a crop was the most important factor for virus spread. However, other factors, apart from aphid density, should also be considered. Shanks (1965) found that aphid movement, rather than aphid numbers, was important in the spread of strawberry viruses. Gill (1970) discovered that the timing of the migratory flight of aphids, in relation to the age of the crop, was important for primary infection of BYDV. He found that virus incidence was highest on the latest sown crops. This was probably caused by immigrant alates alighting preferentially on the latest-sown crops in response to the visual stimulus given by the mosaic of infected plants against a soil background. This is known to be more attractive to alates than a continuously overlapping foliar canopy (A'Brook, 1968).

Aphid population dynamics can be considered as four simultaneously occurring processes, migration, including crop colonization, development, survival and reproduction.

2.3.3.1 Migration and Crop Colonization

Migration of aphids to cereals can be conveniently be divided into the three periods, May and June, June to August, and August to November (Taylor, 1977). Migrant alate *R. padi* are most numerous in the autumn (Tatchell et al., 1988). Autumn-sown cereals emerging from early-September to late-October are colonized by migrant virginoparae. At that time males and gynoparae of *R. padi* are returning to *P. padus*. Males are easily distinguished from the two female morphs which are morphologically very similar (Rogerson, 1948). Tatchell et al., (1988) were able to differentiate between the female morphs by offering aphids caught live in suction traps, a choice of leaf discs of *P. padus* or a strip of winter barley. Dixon (1971) showed that gynoparae do not reproduce on cereals. In 1986, males were present throughout the autumn, also up to mid-September all alate females that were trapped were virginoparae but, from mid-September onwards, the females that were caught were mainly gynoparae. Therefore, autumn-sown crops emerging before mid-September may be particularly susceptible to colonization by alate viviperous *R. padi* and therefore to infection with BYDV (Tatchell et al., 1988).

Immigrant alate *R. padi* accumulate in the margins of fields with hedgerows (Dean & Luuring, 1970) although their subsequent dispersal throughout the crop is usually rapid (Carter, McLean, Watt & Dixon, 1980). Alate *R. padi* may make repeated short flights between plants depositing a few offspring on each whereas *S. avenae* does not (Dry & Taylor, 1970). Dispersal of aphids throughout a crop, and hence virus spread, is also the result of movement by apterous aphids walking from plant to plant (Itô, 1960).

2.3.3.2 Survival

Survival of aphids depends upon both abiotic and biotic factors.

Abiotic factors include temperature and rain. Laboratory experiments on the effects of high temperatures on aphid survival have shown that all *R. padi* nymphs were killed when kept at 30°C (Dean, 1974). Little information is available on the lethal effects of low temperatures on *R. padi*. Williams (1980) found that viviparous *R. padi* were killed when they were kept for 90mins at -4°C. Knight et al. (1986) compared changes in field populations of *S. avenae* as affected by winter temperatures and found that populations were halved after experiencing temperatures of -8.1°C. Laboratory experiments using *M. persicae*, reared at 20°C, showed that 50% of the aphids were killed by a temperature of about -8°C (Bale et al.,

1988). Bale et al. (1988) hypothesized, based on the similarity between their results and those of Knight et al. (1986), that pre-freezing mortality may be a common occurrence in aphids. However, Evenhuis (1968) found that nymphs of *Rhopalosiphum insertum* (W.) survived when kept at -7.5°C unless they were moistened first.

Little is known of the fatal effects of rain on any aphid species. Simulated rain of 1-2cm killed 95% of *Sitobion miscanthi* (Tak.) tested in the laboratory (Dhalival & Singh, 1975). Dean & Wilding (1971) attributed a 65% reduction in a field population of *M. dirhodum* to rain, although a similar reduction was not observed after an equivalent amount of rain had fallen a week later. Donn & Wright (1955) found that heavy rainfall was one of the most consistent factors in reducing field populations of the pea aphid, *Acyrthrosiphon pisum* (H.). The effects of rain on cereal aphid survival varies as the shelter from preferred feeding sites varies between aphid species and crop growth stage (Vickerman & Wratten, 1979).

Cereal aphids are attacked by a number of predators, parasitoids and fungal pathogens. These natural enemies may regulate populations below pest damage thresholds and in doing so possibly inhibit virus spread (Hodek & van Emden, 1972). Although much attention has been given to the effects of natural enemies during the spring and summer months (for detailed reviews see Carter et al., 1980; Vickerman & Wratten, 1979) much less information is available for the autumn and winter.

Much of the literature on cereal aphid predators has focused on aphid-specific predators, which include Coccinellidae (Coleoptera), Syrphidae (Diptera) and Chrysopidae (Neuroptera). Both larvae and adults of coccinellids and larvae of syrphids and chrysopids eat aphids. Adult syrphids feed on nectar and pollen, while adult chrysopids eat honeydew and pollen (Carter et al., 1980). The effectiveness of aphid-specific predators is dependent on their density and phenology coinciding with that of their prey. All require a threshold density of aphids to remain and breed in a crop and although the values of these thresholds are not known they may be relatively low (Vickerman & Wratten, 1979).

Although the numbers of aphid-specific predators may be low in the spring it is possible that a small amount of predation at this time may significantly reduce the subsequent peak aphid population (Chambers, Sunderland, Stacey & Wyatt, 1983; Rautapää, 1976). Aphid-specific predators may also be important later in the season, if numbers are sufficient, in preventing outbreaks or bringing forward

the 'crash' in aphid numbers (Chambers & Adams, 1986; Chambers, Sunderland, Wyatt & Vickerman, 1985). Rautapää (1977) used caged experiments to investigate the effect of known predator densities on the population dynamics of cereal aphids. Initial ratios of 50 and 5 *R. padi* per *Chrysopa carnea* (S.) larva resulted in population reductions by 10 and 50%, respectively.

Polyphagous predators include Carabidae and Staphylindae (Coleoptera), Dermaptera, Araneae and Acari. Potts & Vickerman (1974) found significant negative correlations between the numbers of aphids and the number of predatory insects in a cereal crop. Although aphid-specific predators were present, their numbers were insufficient to account for the relationship, implying that polyphagous predators were responsible. In studies that involved either dissection (Potts, 1977; Vickerman & Sunderland, 1975) or serological techniques (Sopp & Chiverton, 1987) to test for the presence of aphids, a high proportion of polyphagous predators were found to have eaten aphids. Sopp & Chiverton (1987) discovered, during the autumn and winter, that up to 75% of carabids and 43% of staphylinids collected had eaten aphids. There was also a significant relationship between aphid density and the percentage of linyphiids containing aphid remains, although no significant relationships were found for either carabids or stapylinids (Sopp & Chiverton, 1987). The role of polyphagous predators is further confounded as some species are nocturnal and so their impact is easily overlooked by researchers in the field during the day (Vickerman & Sunderland, 1975).

Although studies have shown that polyphagous predators reduce cereal aphid populations, little data have been published on food preferences, searching behaviour and consumption rates of individual species (Sunderland, 1988).

Cereal aphid parasitoids belong to the families Aphelinidae and Aphidiidae of the Hymenoptera. The most common species belong to the latter, and are *Aphidius ervi* (H.), *A. picipes* (N.), *A. rhopalosiphi* (S.), and *Praon vulucre* (H.) (Carter et al., 1980).

Although much work has been reported on cereal aphid parasitoids (for a detailed review see Vickerman & Wratten, 1979) little quantitative data have been presented. Rautapää (1976) discovered that only about 5% of aphids found in fields in Finland were parasitised. However, the method used by Rautapää (1976), counting the number of mummified aphids in the field, greatly underestimates parasitism (Dean, 1974) as aphids parasitised but not yet mummified are classified as 'live'. Also the length of time from oviposition to mummification is nearly twice as long as that from

mummification to emergence, so mummies have a shorter life span than live but parasitised aphids and, therefore, they will be found in lower numbers at any one time (Carter et al., 1980). Dean (1974) found that parasitism varied from year to year and between aphid species, in 1970 10% of *S. avenae* and 24% of *M. dirhodum* were parasitised, while in 1971 39% and 49% were, respectively. Vorley (1983) found similar results between fields and years, 29-32% of *S. avenae* were parasitised.

Several species of hyperparsitoids attack cereal aphid parasitoids and, therefore, diminish their influence on limiting aphid numbers. The extent of hyperparasitism varies from year to year and between aphid hosts, possibly because of aphid numbers and preferred feeding sites (Dean, 1974). Hyperparasitism can reach high rates. Vorley (1983) reported that it was possible for 80-90% of parasitised *S. avenae* to be hyperparasitised, and Jones (1972) suggested that heavy hyperparasitism one year would result in fewer parasites the following year, which could in turn lead to an outbreak in aphids.

Fungal pathogens of the Entomophthorales infect cereal aphids (Dean & Wilding, 1971; 1973). Pathogen incidence varies from region to region and from year to year, with peak incidence occurring late in the summer (Dean & Wilding, 1971; 1973). Such diseases have, often been considered unimportant, except in occasional years, for example in 1970 and 1971 when up to 80% and 53%, respectively, of aphids were infected (Dean & Wilding, 1971; 1973). High levels of disease late in the summer could result in high levels of inoculum in autumn-sown crops, or in the following year (Vickerman & Wratten, 1979). One of the principal factors influencing fungal pathogens is rain. Dean & Wilding (1971) found that an increase in fungal infection coincided with heavy rainfall, probably because the pathogens require a humid atmosphere for successful sporulation (Wilding, 1969).

2.4 Conclusions

A large number of factors are involved in the epidemiology of BYDV. Our underestanding of the system is further complicated by different researchers giving different priorities to the various factors, for example the effect of BYDV on aphid longevity (Elamin, 1975; Markkula & Laurema, 1964; Miller & Coon, 1964). The influence of environmental variables seems to account for many of the controversies, although vector species and virus strains may also be an influence. There are too many interacting variables to manipulate empiracally, so systems analysis may offer a solution to imrove understanding. A model of BYDV epidemiology should take into account

all of the factors discussed above that are considered important, and by quantifying and manipulating them mathematically greater insight will be achieved into how they are all inter-related.

3. Modelling

The complexity of virus epidemiology makes it difficult to assess the importance of individual components experimentally. However, by constructing a model which describes the processes involved, and their relationships to each other, it is possible to experiment with the system and increase knowledge. This chapter describes :
(i) the different types of models that can be used in virus epidemiology,
(ii) one approach to construct and develop a simulation model,
(iii) a number of examples of models used in aphid population dynamics and virus epidemiology.

3.1 Types of Model

3.1.1 Analytical Models

Analytical models are simple problem-orientated models. Their structure is characterized by having one or two differential, difference or algebraic equations with few variables with complex coefficients. Input is small and solutions have closed-forms. The simplicity of an analytical model makes it is possible to carry out a rigorous mathematical analysis of its behaviour.

3.1.2 Simulation Models

Simulation models are complex, goal-orientated models. Their structure consists of many differential, difference or regression equations with many variables and simple, easily measurable coefficients. Input is large and solutions are quantitative. Their complexity dictates that they be solved using computers and a rigorous mathematical analysis of their behaviour is rarely possible.

Both types of models can either be deterministic or stochastic. A deterministic model uses mean values of parameters and variables and given one set of input only one set of output is possible, whereas a stochastic model uses probability functions to define values of parameters and one set of input produces many possible sets of output. Deterministic models are more convenient to develop but as there are many possible outcomes to an ecological process, stochastic models tend to be more realistic.

3.2 Modelling Approach

Although Fransz (1977) said that there are as many approaches to the construction and development of a model as there are modellers. This section briefly describes one possible procedure to construct, develop and implement a simulation model.

The approach uses a series of inter-dependent steps. The first step is to clearly define the objectives of the study. Although this may appear an obvious beginning it is essential as the definition of the objectives determines the type of model to be constructed, the precision of the data required and the criteria to appraise the model. If there is a limit to the amount of money, labour or equipment available then clearly the objectives of the study are affected. Therefore a compromise has to be made between the quality of the data which can be collected and the complexity of the model which can be developed.

The next step is to describe the system being modelled by listing the components which the modeller feels are important. All the elements thought to be unimportant are not considered further. The inter-relationships between components of the model can be described using relational diagrams (de Wit & Goudriaan, 1978). Types of variables are represented by different symbols :
(i) state variables (shown in rectangles) characterize and quantify the state of the system, eg, number of plants infected;
(ii) driving variables (shown in parentheses) influence the system but are not influenced by the system, eg, temperature;
(iii) rate variables (shown in valve symbols) quantify the rate of change of state variables, eg, development rate of insects;
(iv) auxiliary variables (shown in circles) are intermediate variables which help simplify complex calculations and help understanding of the processes, eg, physiological time (the integration of time and temperature above a threshold), and
(v) output, chosen by the modeller to fulfil the objectives of the study, they can be state, rate or auxiliary variables.

The third step is to quantify the relationships described in the relational diagrams. This begins with an extensive review of the relevant literature so that published data can be used. However, during this process it may become apparent that some relationships, thought to be important by the modeller, have never been studied, or if they have, the data are not in a suitable format to be used, and so experiments have to be carried out to provide the information.

Once a model has been constructed it has to be verified and validated before it can be used experimentally or to provide advice on control measures. Verification is the process of confirming that the computer program is working in the intended way, it is a form of debugging computer code. Validation is the quantitative comparison of the model's predictions with the observed results. Although there are statistical tests available to do this robustly (Shannon, 1975) the most common method is to compare the data visually using graphs of observed and predicted trends.

Once the modeller is satisfied with the accuracy of the predictions of the model, in relation to the objectives of the study, the model can be used experimentally. Sensitivity analysis, the process of altering parameter values, can be made to determine the importance of a process on the system. An analysis can either be coarse, where processes are omitted from the model, or fine, where small positive or negative changes are made to parameter values. Fine sensitivity analysis can be used to determine the precision to which the value of the variable should be known, for example, if a small change in a parameter results in a large change in model output then the value of the parameter needs to be known accurately. Conversely, if a small change of the parameter results in a small change in output the parameter value need not be known so accurately. Therefore sensitivity analyses can highlight processes in the system which are important and so stimulate further experiments. However, the more complex a model, the larger the number of relationships it has, the greater the potential number of runs needed to study the interactions in the system and the greater the cost in computer resources.

3.3 Review of Simulation Models

Simulation models have been used for over 20 years to investigate the problems of controlling aphids (Conway, 1978). However, the approach has tended to be under-used to study virus epidemiology, possibly because of the complexity of a host-virus-vector system (Carter, 1986). This section reviews the use of simulation to study both aphid populations and virus spread (Table 1).

3.3.1 Simulation Models of Aphid Population Dynamics

Hughes & Gilbert (1968) pioneered the variable life-table approach to modelling aphid population dynamics. They constructed a model describing the relationships between the cabbage aphid, *Brevicoryne brassicae* (L.), syrphid larvae and adults, the parasitoid, *Diaeretus rapae* (Curtis), one of its hyperparasitiods and

Table 1. Summary of the review of simulation models of aphid population dynamics.

Authors	Pest	Natural Enemies	Driving Variables	Comments
Hughes & Gilbert (1968)	*B. brassicae*	Syrphids, parasitoids and hyper-parasitoids	Temperature and rain	Introduced QUIPS
Gilbert & Hughes (1971)	*B. brassicae*	Parasitoids	Temperature	Stochastic parasitism
Gilbert & Guiterrez (1973)	*O. maxima*	Parasitoids	Temperature	Parasitoid strategies
Gutierrez et al. (1974)	*A. craccivora*	Syrphids, coccinellids and parasitoids	Temperature	Low temperature mortality
Frazer & Gilbert (1976)	*A. pisum*	Coccinellids	Temperature	
Chua (1978)	*B. brassicae*	Parasitoids and hyper-parasitoids	Temperature	
Rabbinge, Ankersmit & Pak (1979)	*S. avenae*	Syrphids, parasitoids and hyper-parasitoids	Temperature	Functional response of parasitoids
Barlow & Dixon (1980)	*E. tiliae*	Coccinellids and capsids	Temperature	Alternative prey and sptial dynamics
Carter, Dixon & Rabbinge (1982)	*S. avenae*	Coccinellids, parasitoids and fungal diseases	Temperature	Effect of host plant
Vorley & Wratten (1985)	*S. avenae*	Parasitoids	Temperature	Parasitoid strategies
Wiktelius & Patterson (1985)	*R. padi*	Parasitoids	Temperature	Plant breeding strategies

weather conditions. They introduced the concept of QUIPS, a unit of physiological time, equivalent to one QUarter of an aphid Instar Period (an instar period is the time taken between moulting from birth to the fourth instar). All relationships incorporated in the model were estimated from laboratory and field data, except mortality caused by rainfall. Initially they assumed that this mortality was low and constant. However, discrepancies between observed and predicted results suggested that heavy rain could reduce aphid populations in the field. Therefore they applied 'an appropriate instantaneous mortality due to heavy rainfall in the model. The authors failed to quote the relationship or the data used in its genesis and it appears that some fine tuning of the model was carried out which then reduces its explanatory usefulness.

Gilbert & Hughes (1971) used the model presented by Hughes & Gilbert (1968) to examine stochastic variation of aphid populations, different parasitoid strategies, and biological control of the aphids. Stochastic variation was achieved by using either the binomial or Poisson distributions to generate probabilities of extinction of aphids. However, they had no biological data to support the choice of distribution, or to estimate its parameters. They found that there was no difference between probabilities of extinction estimated stochastically or deterministically and suggested that this was due to the small densities of aphids in the study masking the stochastic variation. Next they addressed the dilemma of parasitoid efficiency and host extinction by carrying out a sensitivity analysis of parasitoid fecundity, the ratio of immigrant pests to immigrant parasitoids and parasitism timing. They found that the approaches naturally adopted by the parasitoids were the best possible without eliminating their hosts. Comparing attempts of biological control experimentally with output from the model, Gilbert & Hughes (1971) found large differences. This they attributed to gaps in the model, such as, whether parasitism was randomly distributed or aggregated.

Using a model of the population dynamics of the thimbleberry aphid, *Oestlundia maxima* (M.), as a 'biological model', Gilbert & Gutierrez (1973) showed how an aphid employs the most efficient strategy, namely increased fecundity, reduction of its teneral period and production of a higher ratio of gynoparae to virginoparae, to utilise its host plants resources. They also showed that aphid parasitoids were not employing the most efficient method of utilising the aphids and observed parasitism was not as great as was expected.

The effect of biotic and abiotic factors on the survival of the cowpea aphid, *Aphis craccivora* (Koch), was studied by Gutierrez, Havenstein, Nix & Moores (1974). Initially they arbitrarilly set

predation by syrphids, coccinellids and chamaemyiids at 1% and parasitism at 1% but later varied predation to study its importance. How they arrived at these values was not mentioned. Comparison of field observations with predictions from the model differed and this the authors attributed to low temperatures killing off the aphids. Thus another mortality factor was introduced into the model, death by frost, and was calculated as the difference between observed aphid numbers and those predicted considering mortality from biotic factors. The model was re-run, with mortality from both biotic and abiotic factors, and output from the model resembled field observations very closely.

One of the most important models of the relationships between aphids and predators was that developed by Frazer & Gilbert (1976) between the pea aphid, *Acyrthosiphon pisum* and the coccinellid, *Coccinella trifasciata*. They studied components of predation both in the laboratory and field and related the rate mathematically to predator and prey densities, predator voracity, prey-age distribution and temperature. Field cage experiments of aphids and aphids plus beetles were carried out and the data from these were compared with predictions produced by the model. However, the authors carried out some calibration of the model by manipulating the temperature-dependent activity coefficient of the beetle to improve the fit of the data. Thus the explanatory usefulness of the model was reduced.

Chua (1978) used the relationships between the cabbage aphid, *B. brassicae*, its primary parasitoid, *Diaeretiella rapae* (M.) and the hyper-parasitoid, *Alloxysta brassicae* (A.) as a 'biological model' for similar systems. Using a simulation model and a closed system of caged laboratory experiments Chua (1978) showed that the parasitoids could eliminate the aphids and the hyperparasitoids could eliminate the parasitoids. Mortality in all cases was the result of parasitism and the model and the experiments did not consider other factors affecting insect survival. Thus the use of the model to show how effective parasitoids could be in reducing aphid numbers in the field is neglible, as it can only be used to show how effective they are in closed laboratory systems.

Rabbinge, Ankersmit & Pak (1979) studied the epidemiology of the grain aphid, *S. avenae*. Although limited data were available on natural enemies, they constructed a procedure to describe predation of aphids by the syrphid, *Syrphus corollae* (F.). Predation rate was determined by prey and predator densities and by the relative predation rate which was dependent on temperature and area of search. Thus a functional response was used to calculate the relative predation rate to allow for decreased predation at low aphid

densities due to increased searching time of the predators. Sensitivity analysis of the model suggested that much more work on the system was required, especially on predation, parasitism, *Entomophthorales* infection, emigration and immigration. Rabbinge et al. (1979) concluded that the final aim of the model was to construct simple formulae predicting the epidemiology of *S. avenae* to be used in the control of the pest.

A very comprehensive simulation study of the population dynamics of an aphid species was carried out by Barlow & Dixon (1980). Not only did they study the relationships between the lime aphid, *Eucallipterous tiliae* (L.), and two of its predators, the two-spotted ladybird, *Adalia bipunctata* (L.), and the black kneed capsid, *Blepharidopterous angulatus* (Fall) on lime trees but also considered an alternative prey species, the leaf-hopper, *Alnetoidea alneti* (Dahlbom). Sensitivity analyses of the model indicated that weather was important as both a mortality and disturbance factor. Also they found that the lime trees regulated other components of the aphid's population dynamics, such that a decline in the nutritional quality of the trees resulted in a decline in aphid numbers. The predators only regulated aphid numbers when the latter were low, with the coccinellid being more effective than the capsid.

A second simulation model of *S. avenae* dynamics describing the pest on wheat in southern England was developed by Carter (1978). Mortality was a combination of predation by the coccinellid, *Coccinella septempunctata* (L.), parasitism and fungal pathogens. Carter (1978) also considered the effect of crop growth stage on the morph determination of newly laid nymphs. They found that the major cause of aphid numbers crashing was alates emigrating from the crop stimulated by unsuitable plant growth stages and high aphid densities. Sensitivity analysis indicated that instar durations, survival and reproductive rates were important in population dynamics, and accurate values of the processes should be known. The model also exposed how little was known of the settling behaviour of immigrant, and emigration of alates, and the author felt this was an area that required further research. Carter, Rabbinge and Dixon (1982) used the model to construct simple models to forecast numbers of *S. avenae* but all were unsuccessful, as the models failed to take into account the effect of weather and natural enemies on the dynamics of the aphid populations.

Vorley & Wratten (1985) developed a model of the population dynamics of *S. avenae* to examine the role of parasitoids on controlling aphid numbers relative to other forms of mortality. They developed their final version by running an initial version, having

no aphid mortality, and the difference between its output and field observations was called 'total mortality'. They then predicted the number of aphids mummifying from dissection data and physiological time and subtracted this from total mortality to leave 'residual mortality'. The authors suggested that residual mortality was the result of predation of aphids by natural enemies, while adverse weather conditions were not considered to contribute to this mortality. The role of parasitism was investigated by re-running the model with no mortality, residual mortality and total mortality. They found that parasitism was the main mortality factor when aphid densities were low but as their numbers increased the influence of parasitism declined. One puzzling feature of the model was a 40% (per QUIP) mortality factor applied to the number of first instar aphids. The authors confessed that they did not know the reasons for this value but suggested that it could be the result of a reduction in adult fecundity due to the effects of overcrowding.

Wiktelius & Petterson (1985) modelled the population dynamics of the bird cherry aphid *R. padi* to study the likely effects of plant resistance on aphid numbers. Sensitivity analyses of the model indicated that resistant plant genotypes should cause high nymphal mortality, prolonged development during early plant growth stages and low birth rate close to ear emergence. One major criticism of the study was that the model was not validated, no rigorous comparison of model predictions and field observations were made, the authors were satisfied that 'the trend....agrees well with that observed in the field' but did not produce evidence to support this claim. Although the use of the model satisfied the objectives of the study, to propose characteristics resistant cereal varieties should possess, any conclusions drawn from an non-validated model must be considered carefully.

3.3.2 Simulation Models of Virus Epidemiology

Although the usefulness of simulation modelling in plant virus epidemiology has been recognised for some time (Frazer, 1977) the approach has not been readily adopted, possibly because of the complexity of a virus-vector-host system (Carter, 1986). However, some models have been developed and this section reviews these (Table 2).

Gutierrez et al., (1974) used the simulation model of the cowpea aphid population dynamics to describe the epidemiology of subterranean clover stunt virus. Spread was summarized in one equation in that virus incidence was related to density of infective aphids. However, how they derived this equation was not mentioned and

Table 2. Summary of the review of simulation models of virus epidemiology.

Authors	Virus	Vector	Driving Variables	Comments
Gutierrez et al. (1974)	Subterranean clover stunt	*A. craccivora*	Temperature	Virus incidence related to vector density
Frazer (1977)	Alafalfa mosaic	*A. pisum*	Temperature	Educational model
Ruesink & Irwin (1986)	Soybean mosaic	Many aphid species	Temperature	Resistance strategies to virus and vectors
Kisimoto & Yamada (1986)	Rice stripe	*L. striatellus*	Temperature	Virus management schemes
Sigvald (1986)	Potato virus Y_o	Many aphid species	Temperature	Aphid migration critical
Kiritani (1979); Miyai, Kiritani & Nakasuji (1972)	Rice dwarf	*N. cincticeps*	Temperature	Construction data also used in validation
Sasabata et al. (1973)	Rice dwarf	*N. cincticeps*	Temperature	Chemical and natural control of vectors
Nakasuji et al. (1975)	Rice dwarf	*N. cincticeps*	Temperature	Vector efficiency investigated

although the aphid sub-system was rigorously validated, no mention of any comparison between predicted and observed virus incidence was made.

An 'educational' model of virus epidemiology was constructed by Frazer (1977). He combined an existing model describing the population dynamics of the pea aphid, *A. pisum* (Frazer & Gilbert, 1976) with a description of aphid movement and spread of alfalfa mosaic virus. The object of the study was not to produce a quantitative simulation result, but to stimulate interest in using systems analysis in virus epidemiology. Factors in the aphid model included a density-dependent quantitative function for morph determination, virus transmission success of an infectious aphid, and the number, and instars, of aphid forced to move by coccinellids.

The model was not validated against field data, but a sensitivity analysis was made to investigate the roles of several parameters in virus spread. Frazer (1977) found that if the latent period of the virus in the host plant was extended, or the probability of a vector successfully transmitting virus was reduced, then the rate of virus spread, not surprisingly, was lowered. Also if the number of coccinellids increased at low aphid densities then a reduction in rate of virus spread occurred, but if the number of coccinellids increased at high aphid densities then an increase in virus spread occurred as the coccinellids were causing more aphids to move than they consumed.

The epidemiology of soybean mosaic virus, a non-persistent virus spread by many aphid species, was modelled by Ruesink & Irwin (1986). They considered the importance of distinguishing between infection and inoculation of plants with virus - a plant can only be infected once, while it can be inoculated on many occasions. They predicted the number of daily infections using probability distributions. If the infections were randomly distributed in space then a Poisson distribution would be appropriate, but if they were clumped around a few foci then a distribution such as the negative binomial should be used. However, they did not have the field data to enable them to chose between distributions, or estimate their parameters, and so they calibrated the model by using both distributions and adjusted the values for the parameters until predictions fitted the field observations. They then re-ran the model to investigate the effect of hypothetical resistant cultivars on virus spread and yield. Doubling the number of moves an aphid made in a field reduced yield by 12% and caused the epidemic to occur 5 days earlier.

An important disease of paddy fields is rice stripe virus (RSV), different from other virus diseases of rice in that it is transmitted only by the small brown plant hopper, *Laodelphax striatellus* (Fallen). A model of RSV epidemiology was constructed by Kisimoto & Yamada (1986) which described the population infectivity of each generation of vector, acquiring virus both transovarially and from infected plants, and the proportion of plants infected with virus. Output from the model closely resembled field observations of virus incidence and a sensitivity analysis revealed that a reduction in vector density reduced the virus infection rate. Using this finding the authors proposed ways in which vector density could be reduced. These included the timing of transplanting with relation to vector migration, resistance of cultivars to RSV, thus reducing disease inoculum, and chemical control of the vectors.

Sigvald (1986) used a simulation model of potato virus Y_o (PVY_o) to forecast incidence. PVY_o is transmitted in a non-persistent manner by many aphid vectors. Inputs used in the model were the numbers of migratory alate aphid caught in yellow water traps, the transmission efficiency of vector species, plant maturity resistance, and the number of plants infected from seed potatoes. Output from the model fitted field observations satisfactorily, and so a sensitivity analysis was carried out. This indicated that the proportion of plants acting as virus sources was important as well as the number, and timing, of aphids migrating. If an inefficient vector's migration coincided with the period when the crop was susceptible then this was more important than an efficient vector's migration in a non-susceptible period.

An intensive study, using simulation models, of the epidemiology of rice dwarf virus (RDV) has been carried out since the early 1970s (See Kiritani, 1979; Miyai, Kiritani & Nakasuji, 1986 for reviews). The first model described the population dynamics of the green rice leafhopper (GRL), *Nephotettix cincticeps* (U.), by describing the changes in egg densities, numbers of newly hatched first instar nymphs and adults (Nakasuji & Kiritani, 1972). They related the proportion of infected rice hills to the proportion of infected vectors which was dependent on the acquisition of the virus from infected plants and included the adverse effects of the virus on its insect hosts. Output from the model closely resembled field observations of insect densities in the first to third generations and virus incidence in early sown crops, but did not predict accurately the number of insects in the fourth and fifth generations nor disease spread in late sown crops. However, it appears that the validation of the model was made against data used in its construction which reduces its explanatory usefulness.

A second model was constructed to include more development stages of GRL and the effect of predation by the spider, *Lycosa pseudoannulata* (B & S) (Sasabata et al., 1973). Numbers of spiders were thought to increase logistically and predation rate was dependent on predator densities. The effects of insecticide applications were investigated by considering two hypothetical chemicals. The first was assumed to kill 90% of the spiders and 10% of GRL. Model output indicated that GRL populations rose rapidly under these conditions. The second chemical was more selective so that it killed 10% of the spiders and 90% of the GRL. Simulations showed that GRL populations could be controlled within economical limits using this approach.

The previous model was updated by including the overwintering survival of fifth generation larvae (Sasabata & Kiritani, 1975). Survival rate was related, based on field observations, to the number of frost days in December to February, inclusive.

Nakasuji et al. (1975) combined the population model of GRL (Sasabata & Kiritani, 1975) with the model of RDV spread (Nakasuji & Kiritani, 1972) to study the influence of vector density and transmission efficiency of RDV from plant to vector, and *vice versa*, on the proportion of infected insects and plants. The greatest effect was observed by altering the transmission efficiency of the virus from the vector to the plant which resulted in exponential increase in infected hoppers and rice hills.

3.4 Choice of BYDV Model

One of the objectives of this study mentioned in Chapter 1 was to increase our understanding of the processes involved in BYDV epidemiology. By definition, the complexity of the coefficients in an analytical model constrain the understanding derived from use of such a model, and therefore this type of model was not chosen. Instead it was decided to develop a simulation model, enabling individual variables to be manipulated and allowing processes to be examined either individually or factorially.

Although stochastic models are more realistic, they are very expensive in computing time and resources, and do not necessarily improve the behaviour of the model (Gilbert & Hughes, 1971). Therefore, it was felt that any extra realism gained from using stochastic methods in the model would not repay the extra resources required to do so and, therefore, a deterministic simulation model was chosen.

3.5 Structure of Hypothetical BYDV Epidemiology Model

A simulation model of BYDV epidemiology has, essentially, four basic sub-systems (Figure 1). The first and last are data input and output, respectively. All the necessary data needed to run the model have to be entered at the start for a successful simulation. These should include information on the time period of the simulation, iteration step lengths and values of driving variables and sensitivity parameters. Output should include details of input to verify that the model is working as intended and has not corrupted the input data during the simulation resulting in any erroneous errors. It should also include daily values of different stages of the aphid population and virus infection status of the crop.

The second sub-system is concerned with the population dynamics of the aphid vector. It includes procedures describing crop colonization, development, survival, reproduction and emigration (Figure 1).

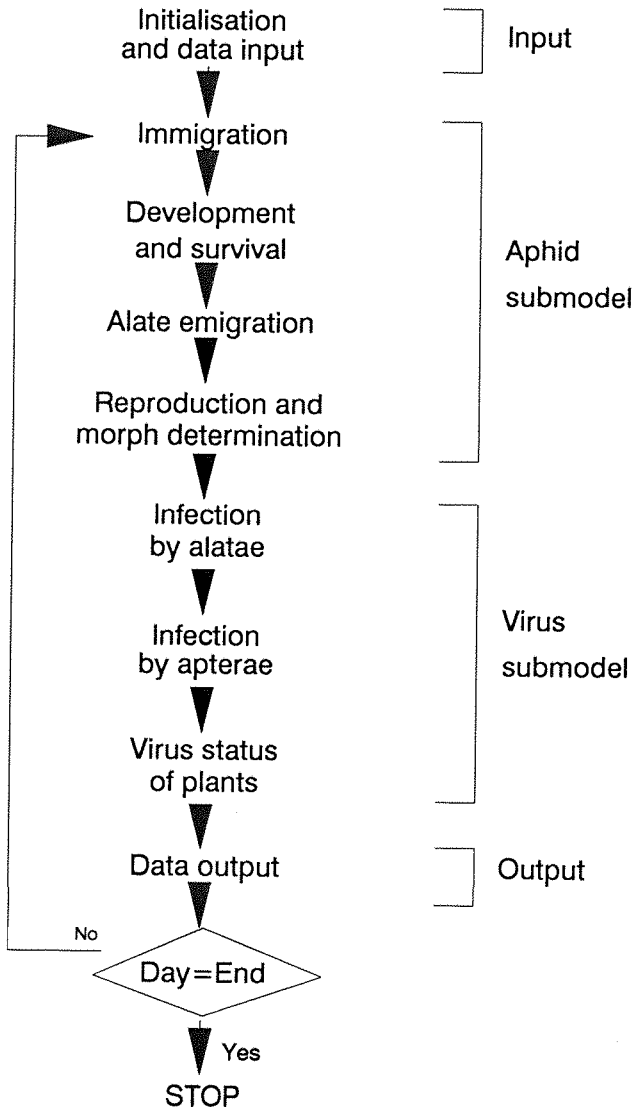
The virus sub-system describes the virus status of plants and vectors and the behaviour of different morphs of vectors and disease spread (Figure 1).

Not all interactions in the system need to be included into the two sub-systems, only those thought to be important. Experimentation with the model and the accuracy of its output will reflect on the wisdom of the choice of interactions included. Interactions, previously thought of as unimportant, may be included subsequently to satisfy the objectives of the study.

3.5 Conclusions

This chapter has shown how a simulation model can be developed and used to increase our understanding of a pest. However, before a model's predictions can be accepted its validity has to be rigorously tested. This procedure involves running the model under a large set of different conditions and comparing these with field observations collected under the same conditions. The field results should be independent of the data used in the construction of the model or its recommendations will be of less value. Also if output from the model differs greatly from the observed data the model should be re-reviewed and new variables and parameters, previously thought to be unimportant, should be considered. Fine tuning of the existing variables to improve the fit of the curves should not be made as this does not increase our knowledge of the underlying biology of the system.

Figure 1. Flow diagram of hypothetical BYDVV epidemiol ogy model.



4. Field Experiments

4.1 Introduction

Field studies were carried out to investigate the effect of crop sowing date on aphid colonization and subsequent population development, the incidence of BYDV and yield. Data from the studies were used to validate the model.

4.2 Methods

Field studies were carried out during the autumns and winters of 1985/6, 1986/7 and 1987/8, at Rothamsted. Four blocks of winter barley, cv Igri, were sown on five dates with two treatments, with and without insecticide application. Plots measured 3x10m in 1985/6 and 3x20m in 1986/7 and 1987/8 with a fallow path of 0.66m between each.

Aphid sampling began in September and continued throughout the autumn and winter at weekly intervals, weather permitting, until no aphids were found on the crop. In 1985/6 and 1986/7 four, 0.5m lengths of row were inspected per plot. In 1987/8 eight lengths per plot were inspected until mid-October when the number of plots that had emerged made this impractical; from then, four lengths of row were inspected. The number of plants in each length of row and the crop growth stage for each plot were recorded. Any aphids found were identified and, in the first autumn, the number of 1-3rd instar nymphs, apterous and alatiform 4th instar nymphs, and apterous and alate adults were recorded. In the two later autumns all nymphs were grouped together. The numbers of mummified, parasitised by *Aphidiidae*, and diseased, by *Entomophthorales*, aphids were also recorded.

BYDV incidence was assessed visually in April by estimating the percentage of plants with virus symptoms per plot. In addition, ELISA tests were done on the last fully expanded leaves of 25 plants per plot, selected at random.

4.3 Results and Discussion

Four aphid species were found in the samples, *R. padi*, *R. maidis*, *R. insertum* and *S. avenae*. *R. padi* was the most common in the first two years with *S. avenae* the most common in the last year.

Immigration occurred during September and October, with the largest number of alate *R. padi* caught in the Rothamsted suction trap in 1985 and the least in 1987 (Figure 2).

The population of *R. padi* increased rapidly in 1985, while in 1986 and 1987 the growth rate was slower. Higher temperatures and lower rainfall early in the 1985 season (Figure 3) were probably responsible for the more rapid population growth and high peak population density in that year (Figure 4).

The decline in numbers of aphids in each winter was probably the result of declining temperatures (Figure 3) and by the end of January no aphids were found in plots in any of the three winters (Figure 4). In 1985 alate aphids were found in the crop in December (Figure 2) and could have contributed to the decline in aphid numbers if they emigrated, but this seems unlikely with the low temperatures possibly inhibiting flight.

Parasitised and diseased aphids were most common in 1985 (Table 3), probably because of the warmer, drier weather in 1985 (Figure 3) and the higher aphid densities (Figure 4). *Aphidius rhopalosiphi* was most common, while *A. picipes* and *A. ervi* were also found. The hyperparasites *Alloxysta* spp. and *Phoenoglyphis* spp. were also found (Powell, pers. comm.).

Figure 2. Rothamsted Insect Survey (RIS) 12.2m suction trap catches of *Rhopalosiphum padi*.

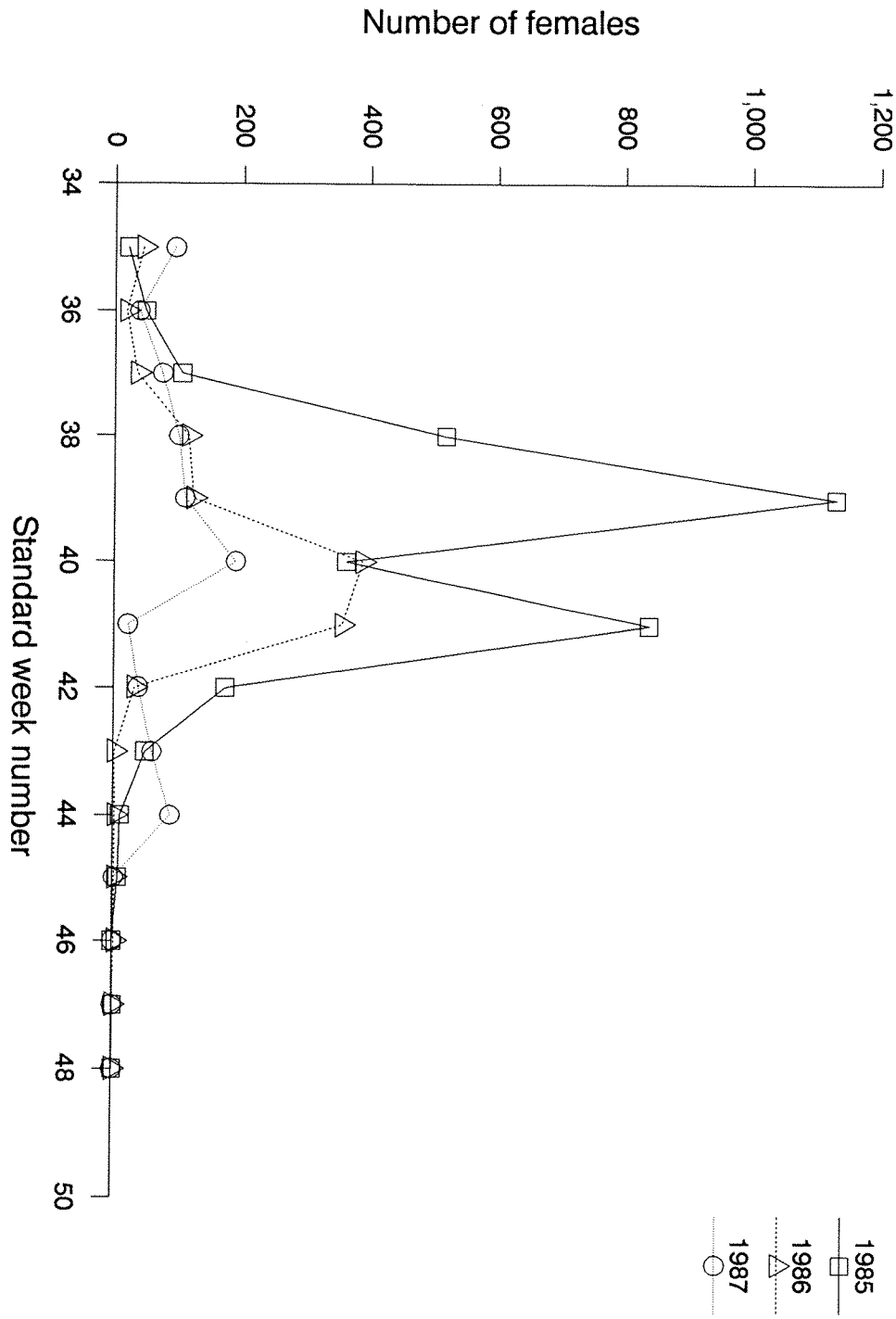


Figure 3. Daily temperature and rainfall measurements at Rothamsted.

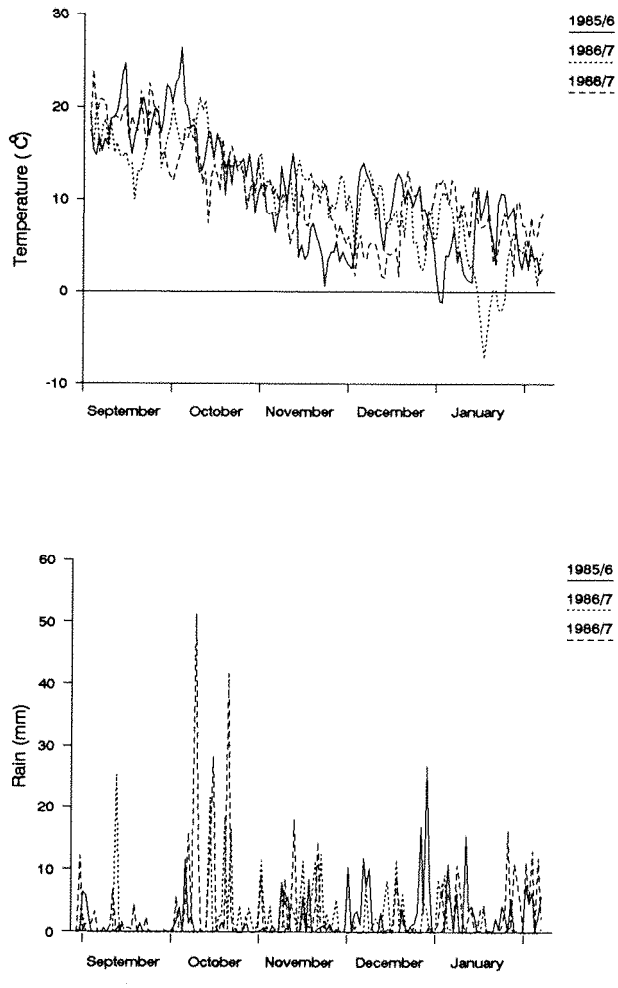


Figure 4. Population devel opment of *R. padi* on winter barley at Rothamsted.

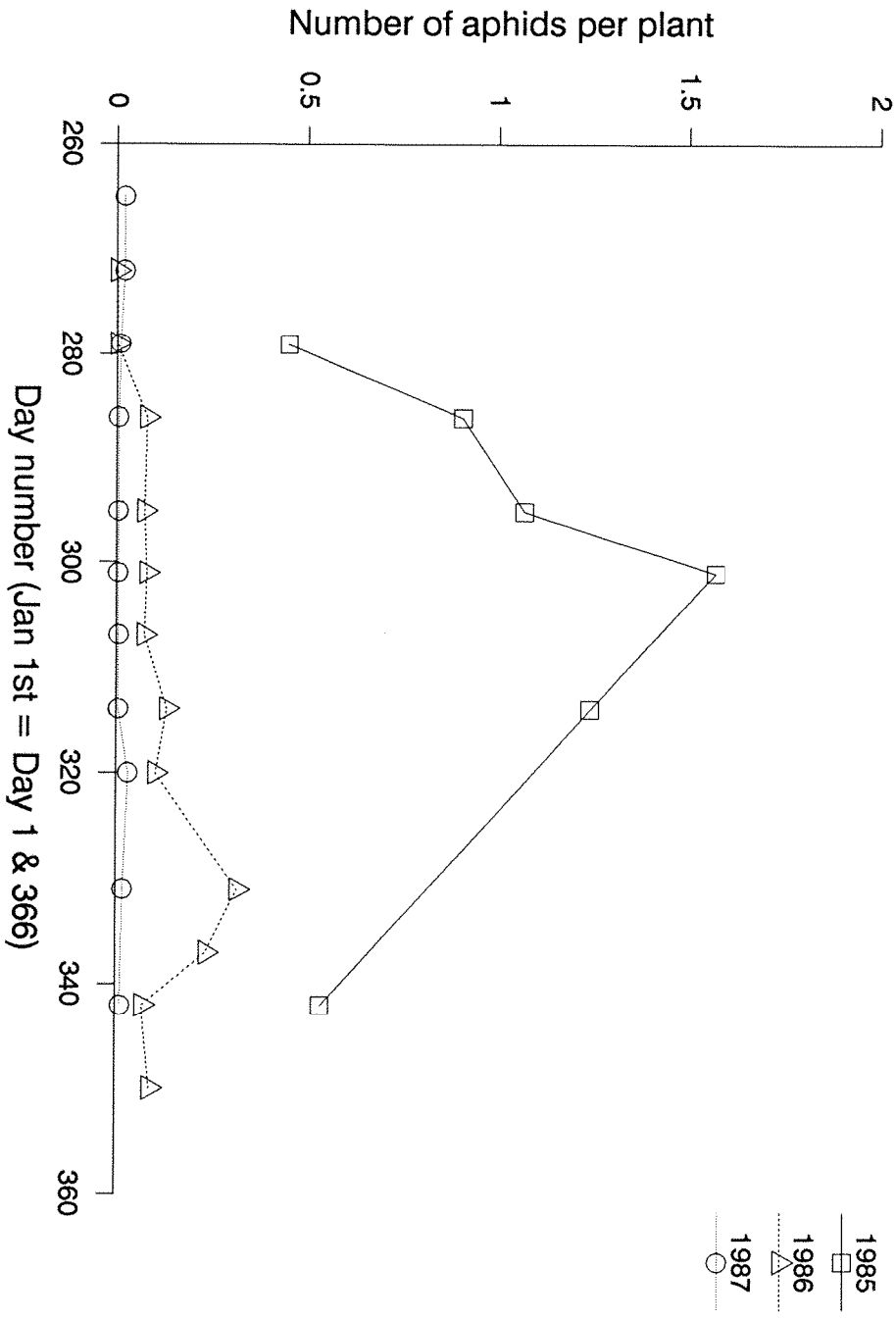


Table 3. Numbers of mummified and diseased aphids found on the first sown plots.

	1985		1986		1987	
Date	Fungals	Mummies	Fungals	Mummies	Fungals	Mummies
7/10	0	0	0	0	0	0
14/10	9	2	0	0	0	0
22/10	6	7	0	0	0	1
29/10	22	14	0	0	0	0
3/11	-	-	0	0	0	0
11/11	2	11	0	0	0	0
17/11	-	-	1	0	0	0
26/11	-	-	0	0	0	0
8/12	0	0	0	0	0	0

5. Aphid Population Dynamcics Subsystem

The aphid subsystem consists of five sections :
1. Initialisation and data input.
2. Immigration.
3. Development and survival.
4. Reproduction and morph determination.
5. Output.

5.1 Initialisation and Data Input

The arrays used in the program were dimensioned and their elements set to zero. The necessary data to run the model were entered into the program. The parameters were:
(i) start and finish days of the simulation (1st January Year i=day 1 and 1st January Year i+1=day 366);
(ii) sensitivity factors for altering parameters and variables, in a standard simulation. These were set to zero or one;
(iii) daily maximum and minimum temperatures (^{o}C);
(iv) plant density (plants/m^2);
(v) start and finish days for alate immigration; and
(vi) daily estimates of numbers of virginoparae caught in a suction trap.

5.2 Immigration

5.2.1 Submodel

It was assumed that any *R. padi* found on the crop originated from immigrant alate aphids. Carry over of aphids from the previous cereal crop or its grass weeds was not considered.

The numbers of alate *R. padi* colonizing a cereal crop were estimated from the numbers caught in the nearest RIS 12.2m suction trap (Taylor, 1983). The number of alate *R. padi* was corrected by subtracting the number of males and an estimate of the number of gynoparae in the sample, as these were returning to their primary host, *P. padus*, and so are probably unimportant in the epidemiology of BYDV (Plumb, 1983).

The number of alates landing per plant was calculated by multiplying an estimate of the number of virginoparae caught in the nearest suction trap by a deposition factor.

It was assumed that alates landing on the crop were reproductively mature and remained on the crop until they had died.

Figure 5 is the relational diagram for immigration.

5.2.2 Data

It is possible to distinguish between males and female alates morphologically but not between gynoparae and virginoparae. However, it is possible to differentiate between these behaviourially, as gynoparae do not readily reproduce on cereals (Dixon, 1971). Data from infectivity trials (carried out by the Plant Pathology Department at Rothamsted (Plumb, Lennon & Gutteridge, unpublished results) were used to estimate the proportion of virginoparae in suction trap samples in order to simulate the 1985/6 field results. In these trials live aphids caught in a 1.5m suction trap were placed individually on oat seedlings and those which reproduced were assumed to be virginoparae. Results from host choice experiments in 1986/7 and 1987/8 (Tatchell, Plumb & Carter, 1988; Tatchell, unpublished; respectively) were used to estimate the proportions of viginoparae by recording the number that fed and reproduced on barley leaves given the choice of doing so on barley or *P. padus* leaf discs.

The mean density of aphids flying (D) decreases linearly with height (Z) (Taylor & Palmer, 1972);

$$\log_{10}(D)=a+b*\log_{10}(Z)$$

As the density height gradient (b) is unknown for *R. padi* it was assumed to be -1.0, an average value for aphids (Taylor & Palmer,1972). The other constant, a, is influenced by the flight time which is also unknown but was assumed to be 2h, the average flight time for aphids in south-east England (Taylor & Palmer, 1972). Taylor & Palmer (1972) calculated the deposition rates, assuming random deposition, for several combinations of density height gradients and flight times (Table 4). With the assumed values for *R. padi*, for each aphid sampled in a suction trap 237 would land per hectare.

5.3 Development and Survival

Three versions of the development process of the submodel were developed.

5.3.1 Version 1

5.3.1.1 Submodel

Development of aphids is dependent on temperature (Kitching, 1977). The stage of development of an aphid can, therefore, be

Figure 5. Flow diagram of immigration and crop colonisation.

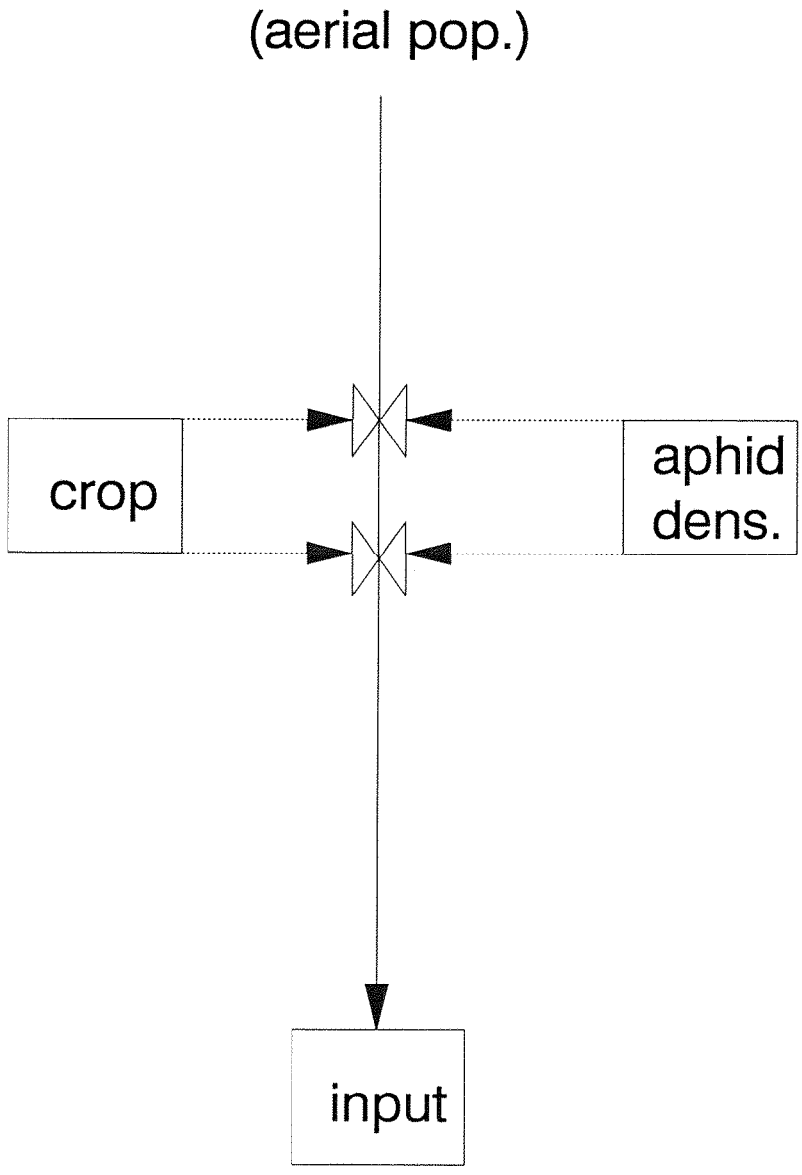


Table 4. Number of aphids landing (ha^{-1}) equivalent to one aphid caught in a Rothamsted Insect survey 12.2m suction trap (After Carter, Rabbinge & Dixon, 1980).

Density gradient	Mean flight time (hrs)				
b	0.5	1	2	4	8
0	10309	5154	2577	1288	644
-0.5	1659	829	414	207	103
-1.0	948	474	237	118	59
-1.5	2014	1007	503	251	125
-2.0	10309	5154	2577	1288	644

calculated using temperature-time summation by estimating the temperature-time above the development threshold measured in hour- or day-degrees (D^{o}). In the submodel, each aphid instar was represented by a two-dimensional array, the first row of each was for the number of aphids in each age-class and the second for their ages, in day-degrees (Carter et al., 1980). Aphids of the same age developed at the same rate and were moved from one array element to the next at each iteration. Updating of the arrays began with the oldest age-class of the adult instar and ended with the youngest age-class of the first instar. The number of aphids in the new Ith age-class was the product of the number in the I-1th age-class and the proportion surviving that day. The age of the Ith class (in D^{o}) was the sum of the age of the I-1th class and the number of day-degrees for that day. The age of the Ith class was checked against the limit for that instar. If it exceeded the limit the aphids in that class were, either, moved into the vacant first age-class of the next oldest instar, their ages set to zero and the original array elements zeroed, or, if they were adults, they were removed from the population. It was assumed that alatiform fourth instar nymphs emigrated on moulting to the adult stage and were therefore a net loss to the population.

5.3.1.2 Data

Dean 1974 studied the development time of *R. padi* at different temperatures, rearing the aphids on barley-leaf discs (Hughes & Woolcock, 1965). The relationship between development rate (DEVRATE), the reciprocal of development time, and temperature (TEMP) is linear over the range 10-25^{o}C (Figure 6)

$$DEVRATE=-0.0254+0.0092*TEMP \quad r=0.997 \quad d.f.=3 \quad p<0.001$$

the equation gives a development threshold of 2.8^{o}C (when the development rate is zero), which was used to calculate the duration of each instar in day-degrees (Table 5). The duration in day-degrees was calculated by subtracting 2.8 from the experimental temperatures and multiplying the result by the duration, in days, of each instar. It was assumed, based on studies of the cabbage aphid, *Brevicoryne brassicae* (Hughes, 1963), that fourth instar alatiform aphids took 1.5 times longer to develop than apterous fourth instar nymphs. Adult longevity differed between morphs, 229.4 day-degrees for alate adults (Dean, 1973) and 224.0 day-degrees for apterous adults (Dean, 1974).

Survival rates were based on field samples of aphid populations on barley plots at Rothamsted in 1985/6. Naturally occurring aphid populations were sampled on a regular basis (See Chapter 4). If the

Figure 6. The effect of temperature on the development rate of *R. padi* (Dean, 1974).

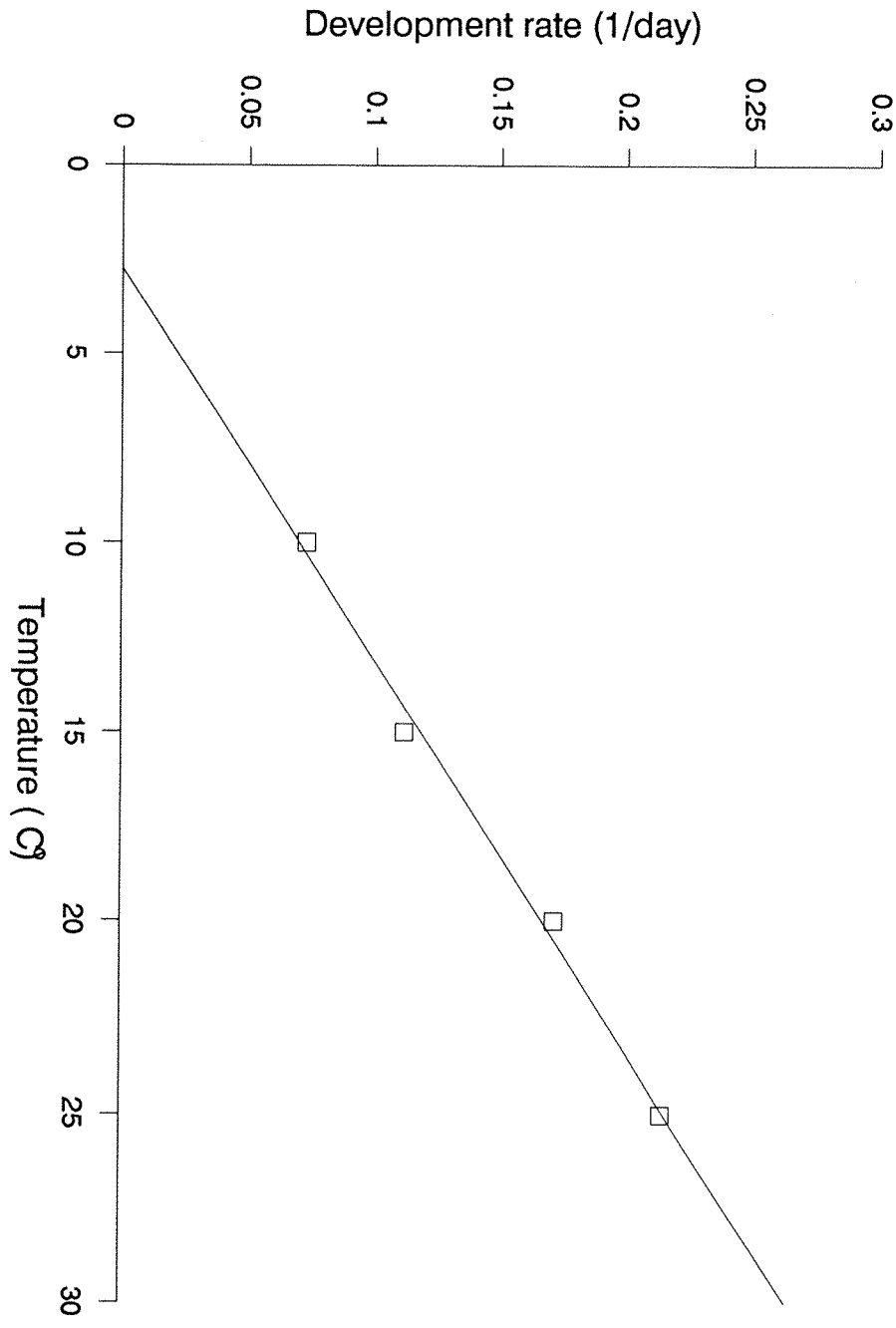


Table 5. Duration (h^{o}) of the nymphal instars (roman numerals) and pre-reproductive delay (PRD) at various temperatures (After Dean, 1974).

Temp. (^{o}C)	I	II	III	IV	PRD
10	668.9	545.8	525.6	627.8	133.2
15	712.5	643.4	649.0	619.8	120.8
20	648.4	565.9	529.8	662.2	123.8
25	654.9	572.8	581.6	677.1	179.8
Mean	676.2	579.7	571.5	646.7	139.4
s.e.	15	19.1	28.8	13.7	13.7
Acc. Daydeg.	28.2	52.3	76.1	103.1	108.9

population declined between two successive samples then a theoretical aphid density for the second sample date, assuming logarithmic growth from the first sample date, was calculated. The difference between the theoretical and observed densities (SURV) was related to the number of day-degrees below 2.8°C (DDB);

$$SURV=0.9511-0.0173*(DDB) \quad r=0.703 \quad d.f.=15 \quad p<0.01$$

5.3.1.3 Appraisal of Version 1

The development rate of aphids is not linearly related to temperature at extreme values and errors can occur in calculating the development rate, on a physiological time-scale, at these extremes (Stinner, Gutierrez & Butler, 1974). This is the result of the day-degree method failing to take into account 'pre- and post-threshold' development (Baker, 1980) produced by the non-linear relationship between development rate and temperature above and below the upper and lower theoretical thresholds, respectively. The approach, therefore, becomes inappropriate.

5.3.2 Version 2

5.3.2.1 Submodel

The proportion of development (PD) experienced by an aphid was calculated each day using Simpson's rule (Barlow & Dixon, 1980);

$$PD=(DR_{max}+DR_{min}+DR_{mean})/3.0$$

where DR_{max}, DR_{min} and DR_{mean} are the development rates at the daily maximum, minimum and mean temperatures, respectively. Logistic functions were fitted to calculate the development rate (DR) at each temperature;

$$DR = \frac{A+C}{(1+\exp(-B*(TEMP-M)))}$$

where A, B, C and M are constants and TEMP is either the daily maximum, minimum or mean temperatures.

The updating of arrays was as used in Section 5.3.1.1 except age was expressed as a proportion of total development rather than in day-degrees.

5.3.2.2 Data

The results of Dean (1974), who investigated the effect of different temperatures on the development rate of *R. padi* over the range 10-25°C (See Section 5.3.1.2), were used with the assumption of zero development rate at -4°C to fit a logistic relationship between temperature and development rate. The assumption of a nil development rate at -4°C derives from the results of Williams (1980) who found that all *R. padi* kept in glass tubes in a waterbath for 90mins at -4°C were killed.

5.3.2.3 Appraisal of Version 2

Each instar was represented by a two-dimensional array. After updating, the procedure checked if the proportion of development of a class had exceeded the limit for that instar. If it had, the aphids were moved into the first age-class of the next oldest instar and their age set to zero, provided they were nymphs. However, this resulted in errors as some development was lost in the calculations. For example, if the age of a class on day i-1 was 0.98 and the proportion of development on day i was 0.10, then the new age on day i was 1.08, exceeding the limit of 1.00, so the aphids were put into the next oldest instar, their ages set to zero and 0.08 of their development was lost.

5.3.3 Final Version

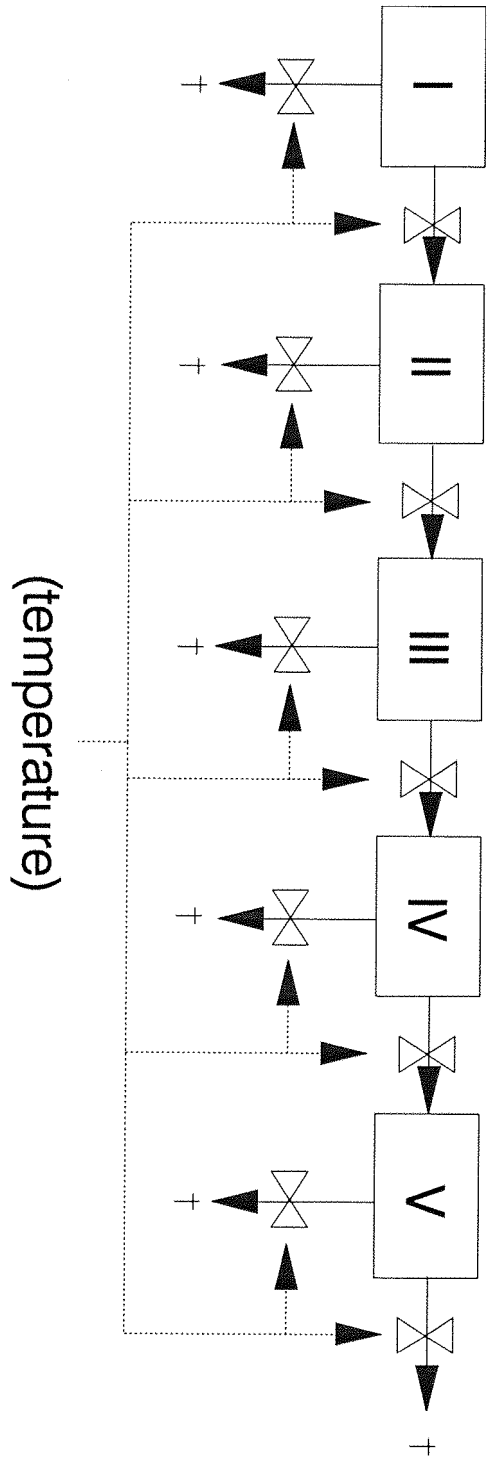
Individual arrays for each instar of both morphs were replaced by four, larger two-dimensional arrays, one for each morph of nymphs and one for each morph of adults. Fourth instar nymphs became adults if the proportion of development exceeded 1.00. They were moved into the first age-class of the corresponding morph of adult and their ages were set to zero. Values of proportion of development between 0.00 and 1.00 determined which instar the nymphs were in. Development rates were calculated using logistic functions and proportions of development using Simpson's rule (See Section 3.3.2.1). Updating followed the procedure used in the two earlier versions.

Figure 7 is a relational diagram of development and survival.

5.3.3.1 Data

Total development times, from birth to first reproduction (Dean, 1974) and a development threshold of -4°C (Williams, 1980) were used to calculate the development rates using logistic functions. The cumulative proportion of development was calculated by

Figure 7. Relational diagram for development and survival.



expressing the duration of each instar as a proportion of total development time at different temperatures. The mean proportions were calculated for each instar and accumulated from the first nymphal instar (Table 6).

5.4 Reproduction and Morph Determination

Two versions of the reproduction process were developed.

5.4.1 Version 1

5.4.1.1 Submodel

It was assumed that immigrant alate aphids were reproductively mature as soon as they landed on the crop, while apterous adults had to undergo a pre-reproductive period between moult to the adult stage and birth of first offspring. It was assumed that reproductive rate was dependent on temperature and adult morph, as had been found for *S. avenae* and *M. dirhodum* (Wratten, 1977).

Nymphs produced by both alate and reproductively-mature apterous adults were summed and their morph determined at birth (Dewar, 1977). Morph determination was dependent on total aphid density, such that as density increased the proportion of newly laid nymphs that developed into alates increased.

5.4.1.2 Data

Dean (1974) studied the reproductive rate of apterous adult *R. padi* at different temperatures, keeping the aphids on barley-leaf discs (Hughes & Woolcock, 1965). The relationship between reproductive rate (REPRAT) and temperature is linear over the range 10-20°C (Figure 8)

REPRAT= -1.2867+0.3455*TEMP r=0.986 d.f.=2 $p<0.02$

This equation gives a reproductive threshold of 3.7°C, which was used to calculate the reproductive rate in day-degrees (Table 7). Wratten (1977) found that apterous adult *S. avenae* and *M. dirhodum* are about 1.3 times more fecund than alate adults of the same species. Therefore, the reproductive rates of apterous adult *R. padi* were divided by 1.3 to give the rates for alate adults.

Morph determination in aphids is dependent on aphid density and crop growth stage (Watt & Dixon, 1981). However, the crop growth stages found to influence morph determination in *R. padi* (Leather &

Table 6. Proprotion of daily development of nymphal instars (roman numberals) and pre-reproductive delay (PRD) at various temperatures (After Dean, 1974).

Temp. (°C)	I	II	III	IV	PRD
10	0.26	0.21	0.21	0.25	0.05
15	0.26	0.23	0.24	0.23	0.04
20	0.26	0.22	0.21	0.26	0.05
25	0.25	0.22	0.22	0.26	0.07
Mean	0.26	0.22	0.22	0.25	0.05
s.e.	0.003	0.004	0.007	0.008	0.008
Acc. prop.	0.26	0.48	0.70	0.95	1.00

Figure 8. The effect of temperature on the reproductive rate of *R. padi* (Dean, 1974).

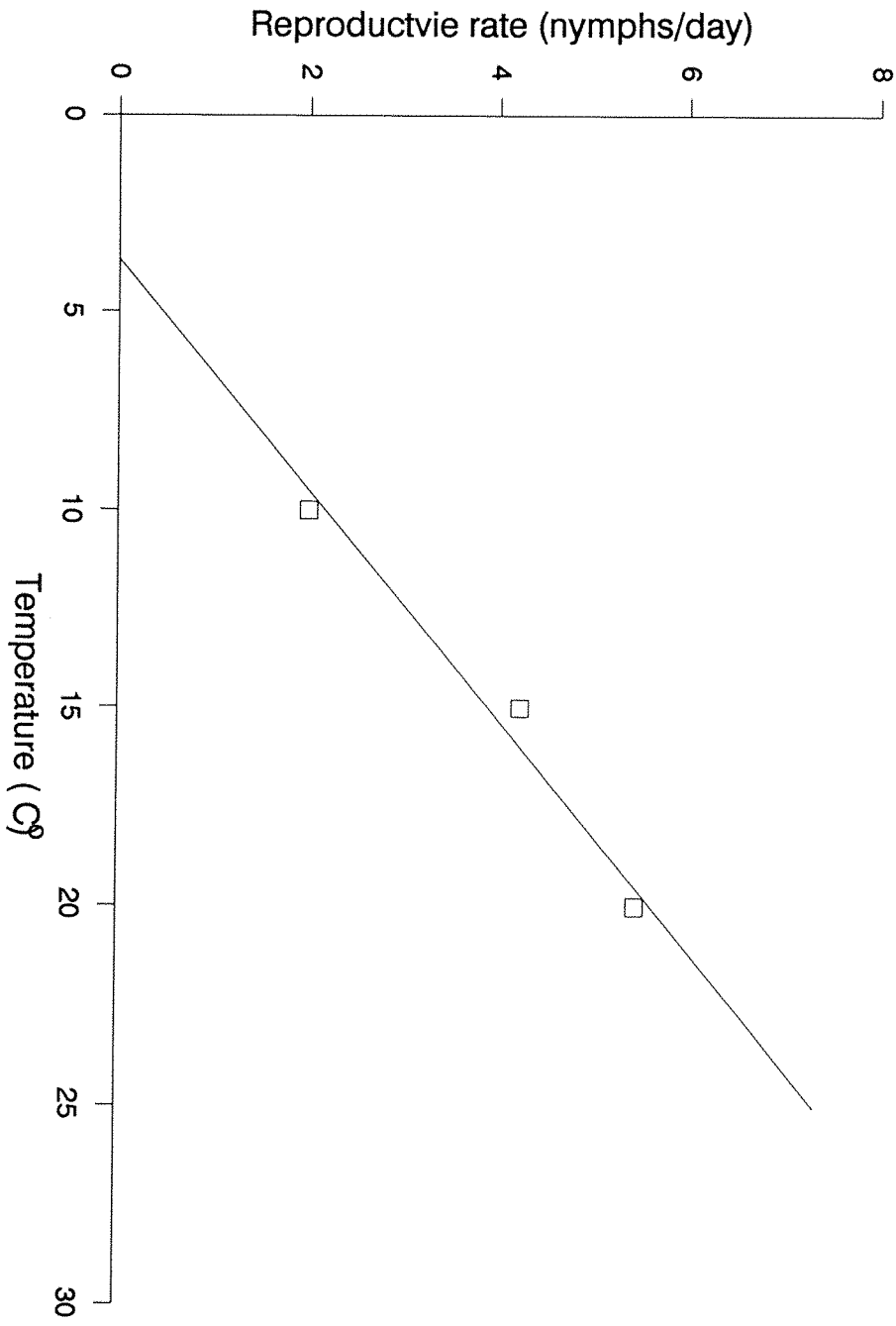


Table 7. Fecundity (nymphs/day) at various temperature (After Dean, 1974).

Temp. (°C)	D^{o}/D	Rep. life (D)	Rep. life (D^{o})	Nymphs (N)	Rep. rate (N/D^{o})
10	6.3	15	94.1	30	0.32
15	11.7	13	146.6	55	0.38
20	16.3	11	179.0	60	0.34
Mean					0.34
s.e.					0.02

Dixon, 1986) did not occur in the autumn and winter and so this effect was not considered in the model. Rautapää (1976) found that the proportion of nymphs that became alate adults (ALPROP) was related to total aphid density (TOTDEN)

$$ALPROP=0.0590+0.0054*(TOTDEN) \quad r=0.909 \quad d.f.=4 \quad p<0.02$$

5.4.1.3 Appraisal of Version 1

Version 1 assumed that the reproductive rate of aphids was linearly related to temperature. However, like development, this is unlikely to be true at the extremities of the relationship. Therefore, it would be more realistic to use a curvilinear expression to describe the relationship.

The linear relationship between aphid density and the proportion of alatiform nymphs implies that as density increases indefinitely so does the proportion so that it can theoretically exceed 1.0. One way to overcome this would to 'plateau' the relationship at high densities by using another regression equation with a slope of 0.0. However, it would be more realistic to use one curvi-linear expression that describes both the linear and non-linear sections of the relationship.

5.4.2 Final Version

5.4.2.1 Submodel

The mean reproductive rate was calculated each day using the average of the reproductive rate at the maximum, minimum and mean temperatures (Simpson's Rule). These rates were calculated by fitting logistic functions between the reproductive rate and temperature.

The proportion of nymphs that became alatiform was calculated using a logistic function between proportion of alatiform nymphs and total aphid density.

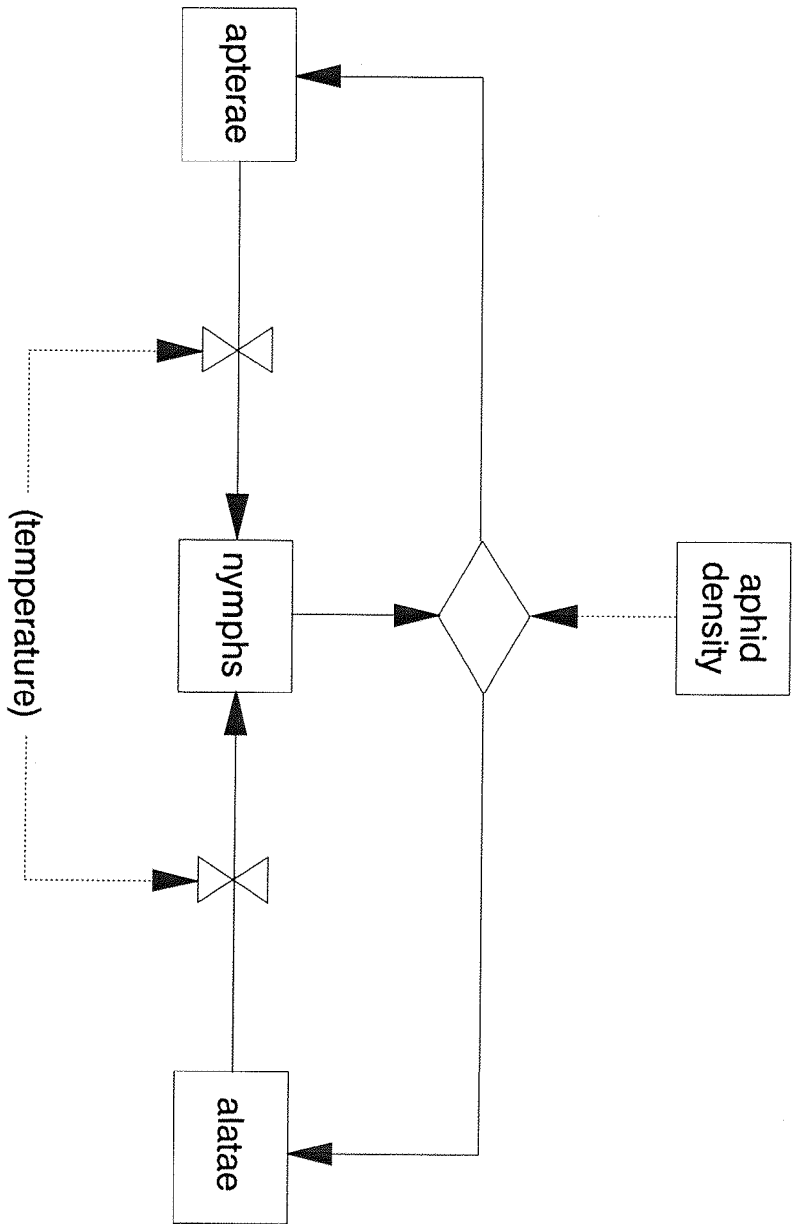
5.4.2.2 Data

As all *R. padi* exposed for 90mins at -4°C are killed (Williams, 1980) it was assumed that the reproductive rate at this temperature was zero. This assumption was used with the data of Dean (1974), as used in Section 3.4.1.1, to fit a logistic function between reproductive rate and temperature.

Morph determination was assumed to be dependent upon total aphid density. A logistic function was fitted to describe the relationship between aphid density and the proportion of alatiform nymphs, using the data of Rautapää (1976), as used in Section 5.4.1.1.

Figure 9 is a relational diagram for reproduction and morph determination.

Figure 9. Relational diagram for reproduction and morph determination.



6. Model Predictions of Aphid Population Dynamics

6.1 Introduction

The aphid population dynamics sub-system of *R. padi* was verified using internal computer checks and a comprehensive examination of the program listing.

It was decided to validate the model by comparing output from the model with field results using graphical analysis. The model was thought to be sufficiently accurate if its predictions fell within one standard error of the observed result. (The vertical bars on all graphs are one standard error of the observed mean). Since, on most sampling dates, 95% of the total numbers of aphids found in the field were nymphs it was decided not to compare model output of number of nymphs with that observed but to use the comparisons of total aphid densities to draw conclusions on the behaviour of nymphal instar numbers.

6.2 Results

6.2.1 1985/6 : Sowing Date 13 Sept.

The predicted population curve was similar to that observed although the model predicted the peak population density 10 days after the observed (Figure 10). A second large peak was predicted about 6 weeks after the population peak but, unfortunately, because of the sampling programme used field data were not collected during this period and so the peak could not be validated. The model also predicted that some *R. padi* overwintered viviparously, but no aphids were found in the field. This may be the result of the model not taking into account the effect of extreme low temperatures, less than $-4^{o}C$ (Williams, 1980), on aphid survival. The peak number of alate aphids observed in the field was 4.5 times greater than that predicted by the model, although the timing was similar (Figure 11). The model also failed to predict the low number of alates found on the crop throughout the sampling schedule. Either, the alates had a longer longevity than assumed for in the model, or, more likely, those found on the crop were emigrants, induced by overcrowding, but temperatures were too low for them to fly from the crop. The model assumed that as soon as alatiform fourth instar nymphs moult to the adult stage they leave the field irrespective of temperature. The model predicted the build-up of apterous adult aphids accurately but overestimated the population peak by about 2.5 times (Figure 12). This implied that apterous adults probably lived for a shorter length of time than allowed for in the model. However, if the longevity of

Figure 10. Observed and predicted total numbers of *R. padi* per plant, 1985/6, sowing date 13 Sept.

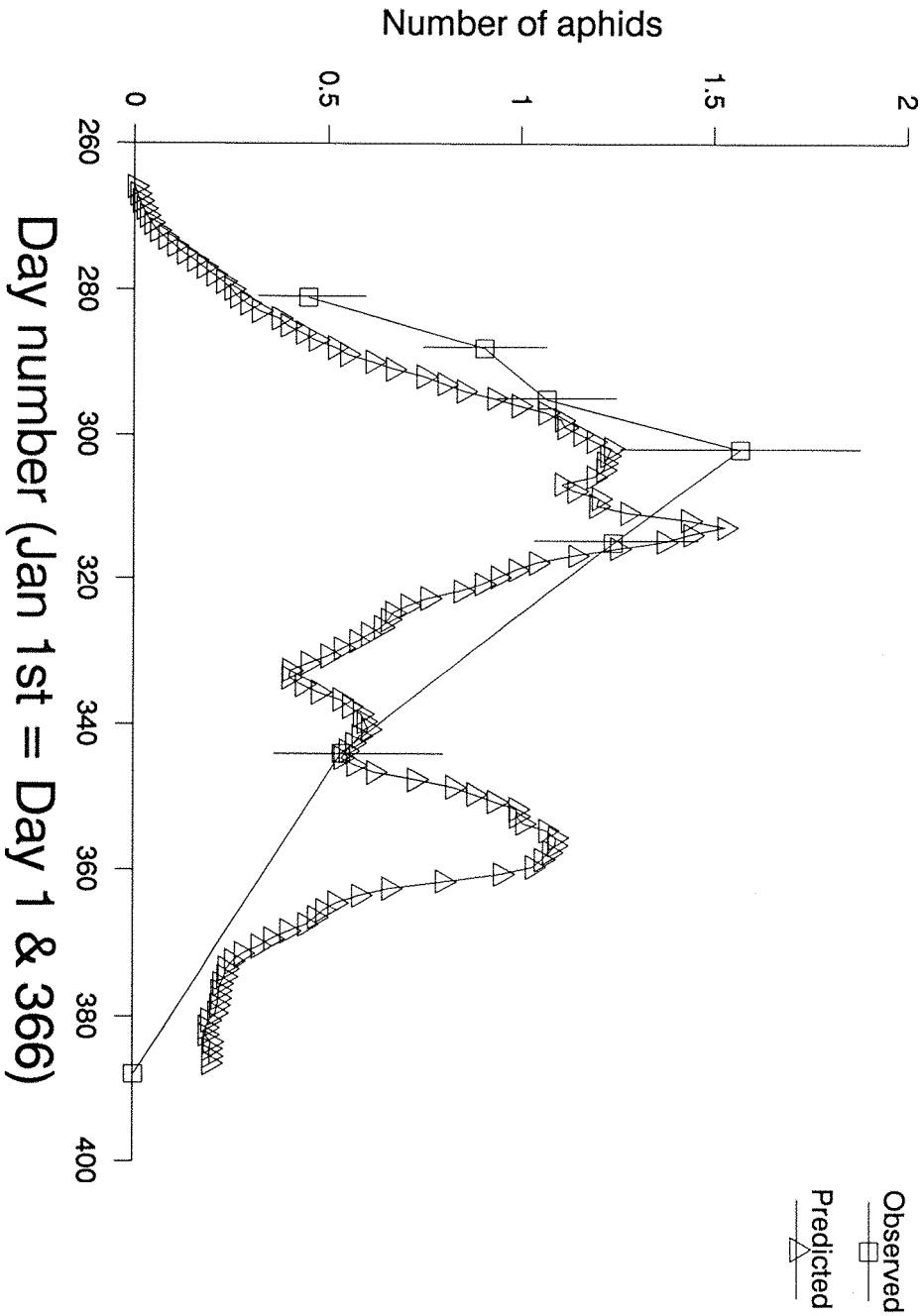


Figure 11. Observed and predicted numbers of alate adult *R. padi* per plant, 1985/6, sowing date 13 Sept.

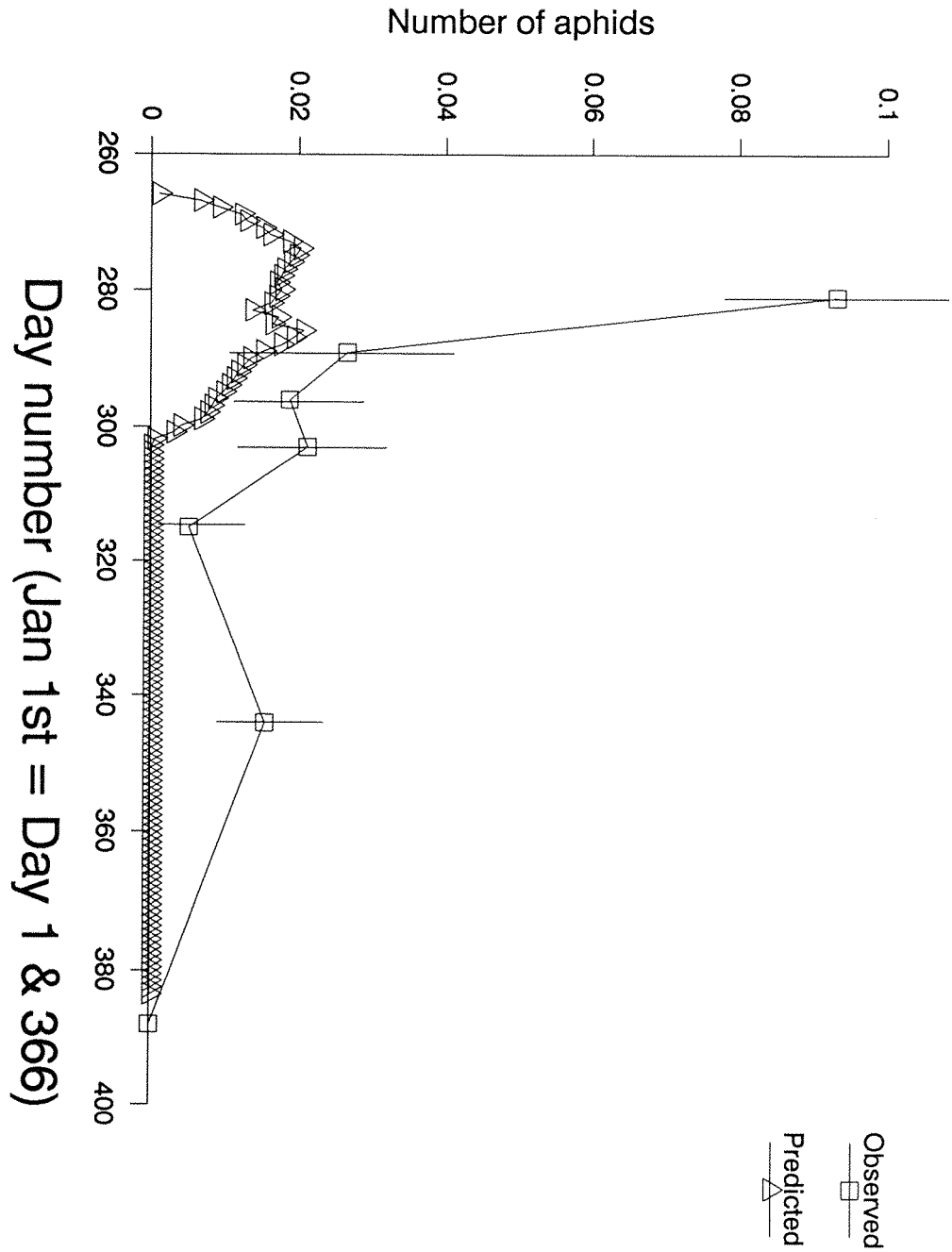
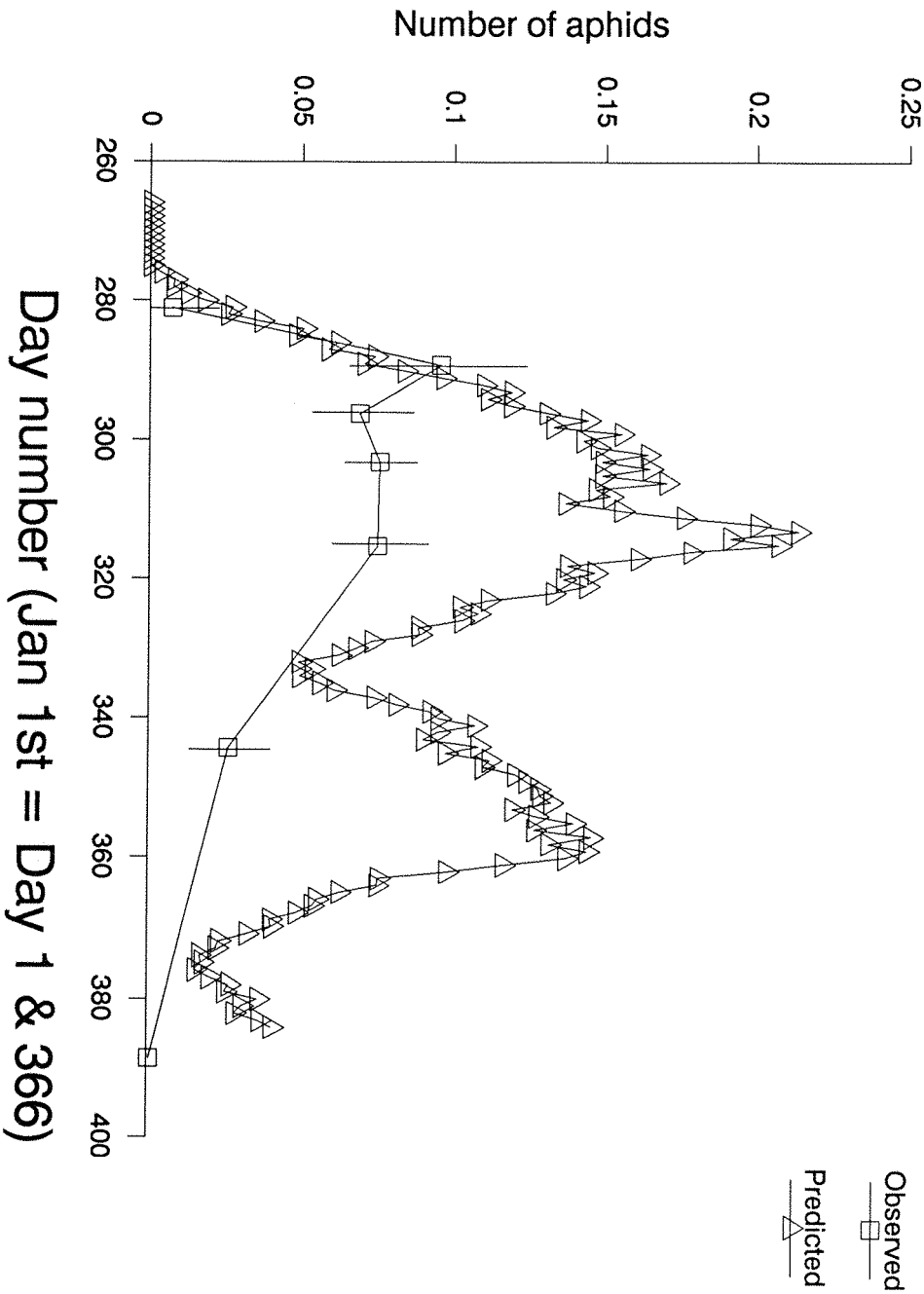


Figure 12. Observed and predicted numbers of apterous adult *R. padi* per plant, 1985/6, sowing date 13 Sept.



apterous adults was decreased in the model this would have to be compensated for by an increase in the survival rate of newly-born nymphs.

6.2.2 1985/6 : Sowing Date 23 Sept.

Although the shape of the predicted population curve was similar to that observed, the predicted peak density was about 3 weeks late (Figure 13). This delay in the timing of the model's predictions was possibly the result of the model underestimating the peak numbers of alates found on the crop although the shape of the predicted curve was similar to that observed (Figure 14). This underestimation could imply that alates live longer than the value used in the model although this was based on a difference, between observed and predicted densities, of about 1.5 alates per square metre. Although the shape of the predicted number of apterous adults was similar to that observed its timing was about 2 weeks too late and the peak was 1.5 times less than the observed (Figure 15). This implies that apterous adults probably live longer than was assumed in the model.

6.2.3 1985/6 : Sowing Date 4 Oct.

Because of the sampling programme used very few samples were taken of the third sowing which must be considered when drawing conclusions from the validation of the model. The model predicted the curve of the observed population accurately (Figure 16). It described precisely the rapid increase in aphid numbers to a peak density and the subsequent decline, although it predicted that aphids would survive the winter viviparously in the field. The shape of the predicted curve of alates was similar to that observed but the peak density occured about 10 days after that found in the field (Figure 17). The shape of the predicted curve of numbers of apterous adults was similar to that observed although the peak density was slightly overestimated (Figure 18).

6.2.4 1986/7 : Sowing Date 12 Sept.

The predicted population curve was similar to that observed although the peak density was predicted 2 weeks after the observed (Figure 19). The model did not predict the sharp decline in aphid numbers after the peak as was found in the field. This implies that other factors, apart from low temperatures, were reducing the survival rates of aphids in the field below those used in the model. These factors could be biotic (the action of natural enemies) or abiotic (heavy rainfall and high winds). The absence of aphids in the

Figure 13. Observed and predicted total numbers of *R. padi* per plant, 1985/6, sowing date 23 Sept.

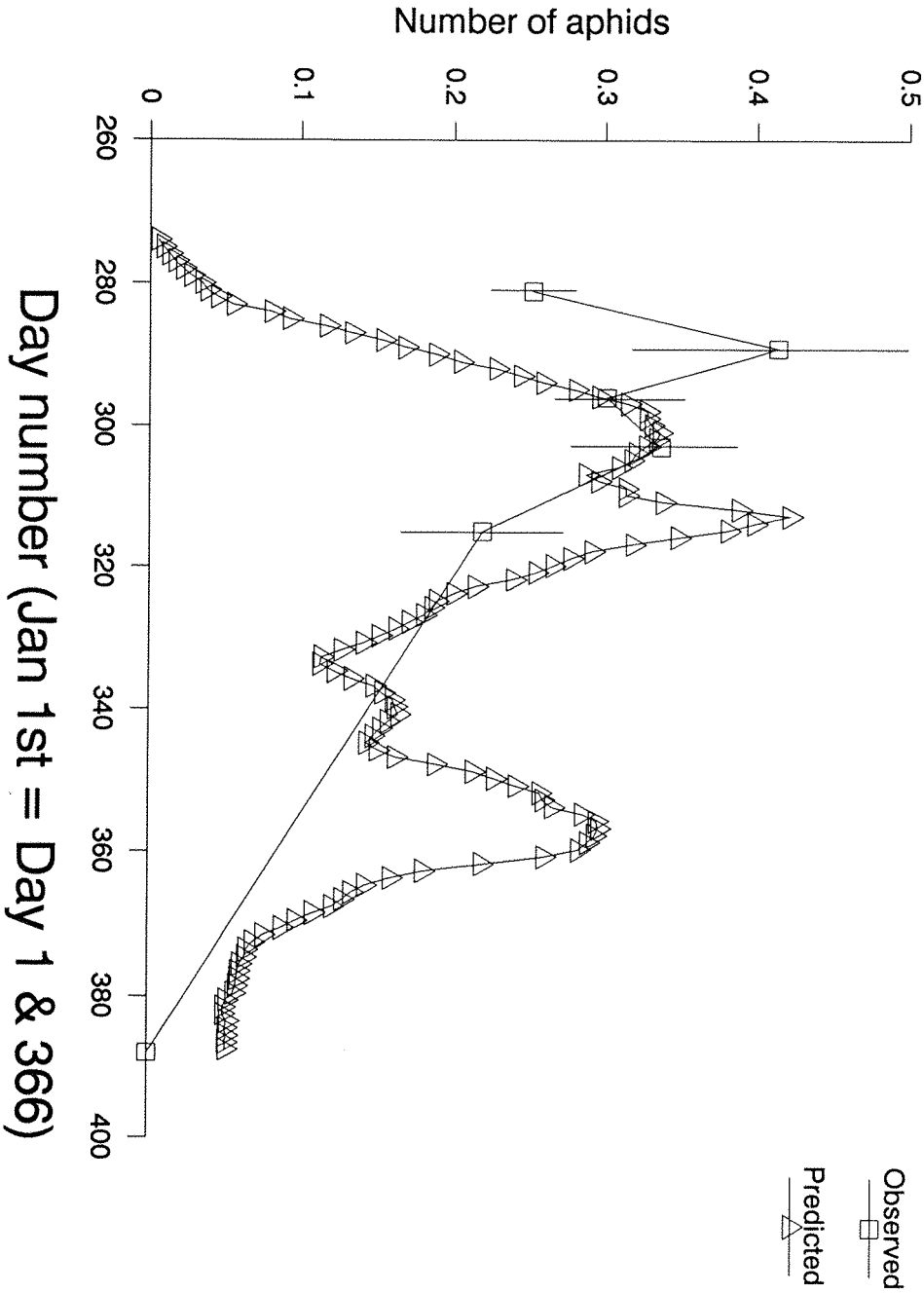


Figure 14. Observed and predicted numbers of alate adult *R. padi* per plant, 1985/6, sowing date 23 Sept.

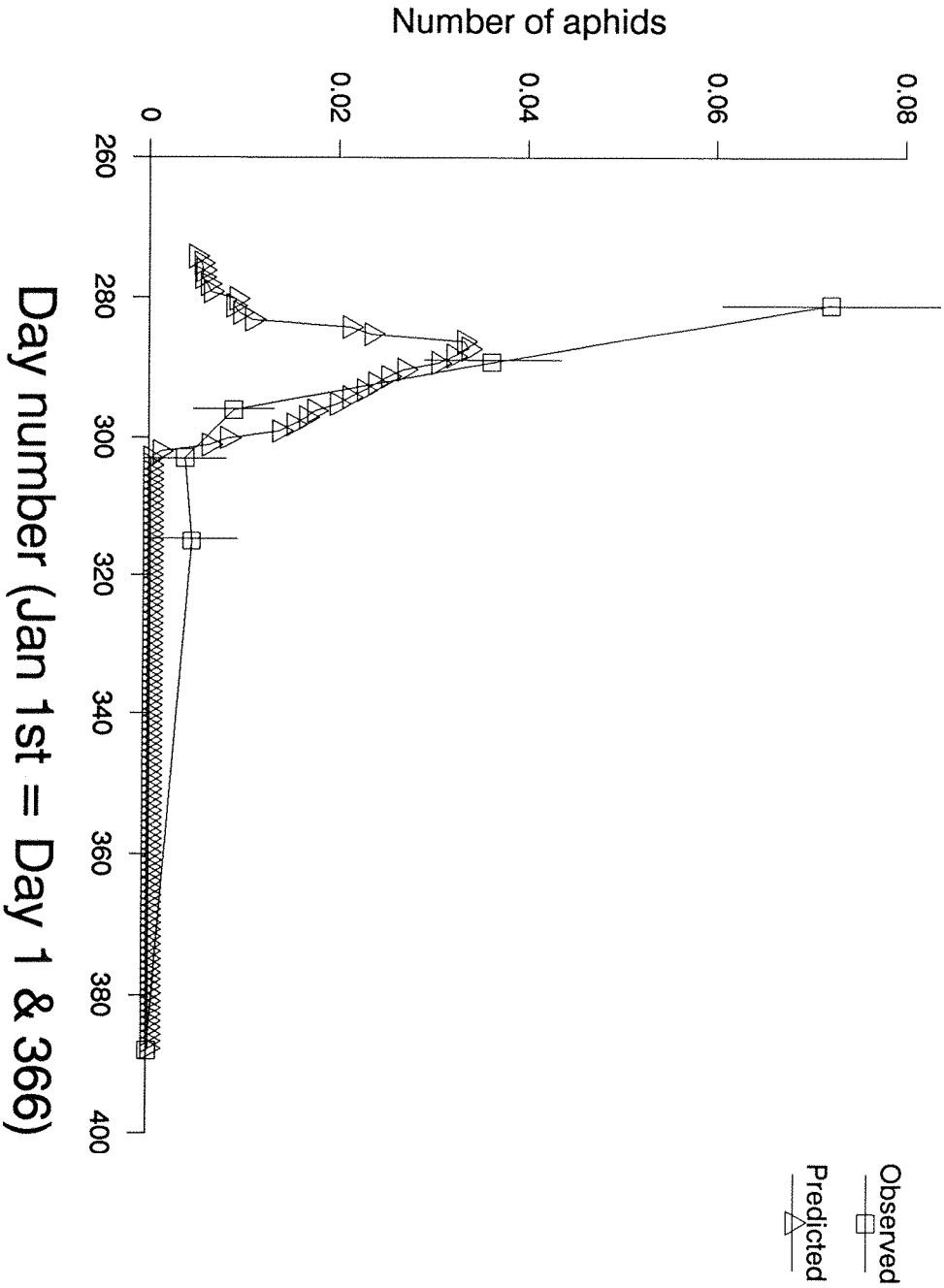


Figure 15. Observed and predicted numbers of apterous adult *R. padi* per plant, 1985/6, sowing date 23 Sept.

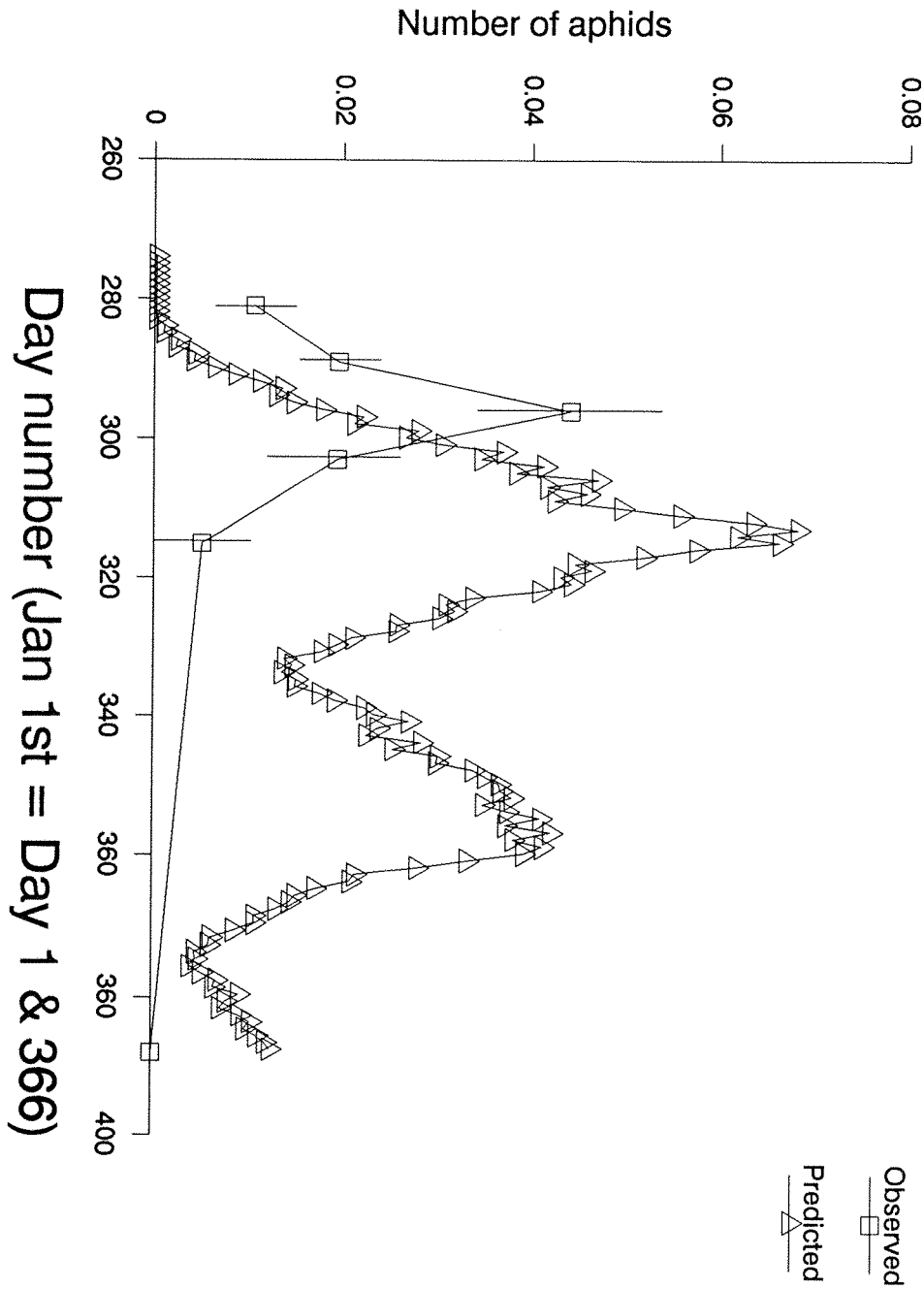


Figure 16. Observed and predicted total numbers of *R. padi* per plant, 1985/6, sowing date 4 Oct.

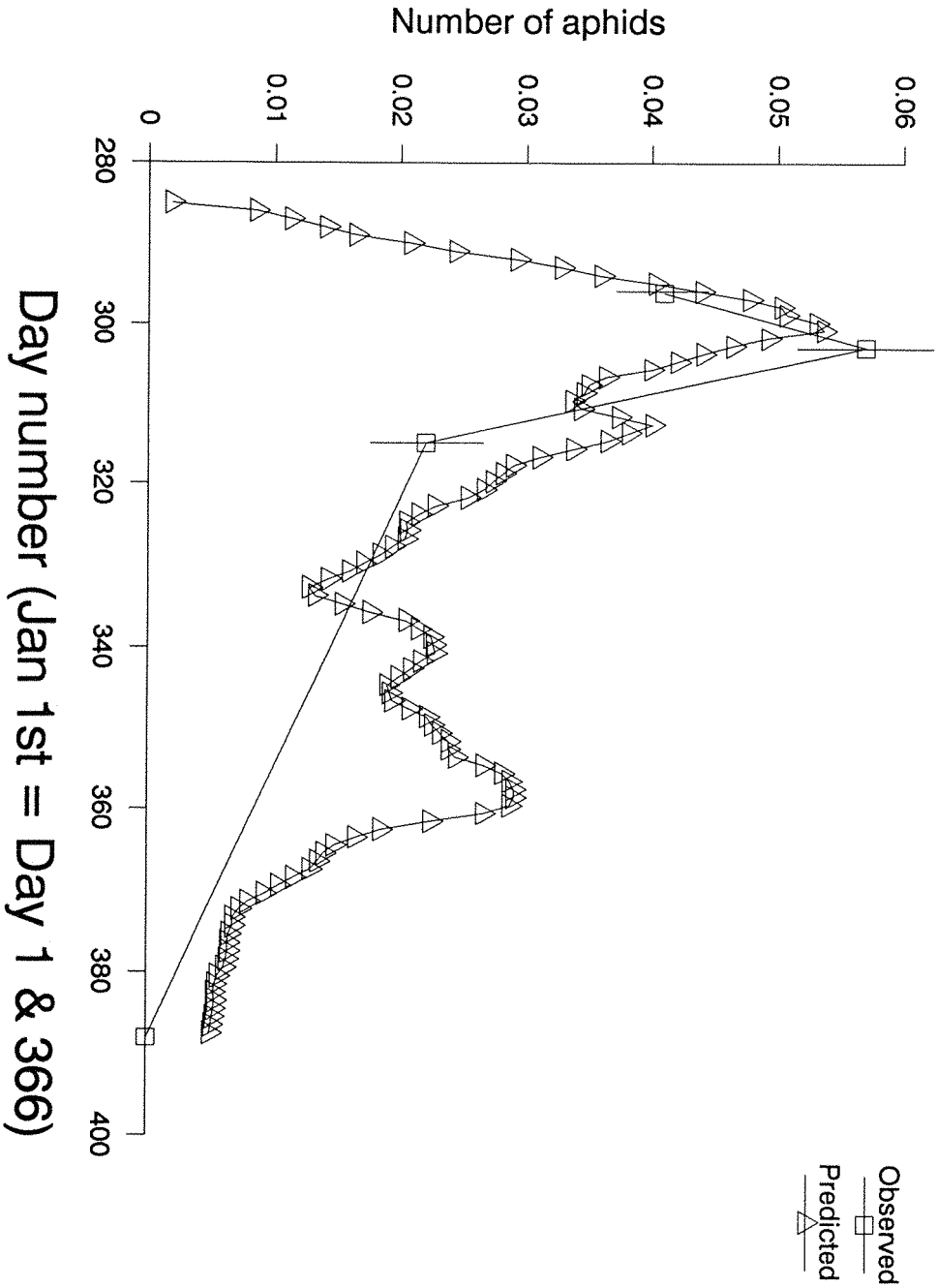


Figure 17. Observed and predicted numbers of alate adult *R. padi* per plant, 1985/6, sowing date 4 Oct.

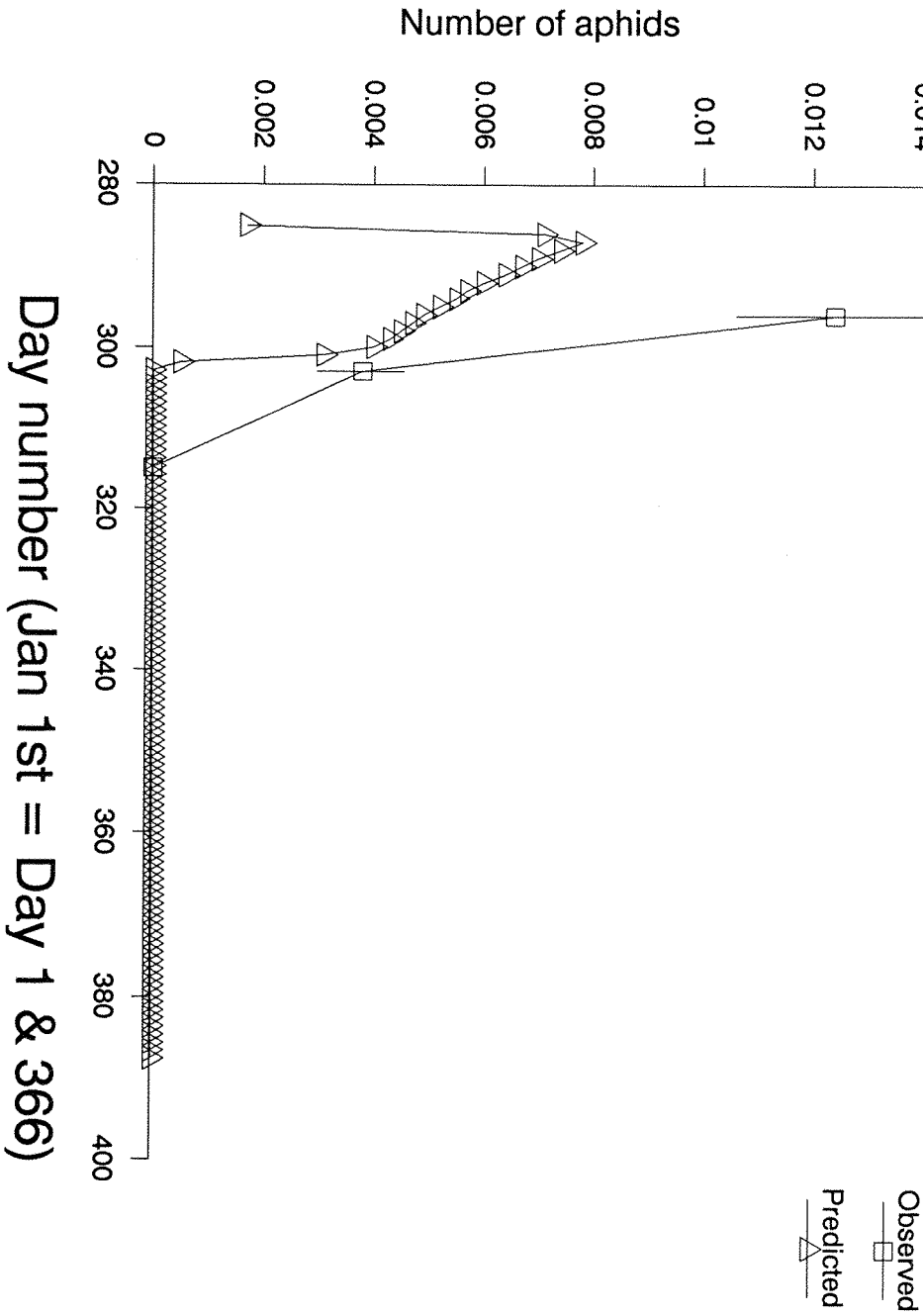


Figure 18. Observed and predicted numbers of apterous adult *R. padi* per plant, 1985/6, sowing date 4 Oct.

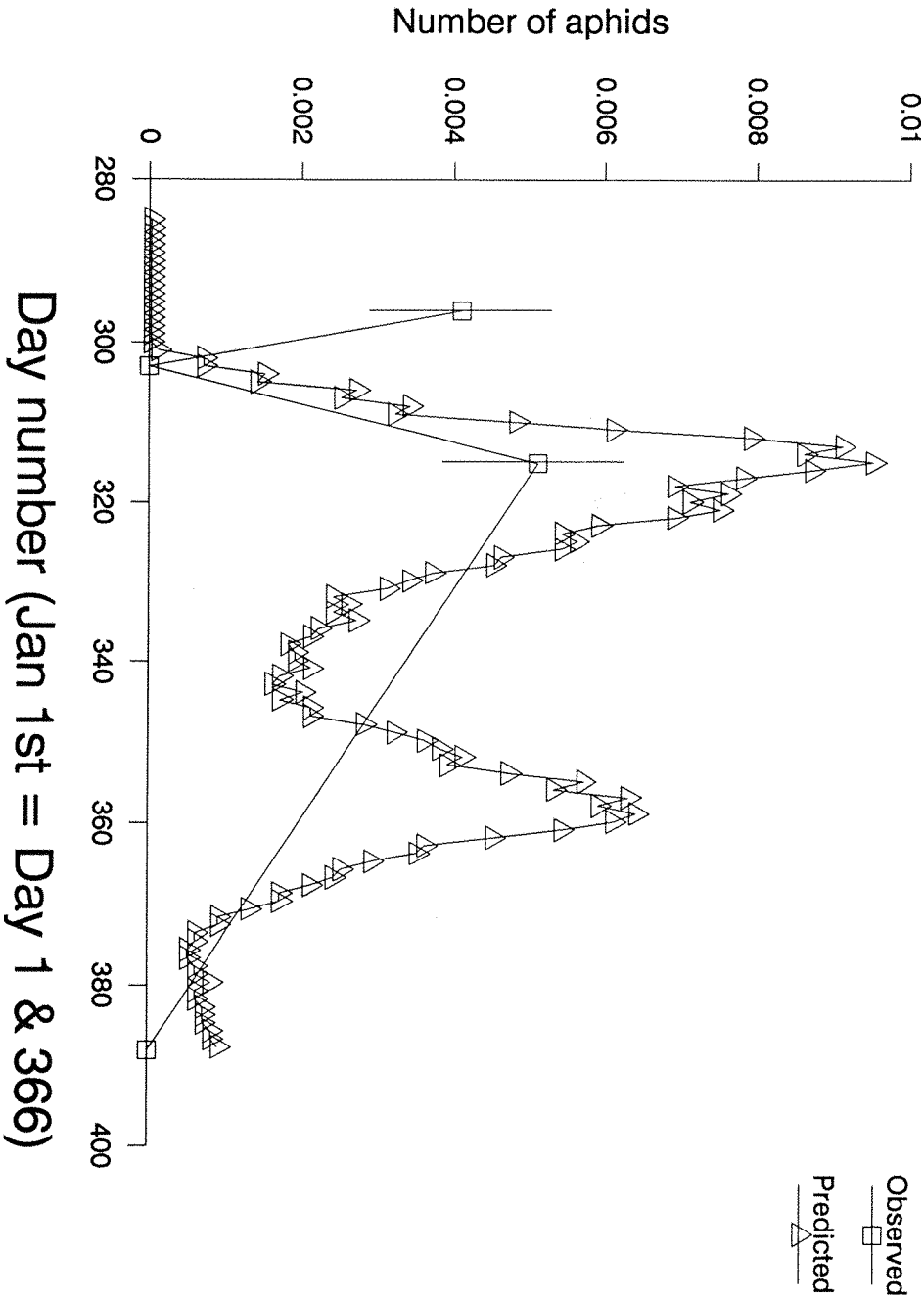
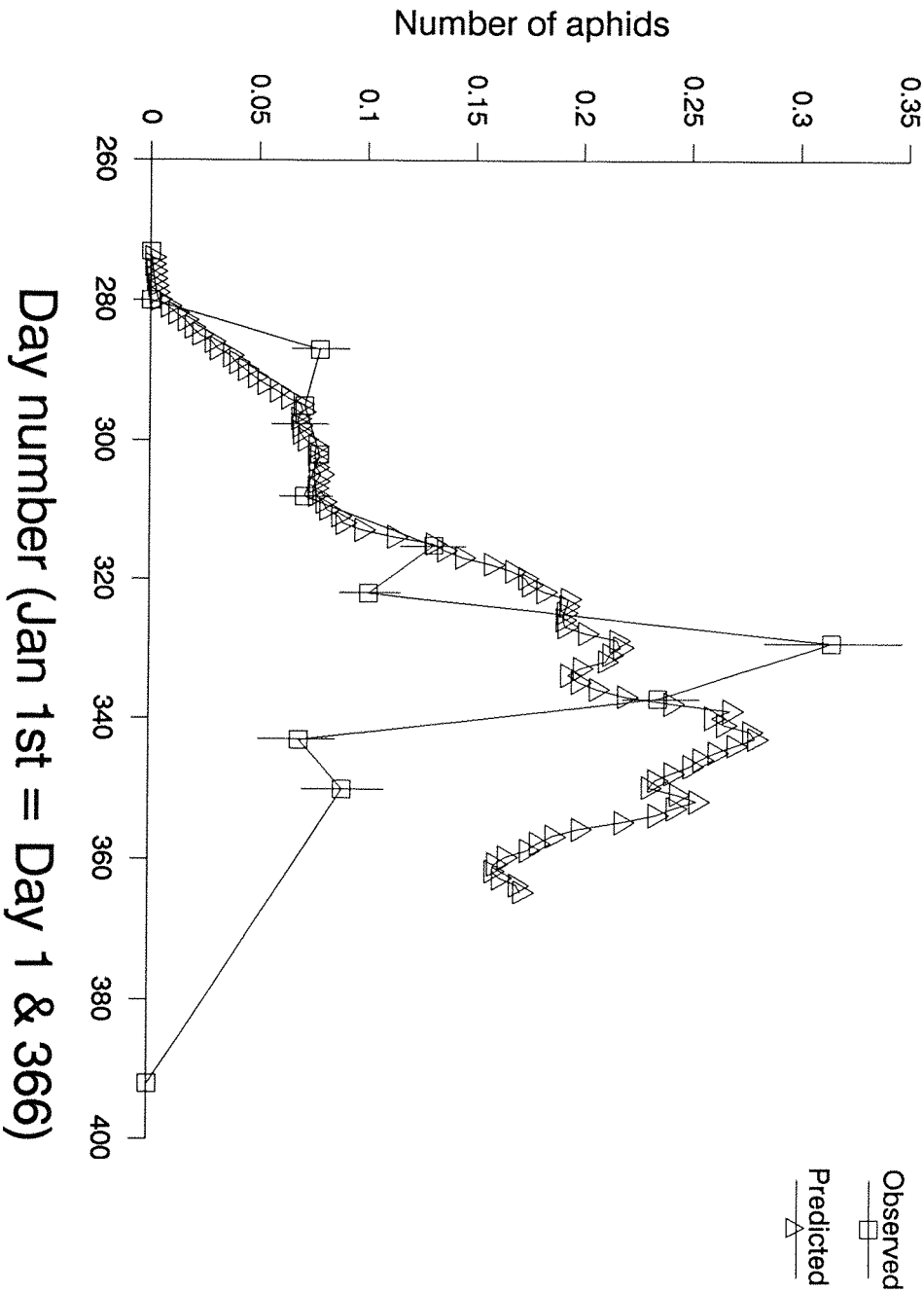


Figure 19. Observed and predicted total numbers of *R. padi* per plant, 1986/7, sowing date 12 Sept.



field was probably the result of very low temperatures, which was not currently in the model. The number of immigrant alates observed on the crop was predicted accurately by the model, although there was a delay of 7 days between predicted and observed peak density (Figure 20). The predicted peak number of apterous adults was similar to that observed but occurred 2 weeks after the peak found in the field (Figure 21). This tends to imply that apterous adults lived for a shorter length of time than allowed for in the model.

6.2.5 1986/7 : Sowing Date 22 Sept.

The model underestimated the early high aphid densities, overestimated the peak density which was 2 weeks too late and did not predict the rapid decline in numbers from the peak density (Figure 22). The failure of the model to predict the early aphid densities was probably the result of underestimating alate numbers, although the shape of the curve was similar to that observed (Figure 23). The overestimation of the peak density was probably the result of overestimating the numbers of apterous adults (Figure 24), implying that adult longevity in the field was less than that allowed for in the model. The failure to predict the rapid decline in aphid numbers was probably because the model did not take into account the effect of low temperatures on aphid survival rates.

6.2.6 1986/7 : Sowing Date 3 Oct.

The predicted population curve did not resemble the observed (Figure 25), although the largest difference between observed and predicted densities was equivalent to about 25 aphids per square metre. The model underestimated the numbers of immigrant alates found on the crop, although the shape of the curves were similar (Figure 26). The peak number of apterous adults were underestimated by the model although the shape of the curves were similar (Figure 27). The underestimation of all of the instars was probably the result of factors, other than low temperatures, increasing the mortality rates of aphids in the field.

6.2.7 1987/8 : Sowing Date 10 Sept.

The model did not predict the relatively high densities found on the first two sample dates nor the relatively low densities found between the fifth and eighth samples (Figure 28). However, it did predict the size and timing of the peak field population accurately and the subsequent decline in numbers. The failure to predict the high densities early in the season was probably the result of underestimating the numbers of immigrant alates (Figure 29). The

Figure 20. Observed and predicted numbers of alate adult *R. padi* per plant, 1986/7, sowing date 12 Sept.

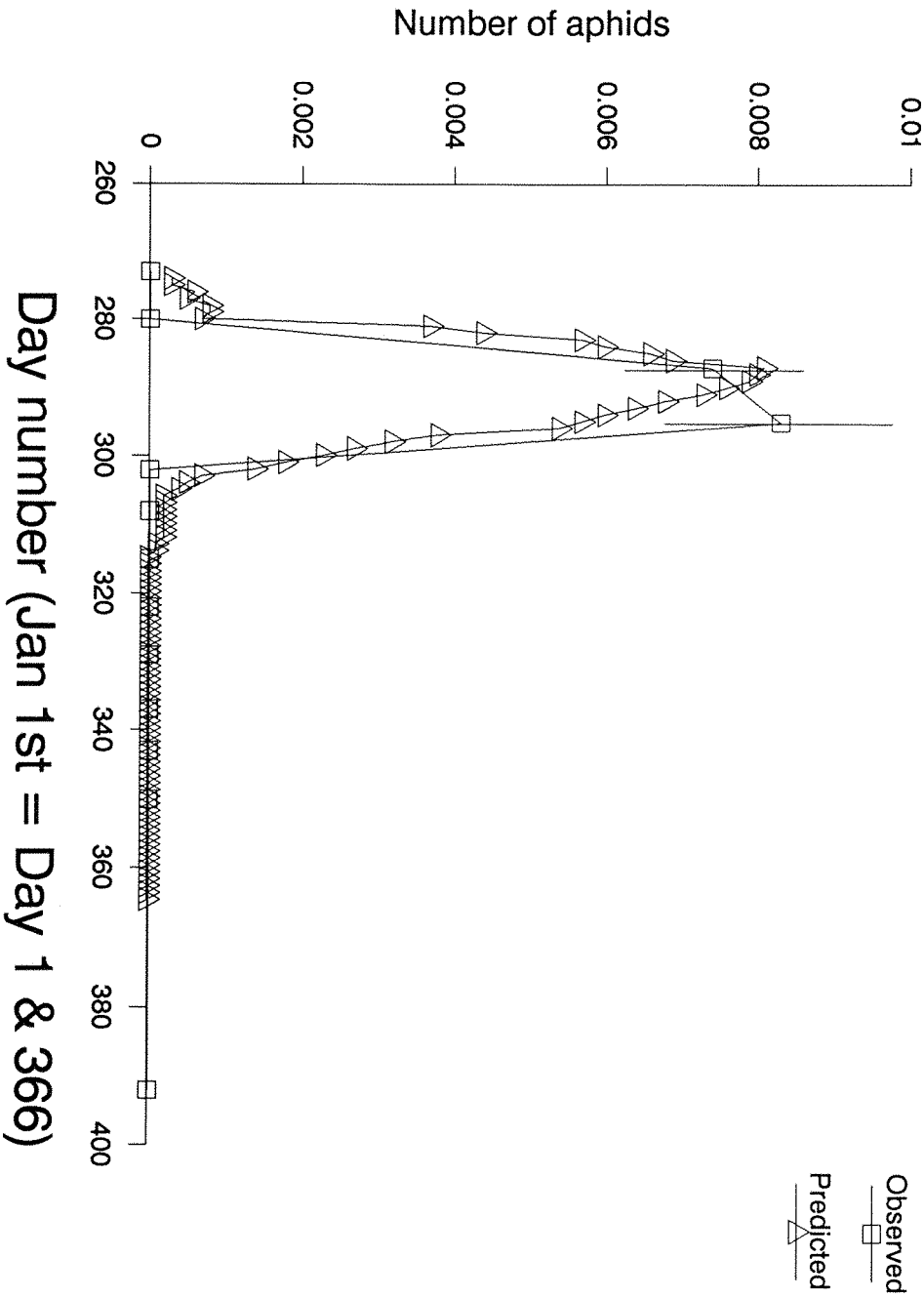


Figure 21. Observed and predicted numbers of apterous adult *R. padi* per plant, 1986/7, sowing date 12 Sept.

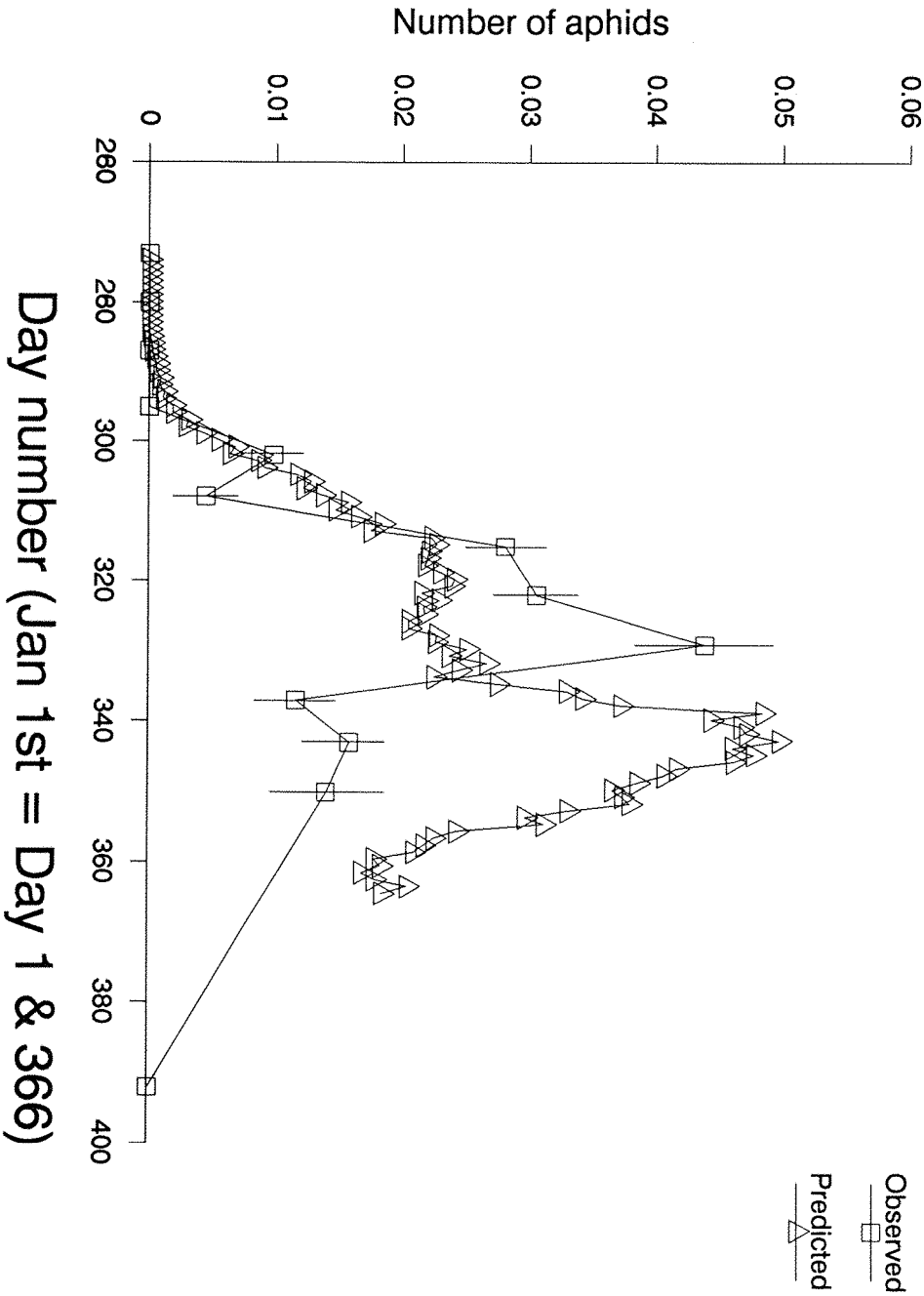


Figure 22. Observed and predicted total numbers of *R. padi* per plant, 1986/7, sowing date 22 Sept.

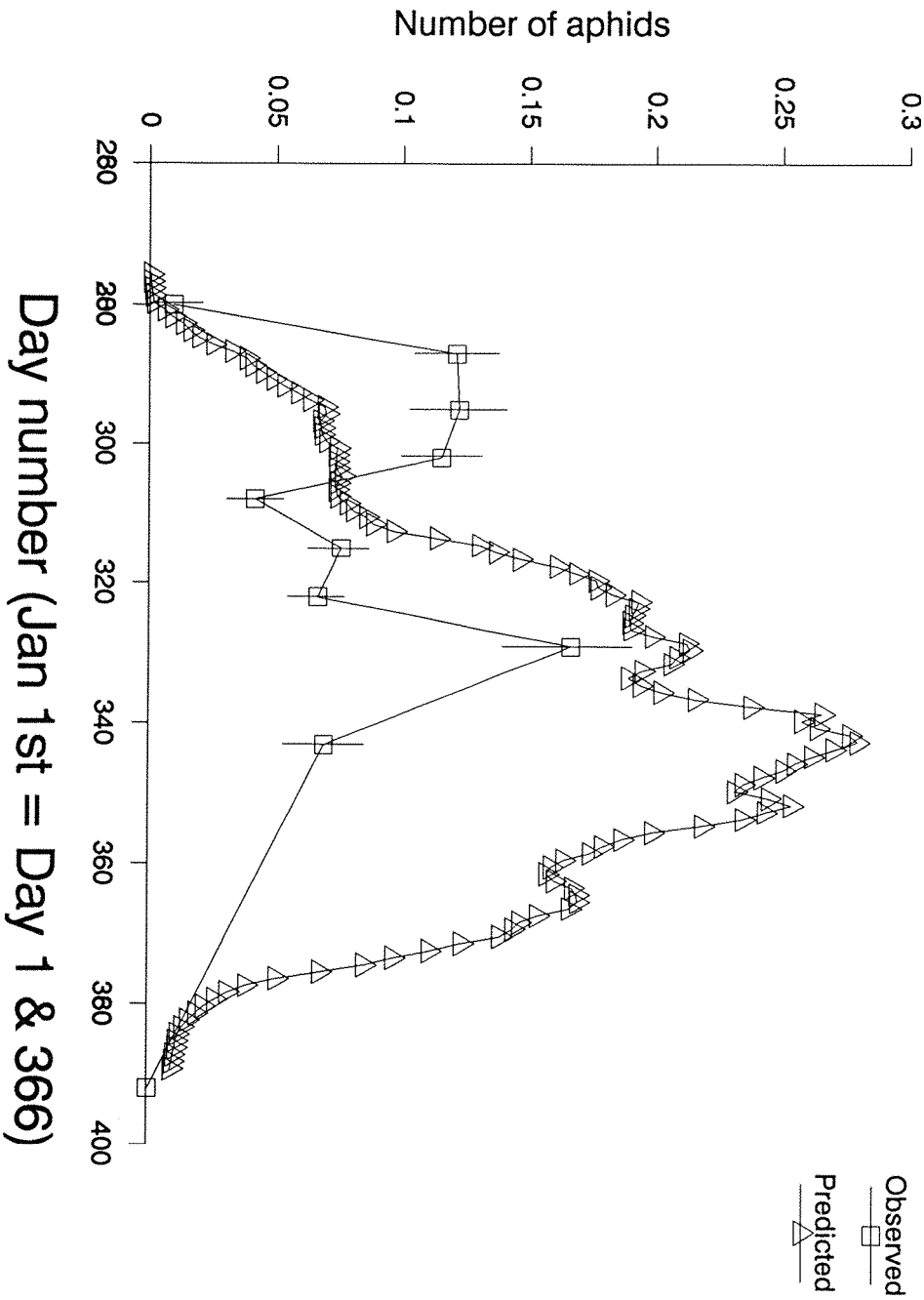


Figure 23. Observed and predicted numbers of alate adult *R. padi* per plant, 1986/7, sowing date 22 Sept.

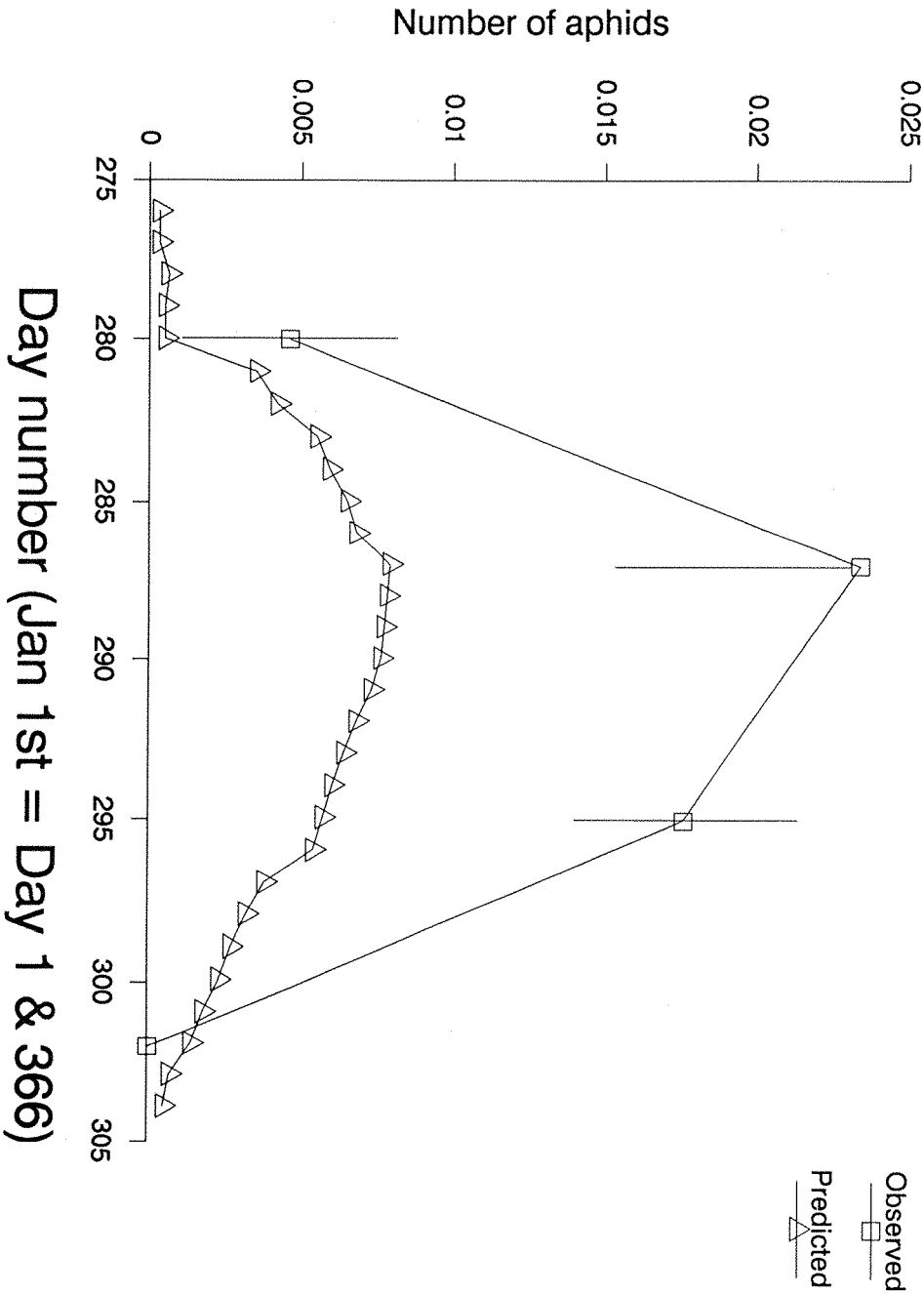


Figure 24. Observed and predicted numbers of apterous adult *R. padi* per plant, 1986/7, sowing date 22 Sept.

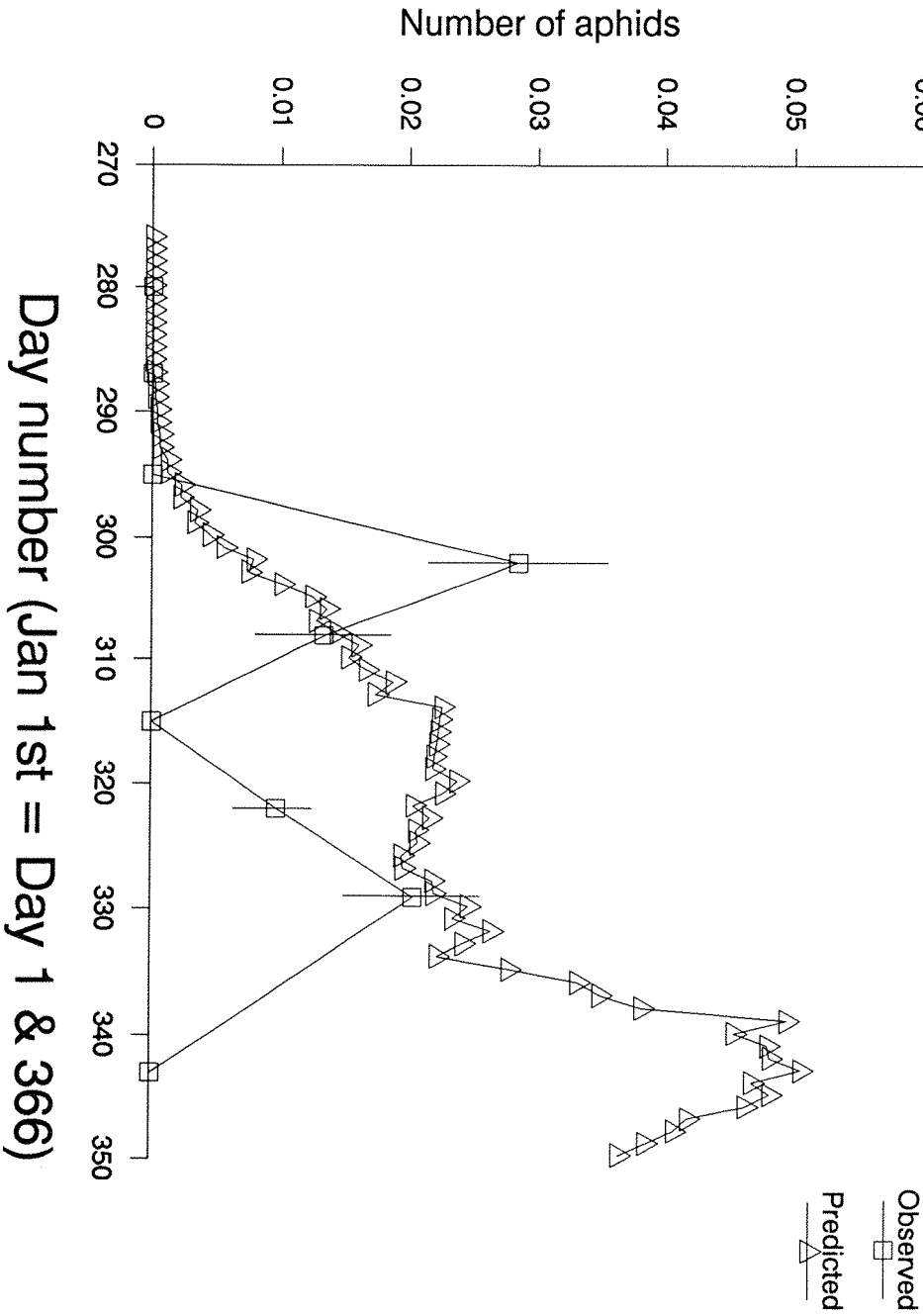


Figure 25. Observed and predicted total numbers of *R. padi* per plant, 1986/7, sowing date 3 Oct.

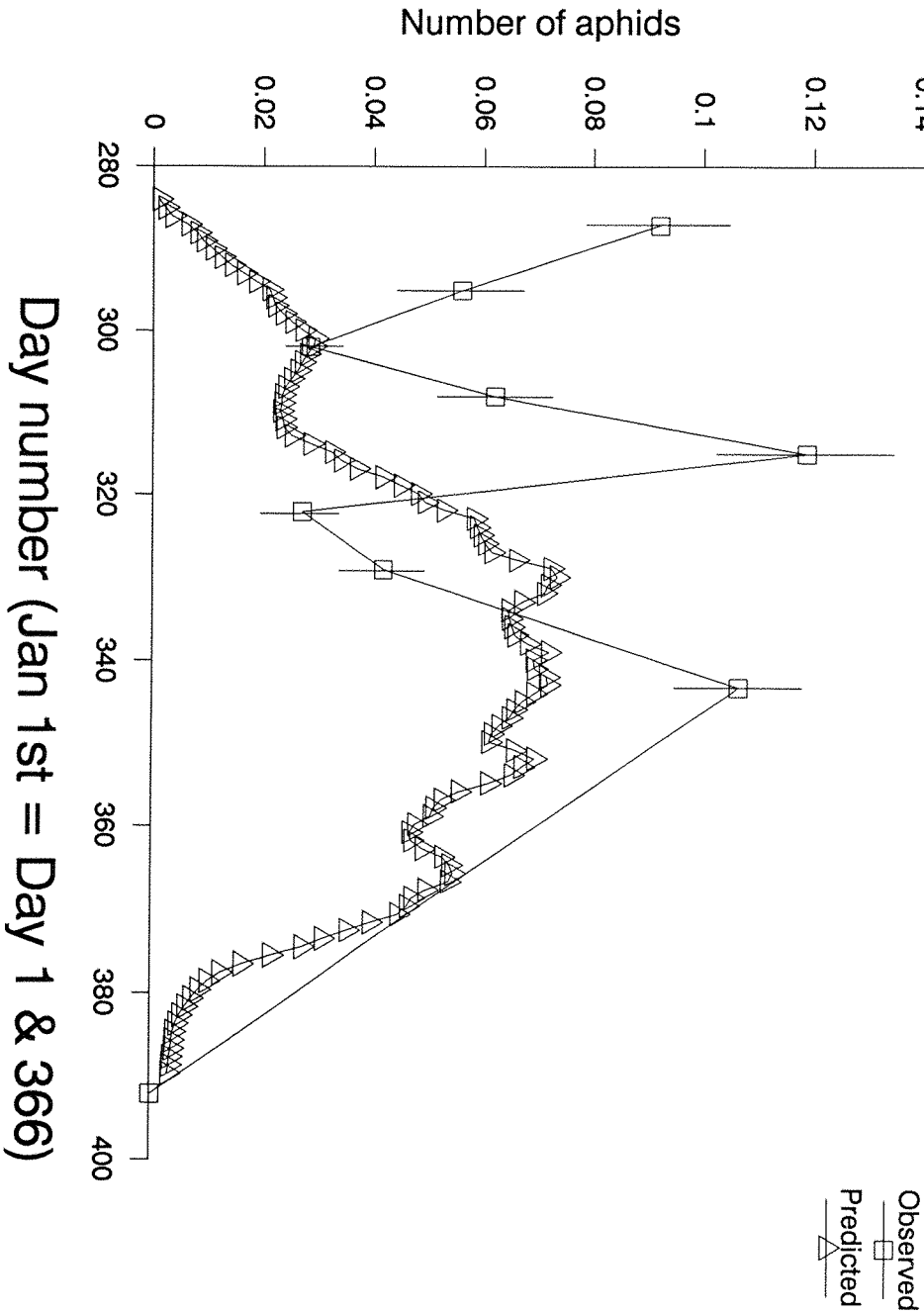


Figure 26. Observed and predicted numbers of alate adult *R. padi* per plant, 1986/7, sowing date 3 Oct.

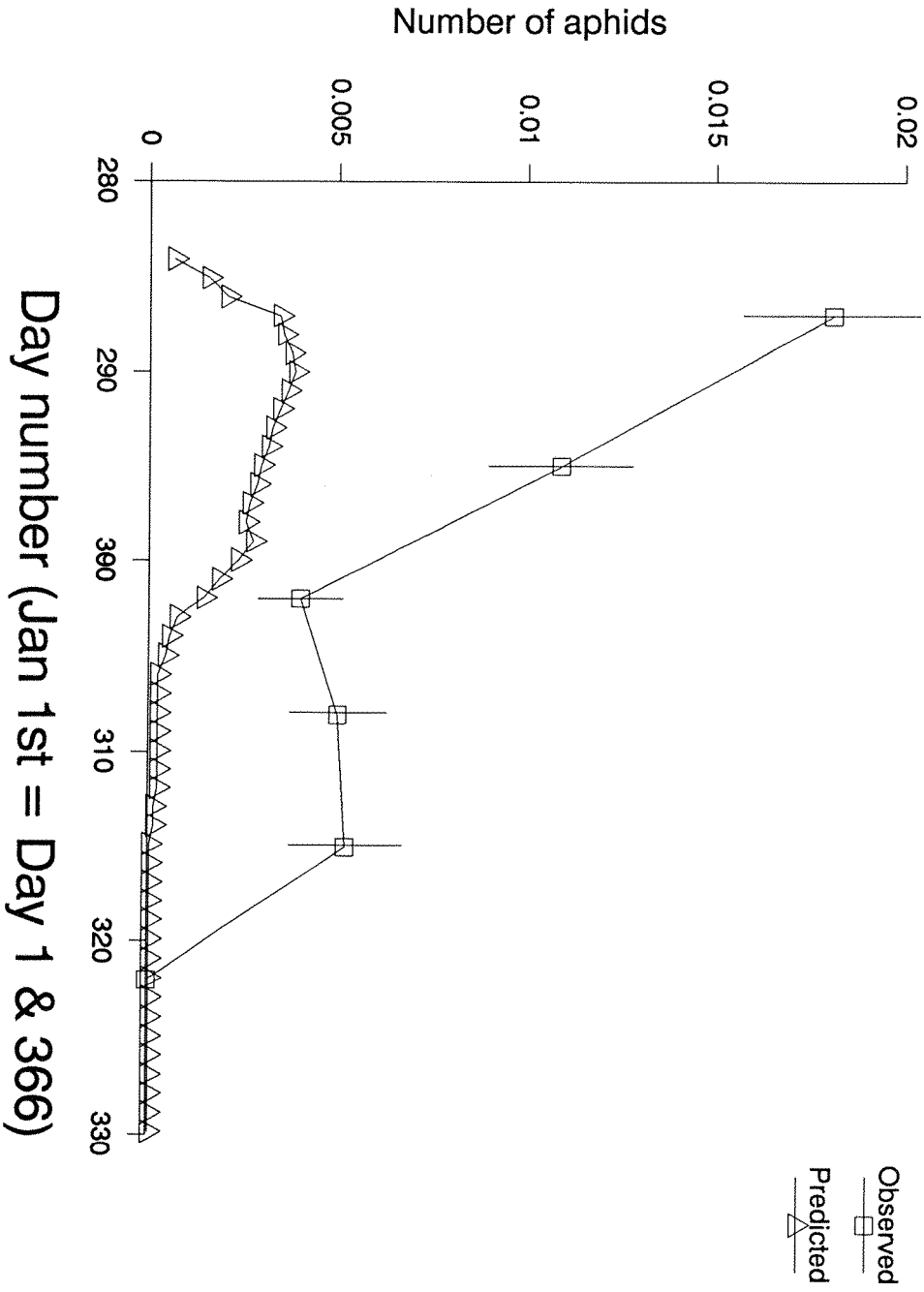


Figure 27. Observed and predicted numbers of apterous adult *R. padi* per plant, 1986/7, sowing date 3 Oct.

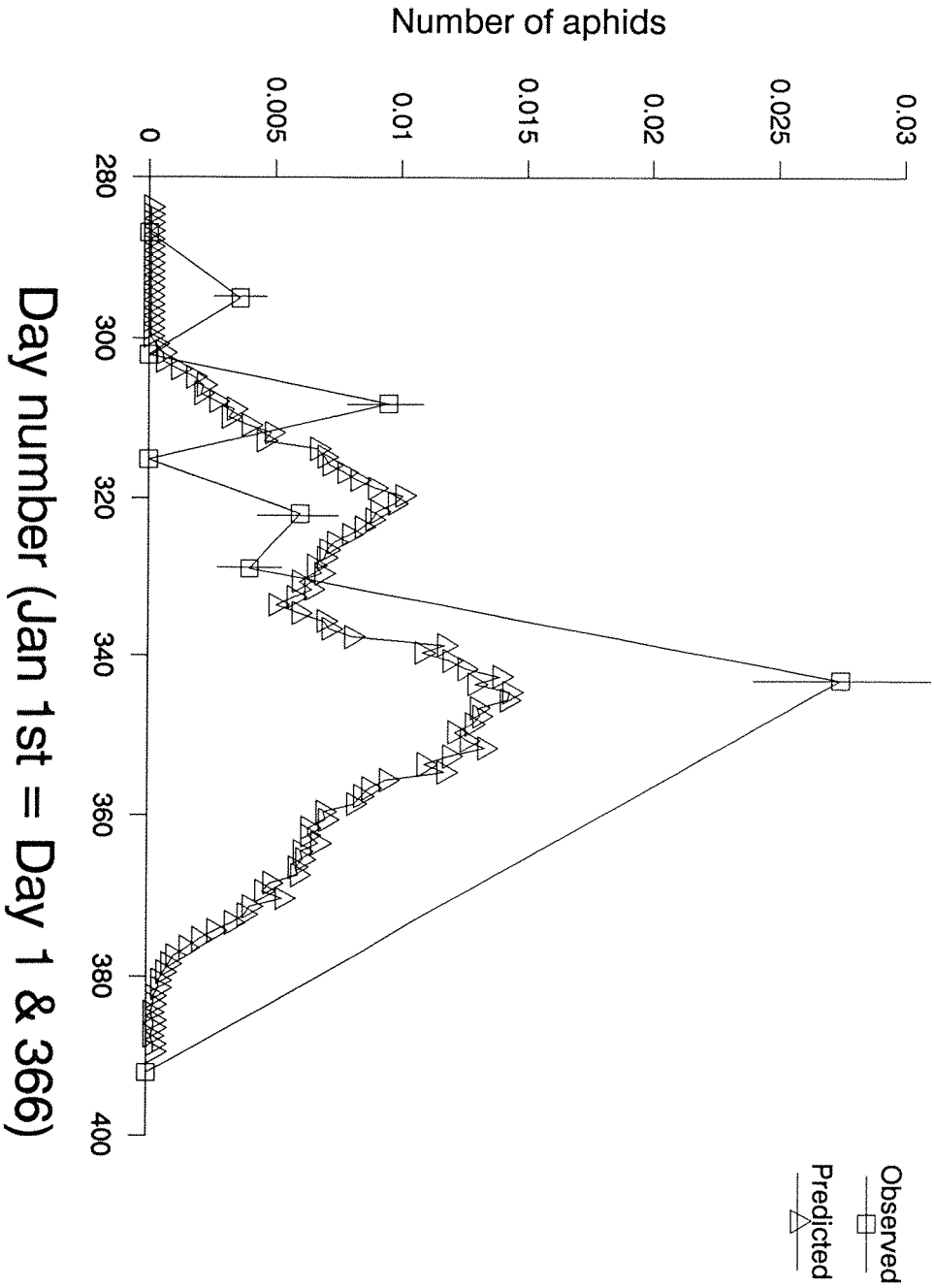


Figure 28. Observed and predicted total numbers of *R. padi* per plant, 1987/8, sowing date 10 Sept.

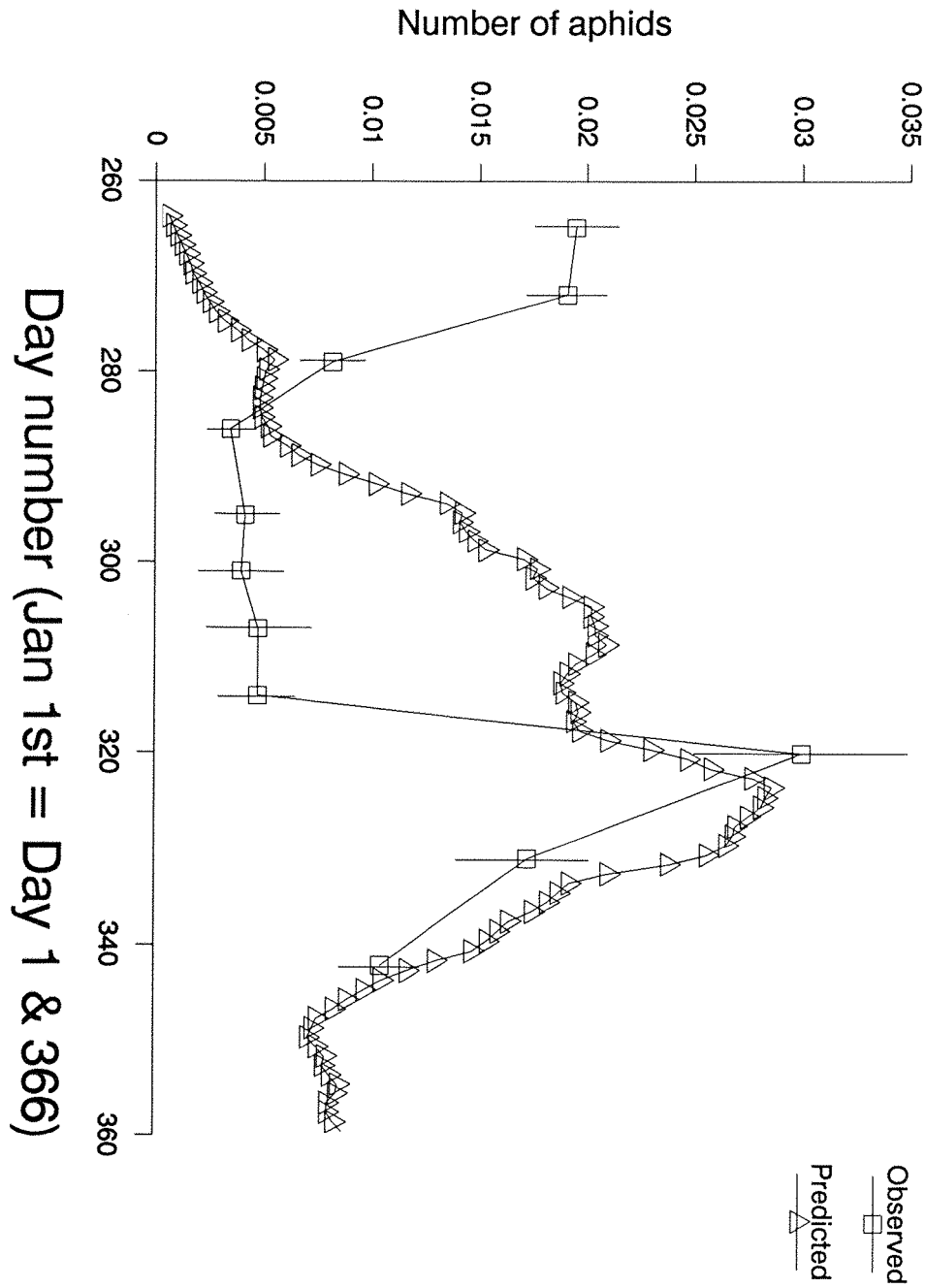
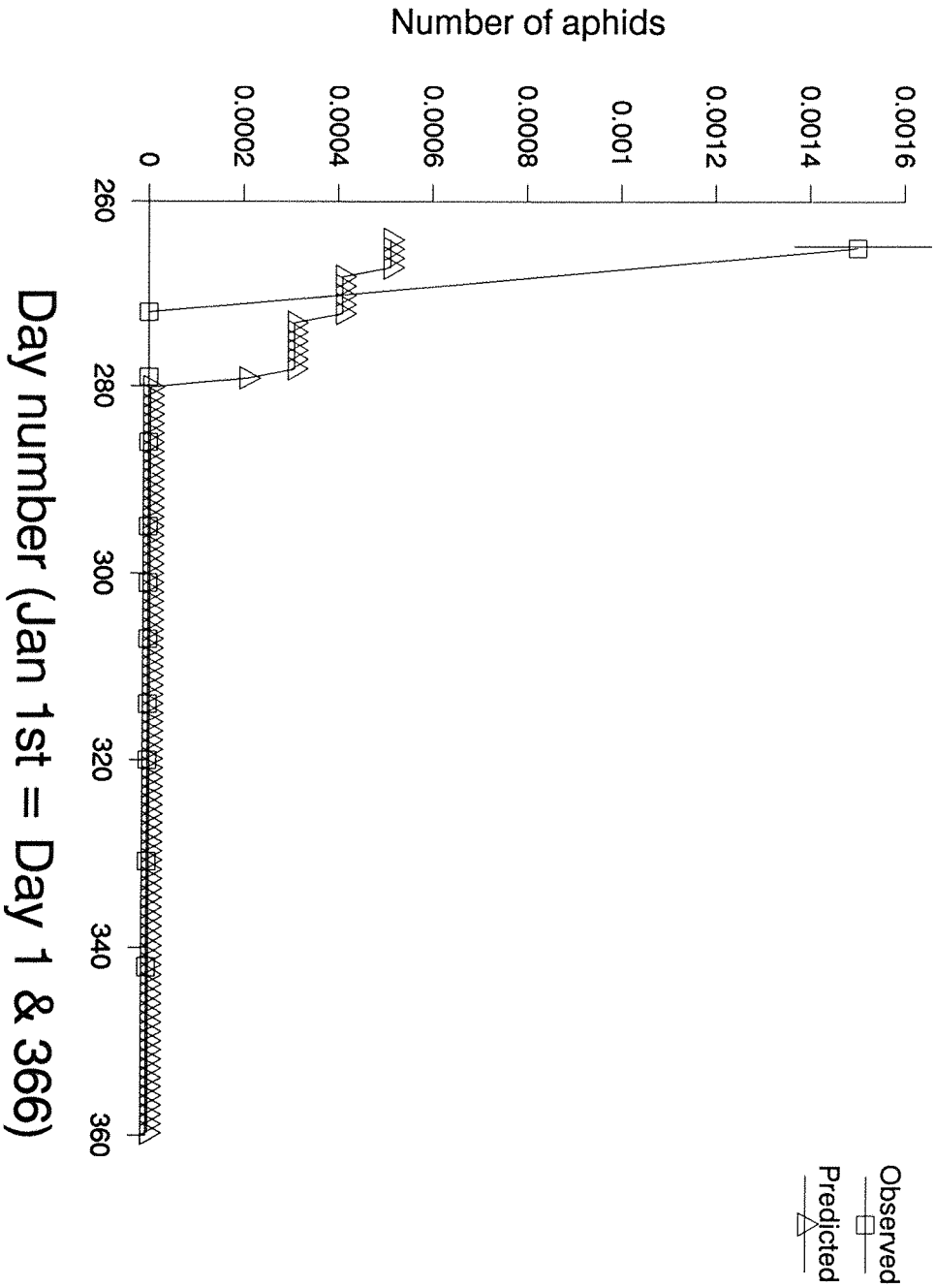


Figure 29. Observed and predicted numbers of alate adult *R. padi* per plant, 1987/8, sowing date 10 Sept.



decline in aphid numbers in mid-season was probably the result of reduced survival rates of aphids in the field, below those used in the model, caused by high rainfall. Over 5cm of rain fell on the 9th October alone. The shape of the predicted curve of the number of apterous adults was similar to that observed with the peak number being slightly overestimated (Figure 30). However, the model again suggested that aphids could overwinter viviparously on the crop although none were found in the field.

6.2.8 1987/8 : Sowing Date 20 Sept.

The predicted population curve was very similar to that observed (Figure 31) even though it predicted the peak number of immigrant alates 15 days before they occurred in the field (Figure 32). The model underestimated the numbers of apterous adults found in the field (Figure 33). However, because of the relatively low densities of aphids found on the crop the difference between the predicted and observed peak densities of apterous adults was equivalent to only 3 aphids per square metre.

6.3 Discussion

The model predicted the population dynamics of *R. padi* with variable degree of accuracy on five different crop sowing dates in three different years, representing a wide range of aphid densities. Some processes of the model require further attention. The need to determine the longevity of instars under natural conditions became apparent from comparwasons of the predicted and observed densities of apterous adults. Results from the model implied that the longevity of apterous adults was less in the field than those used in the model, while the longevity of alates might be longer. The importance of adult longevity and the accuracy to which it should be known will be determined in Chapter 8. However, Carter (1978) has shown that doubling the duration of adult age-classes of *S. avenae* increases peak density by about 30% but that the timing of the peak was not altered.

The model has also highlighted the need for more information on the effects of biotic and abiotic factors on the survival of *R. padi*. Very little was known about the influence of natural enemies on the population dynamics of *R. padi* in the autumn and summer, with the exception of the work carried out by Sopp & Chiverton (1987). Natural enemies were rare in 1986 and 1987, but they were evident, from field observations, in 1985. The influence they have has not been considered in the model because of the lack of suitable data

Figure 30. Observed and predicted numbers of apterous adult *R. padi* per plant, 1987/8, sowing date 10 Sept.

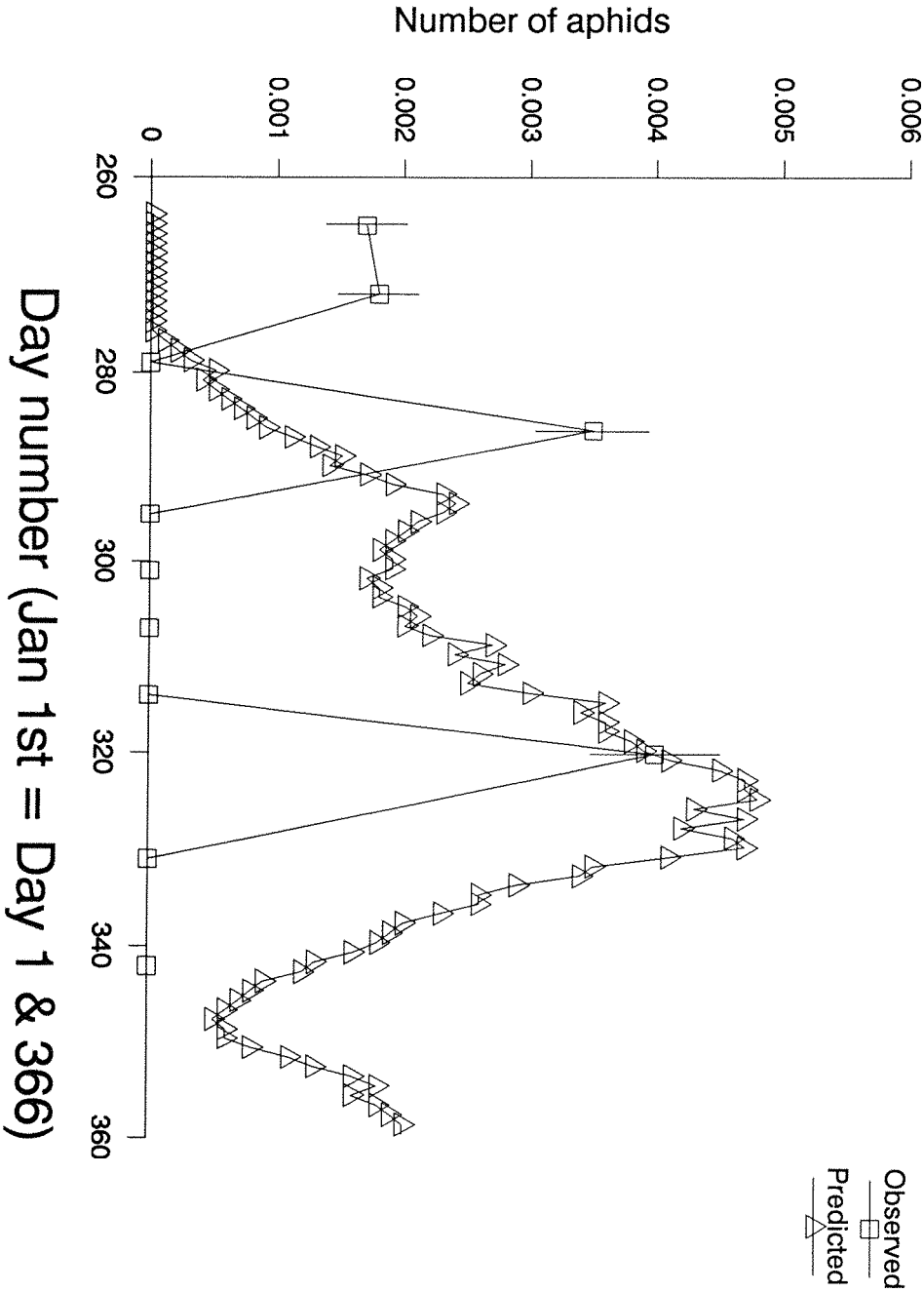


Figure 31. Observed and predicted total numbers of *R. padi* per plant, 1987/8, sowing date 20 Sept.

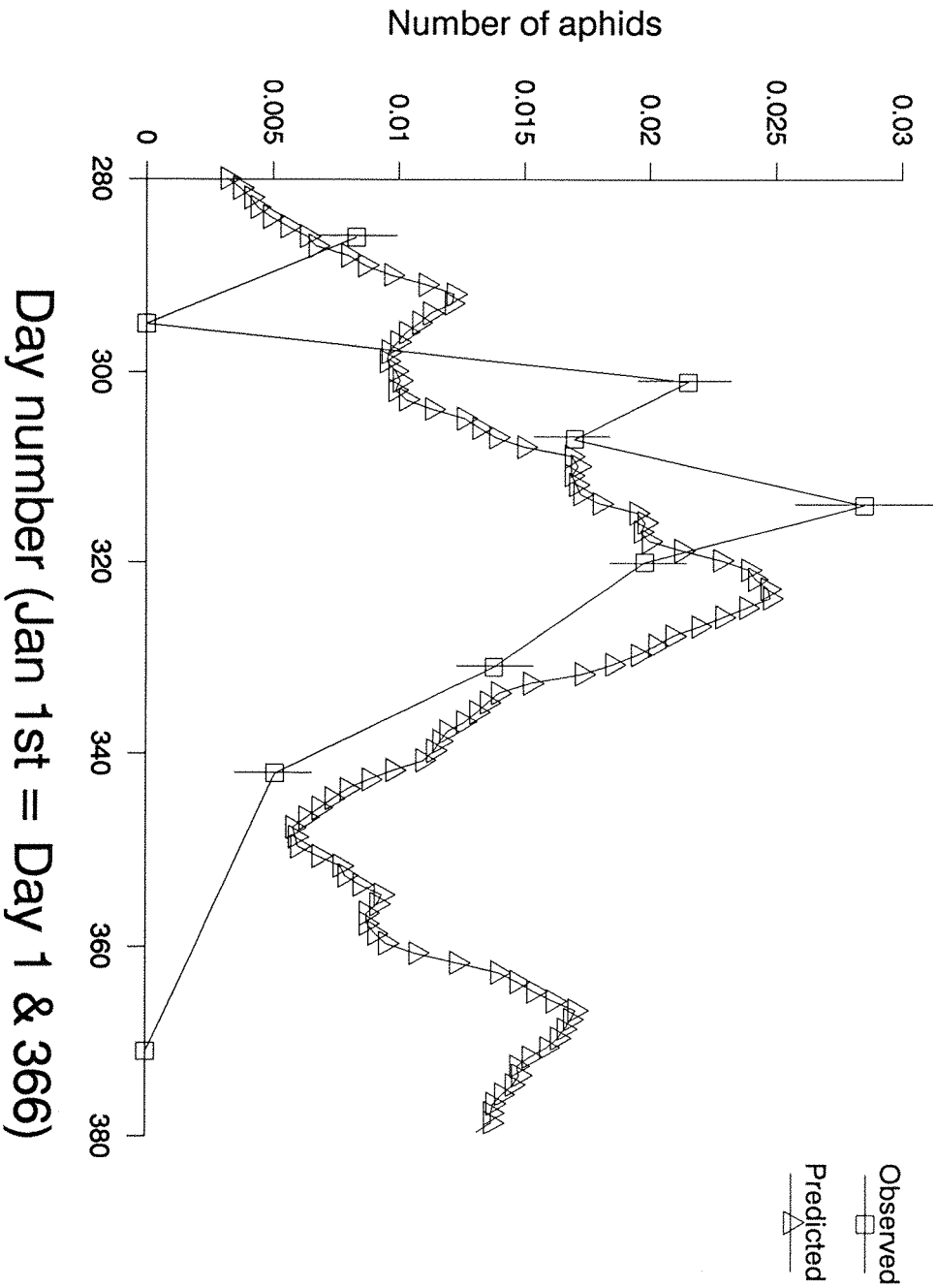


Figure 32. Observed and predicted numbers of alate adult *R. padi* per plant, 1987/8, sowing date 20 Sept.

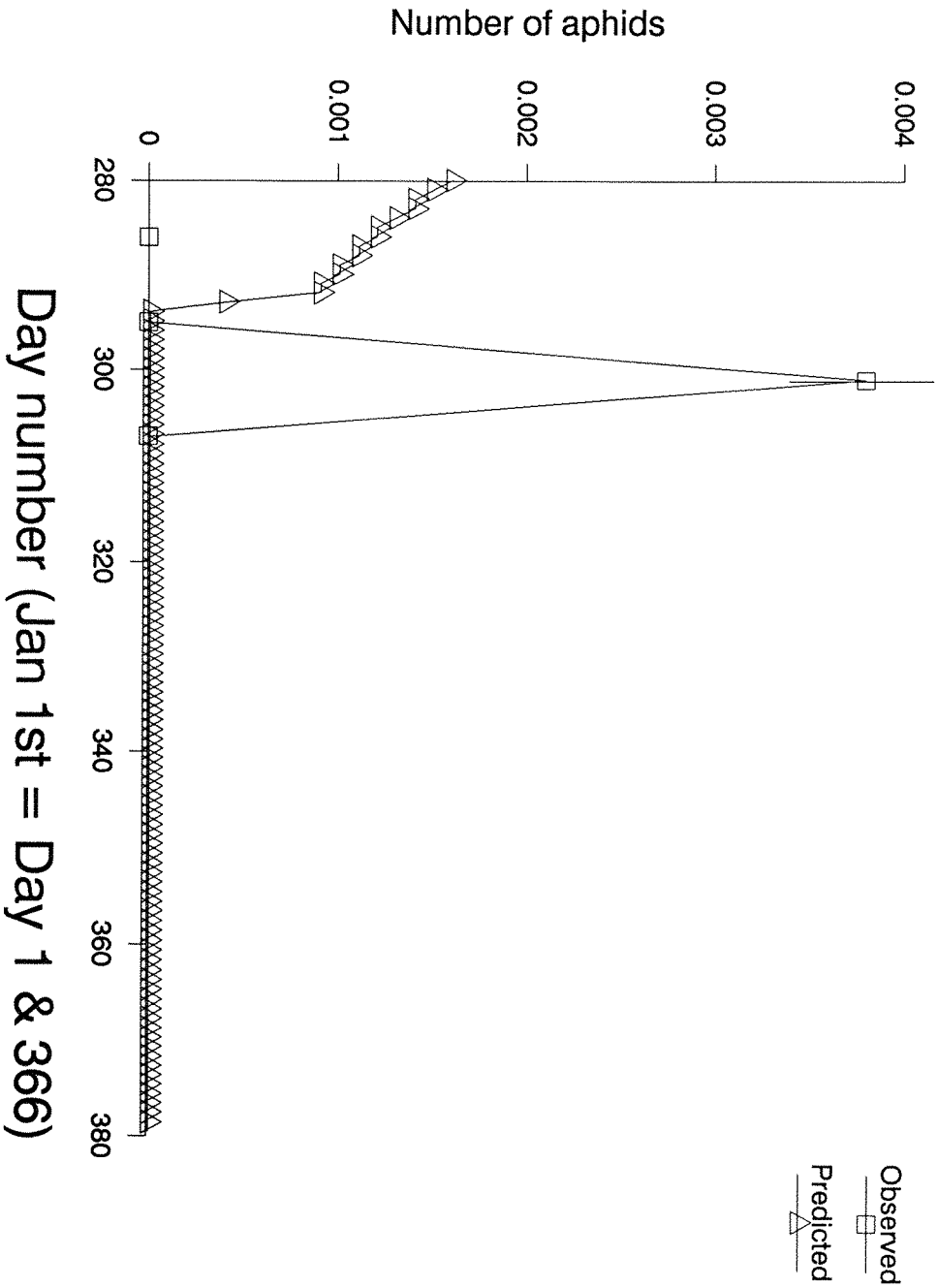
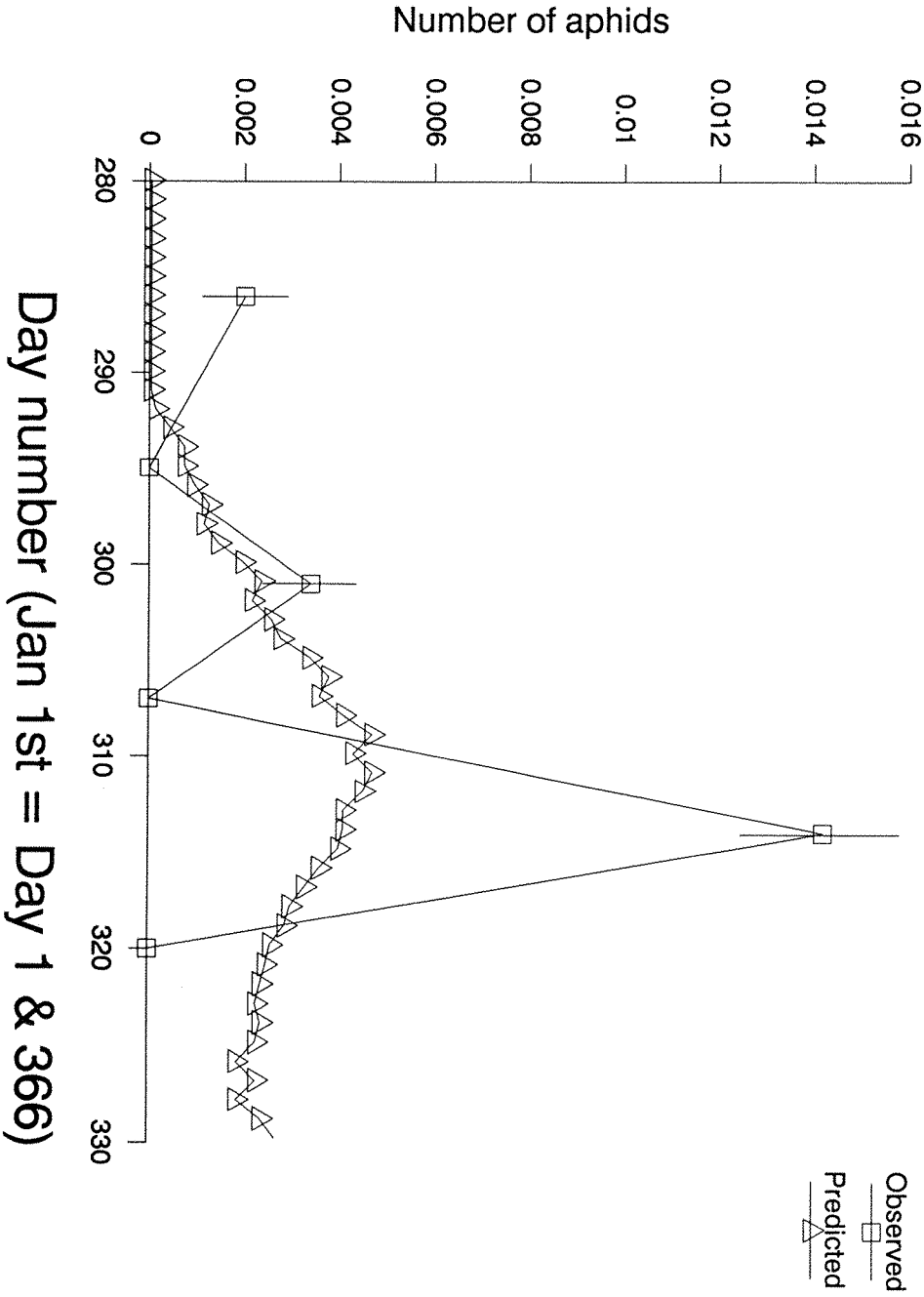


Figure 33. Observed and predicted numbers of apterous adult *R. padi* per plant, 1987/8, sowing date 20 Sept.



available on the underlying biology of predation, parasitwasm and fungal infection.

Even less was known on the effects of rain and wind on aphid survival in the autumn and winter. Comparisons of changes in aphid numbers at Rothamsted with weather recordings imply that heavy rainfall might reduce the survival of the aphids but definite relationships are not apparent. Possibly a combination of laboratory experiments, using simulated rain, and a more intensive field sampling programme, recording aphid population dynamics before and after rain showers and relating any differences to the conditions, might provide the information.

Another abiotic factor on which further research is required is low temperature. Although some work has been carried out (Williams, 1980) the data are not in a suitable format to be used in the model. For example, Williams (1980) related the mortality of aphids in a 7-day period to the minimum temperature during that period. Short term studies with more intense sampling programmes would be more useful.

Although some processes in the sub-model require further research it will be used to investigate the spread of BYDV.

7. Sensitivity Analysis of Aphid Sub-System

7.1 Introduction

A sensitivity analysis was carried out to determine the importance of processes in the model and the accuracy to which the parameters need to be known.

Small changes to temperature (±1°C to the daily maximum and minimum temperatures), adult longevity (±20%), instar duration (±20%), survival rate (±5%, the standard rate was 95% so that any increase greater than 5% would have resulted in a survival rate of greater than 100%), reproductive rate (±20%), immigration (±20%) and morph determination (±20%) were made to assess their effects on the system.

7.2 Results

7.2.1 Temperature

An increase of temperature by 1°C led to a super-proportional (Carter et al., 1982) increase in the size of the peak population density, from 1.54 to 2.92 aphids per plant in 1985/6 (Figure 34), from 0.28 to 0.82 in 1986/7 (Figure 35) and from 0.029 to 0.074 in 1987/8 (Figure 36), with an asymmetrical appearance. However, a reduction of 1°C reduced the size of the peak density and also brought it forward by between 11 to 19 days.

As an increase of 1°C resulted in an increase in peak density of between 90 and 190%, indicating that temperature is an important factor and, therefore, should be measured as accurately as possible.

7.2.2 Adult longevity

Modifying the length of apterous and alate adult instar longevity by 20% had no effect on the timing of peak aphid density but it did alter its size (Figures 37,38,39). The response is symmetrical, an increase in the longevity resulted in an increase in peak density of between 28 to 41%, while a reduction of 20% resulted in a reduction in the peaks by between 32 to 41%. The reason for this may have been that increasing the longevity of adult instars increases the period for which they are reproductively active and hence more nymphs are laid increasing population numbers.

Figure 34. The effect of changes in temperature on density of *R. padi*, 1985/6.

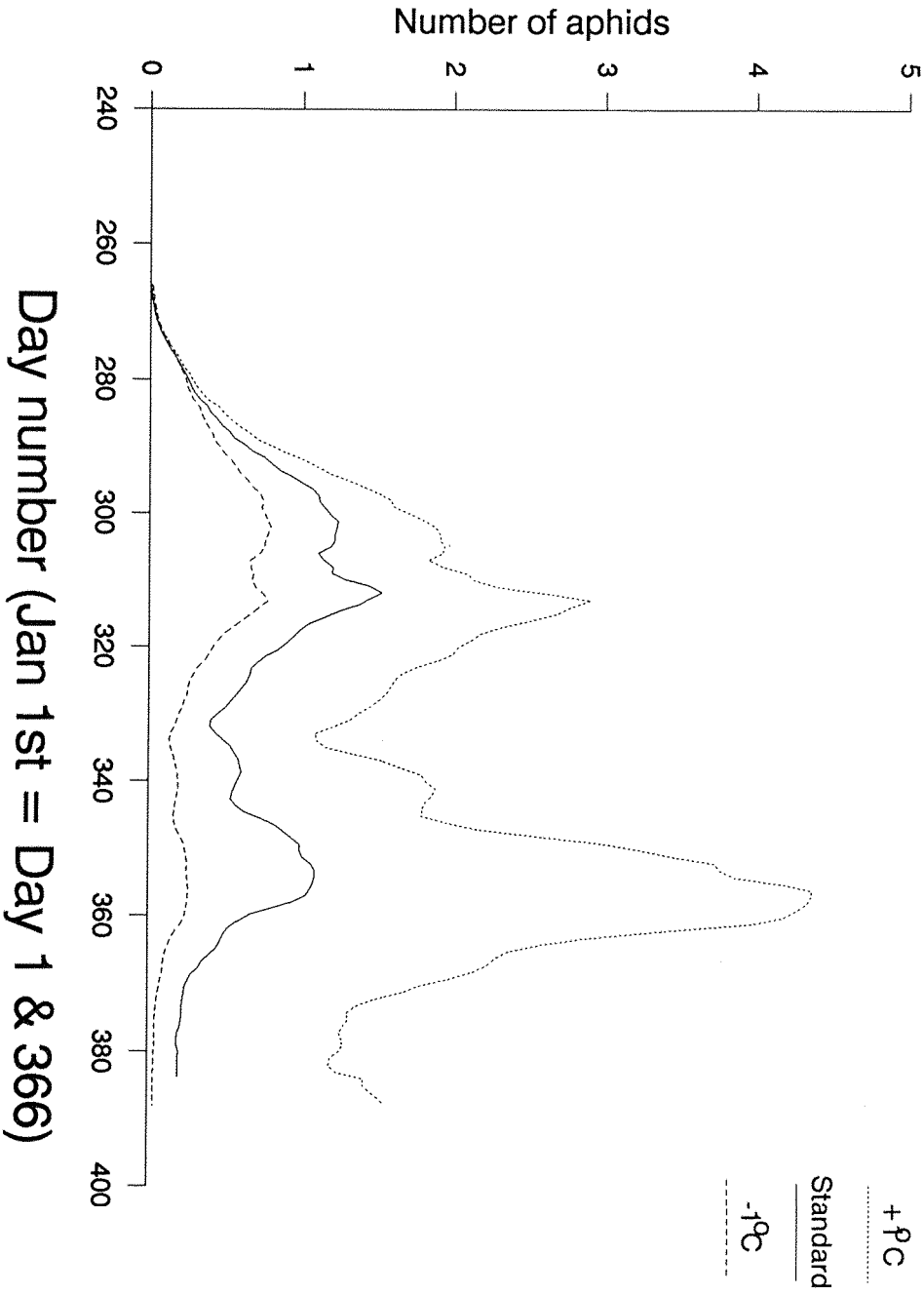


Figure 35. The effect of changes in temperature on density of *R. padi*, 1986/7.

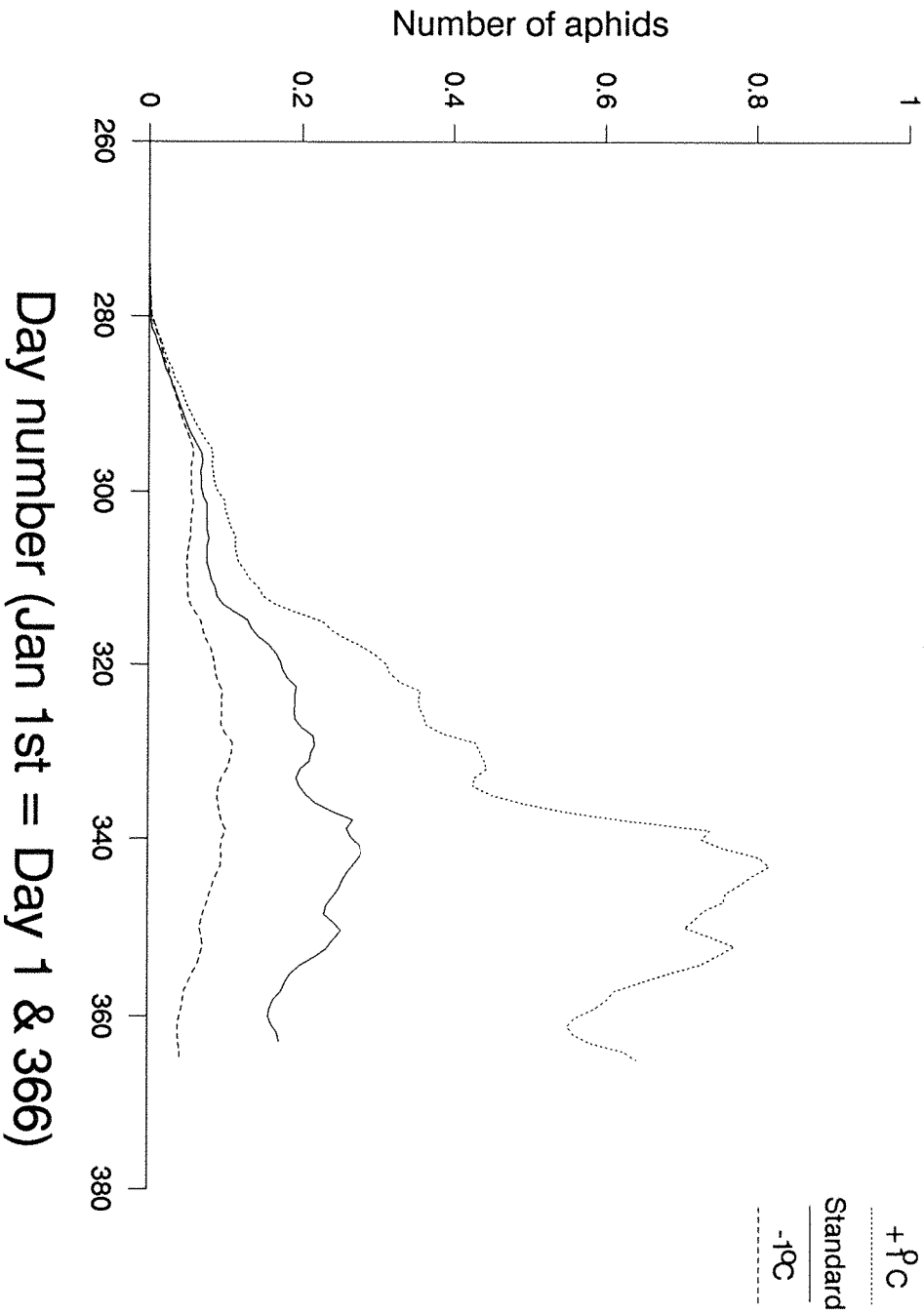


Figure 36. The effect of changes in temperature on density of *R. padi*, 1987/8.

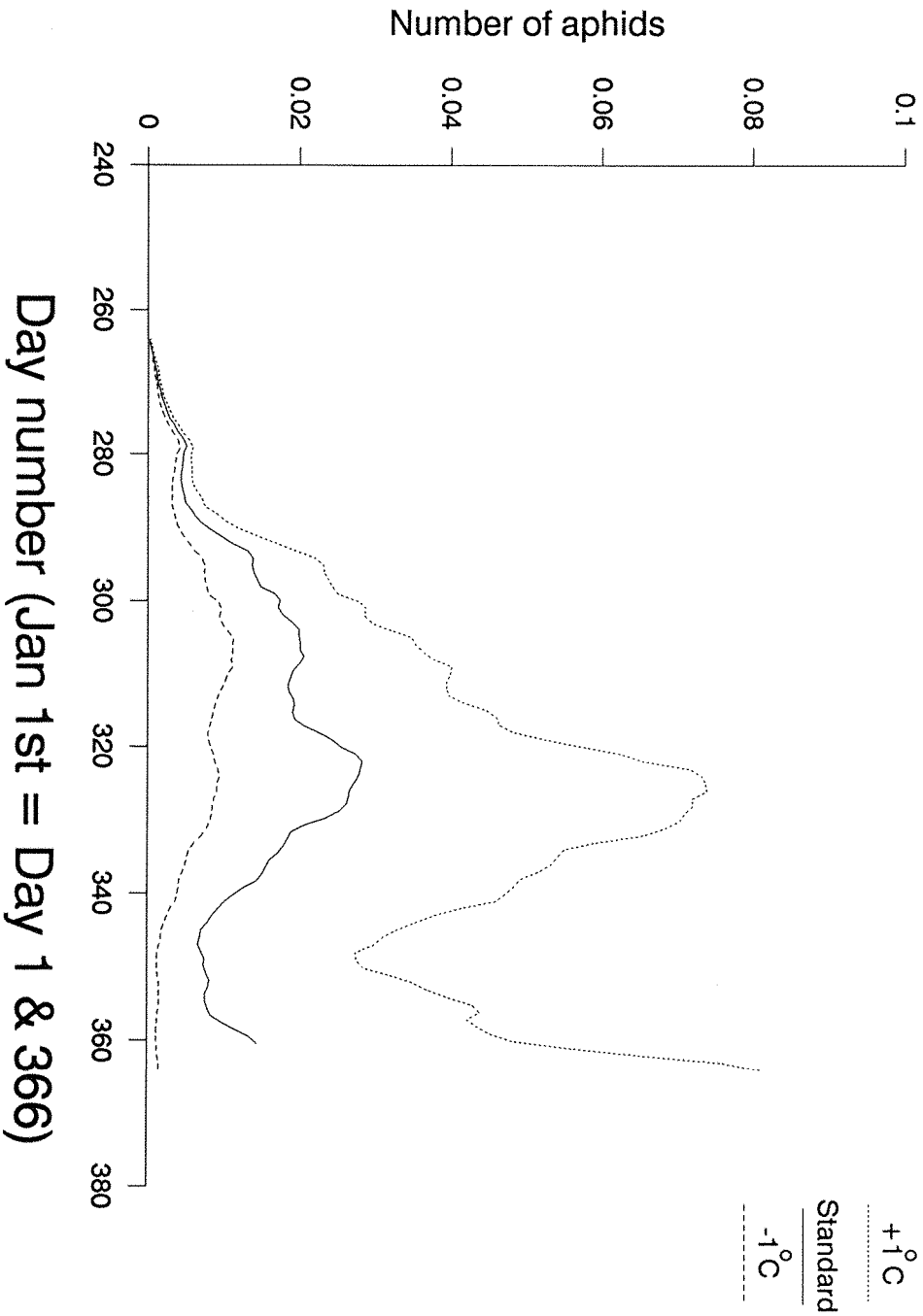


Figure 37. The effect of changes in adult longevity on density of *R. padi*, 1985/6.

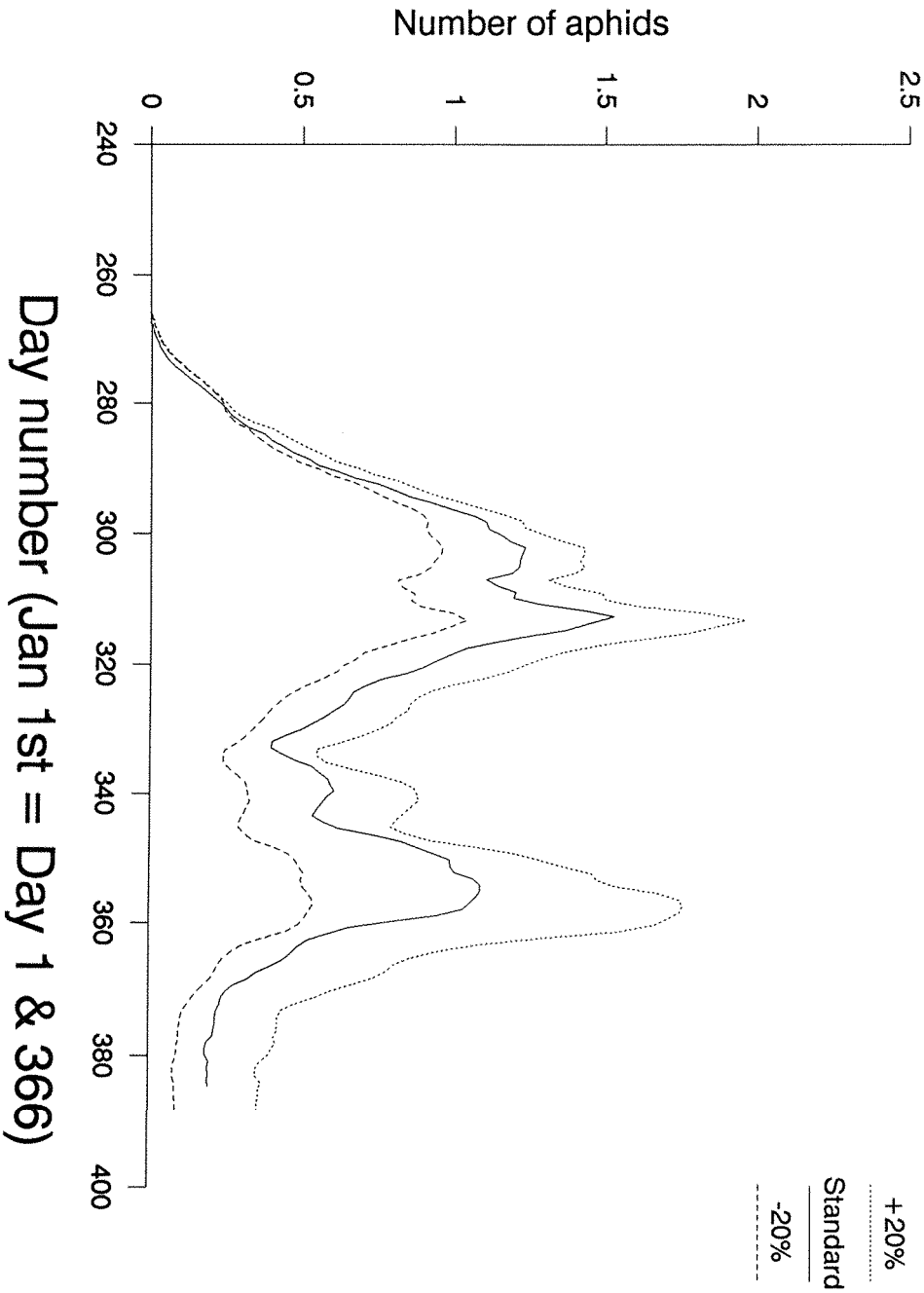


Figure 38. The effect of changes in adut longevity on density of *R. padi*, 1986/7.

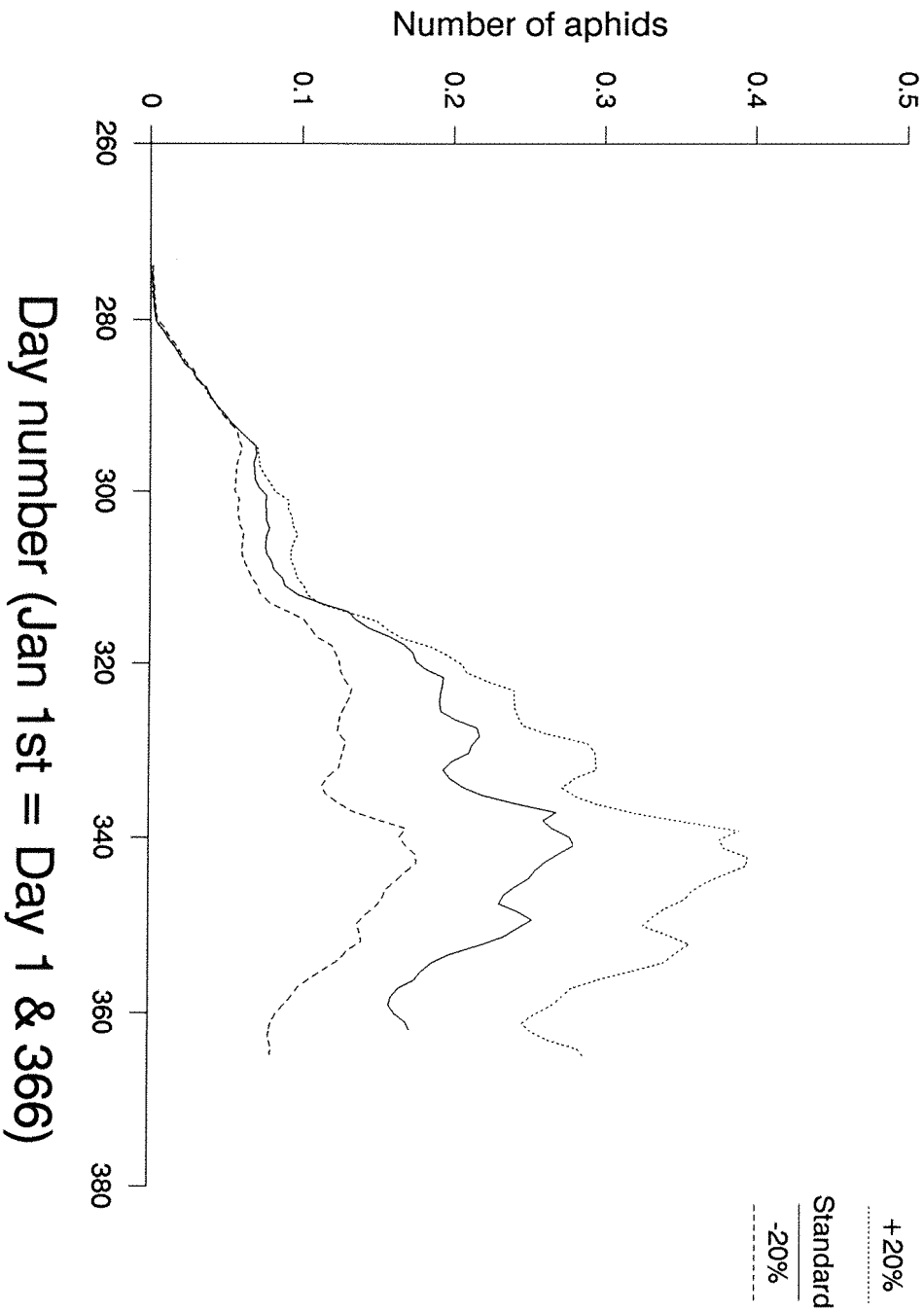
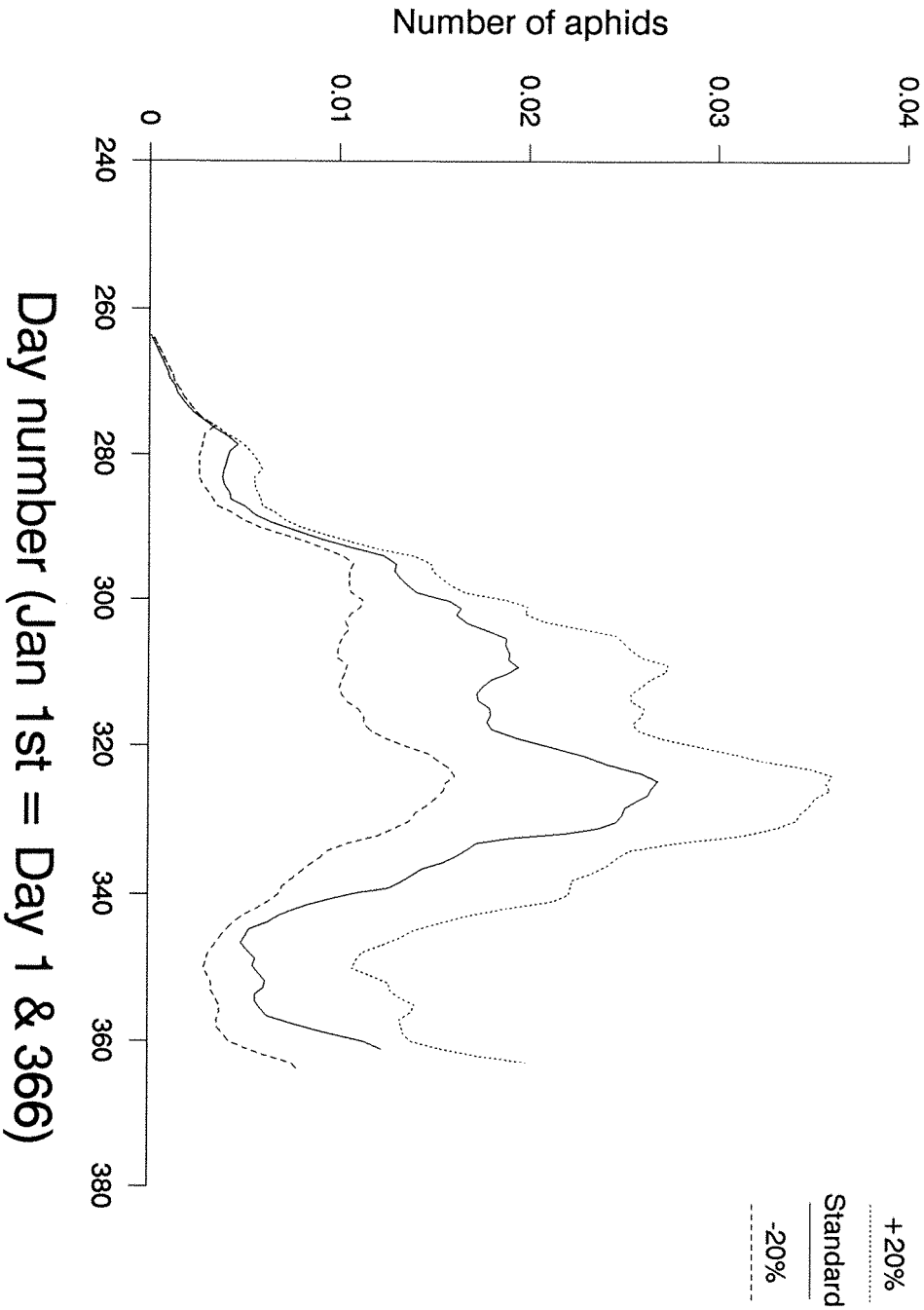


Figure 39. The effect of changes in adult longevity on density of *R. padi*, 1987/8.



7.2.3 Instar duration

An increase in the length of nymphal instars by 20% decreased peak aphid densities dramatically, by 93% in 1985/6 (Figure 40), 141% in 1986/7 (Figure 41) and 120% in 1987/8 (Figure 42), but it did not alter the timing of the peak. The response was asymmetrical, a 20% reduction in the instar longevity had a less dramatic effect on the peak population density, the maximum reduction being only 47% in 1987/8 (Figure 42). A reduction in the length of the nymphal instars brought the timing of the peak forward in 1986/7 and 1987/8 but not in 1985/6. This was probably the result of decreased instar duration advancing the onset of the reproductive maturity of adult aphids in the population to earlier in the winter when warmer temperatures increase their reproductive rate and decrease the mortality rate. However, the timing of the peak was not altered in 1985/6, probably because the temperature conditions allowed the aphids to develop and reproduce sufficiently to compensate for the increase in development time.

Because of the super-proportional effect instar duration had on the aphid population it should be measured accurately.

7.2.2 Survival

Alterations of ±5% to the survival rate of adults and nymphs had a dramatic effect on the shape and timing of the population curve. The response was asymmetrical with an increase of 5% resulting in numbers still climbing at the end of the simulations, while a 5% decrease brought forward the timing of the peak population density by anything upto 48 days and reduced it by 83% in 1985/6 (Figure 43) and 91% in 1987/8 (Figure 44).

The effect survival rates have on the development of aphid populations implies that they need to be measured accurately under natural conditions.

7.2.5 Reproductive Rate

Changing the reproductive rate had no effect on the timing of peak population densities, only their size. The response was slightly asymmetrical and super-proportional, for example a 20% increase in the rate results in a 52% increase in peak population in 1985/6 (Figure 45), a 63% increase in 1986/7 (Figure 46) and 62% in 1987/8 (Figure 47). It appears that increasing reproductive rate, increases aphid numbers and, therefore, reproductive rate needs to be measured as accurately as possible. This should be done in the field and

Figure 40. The effect of changes in instar duration on density of *R. padi*, 1985/6.

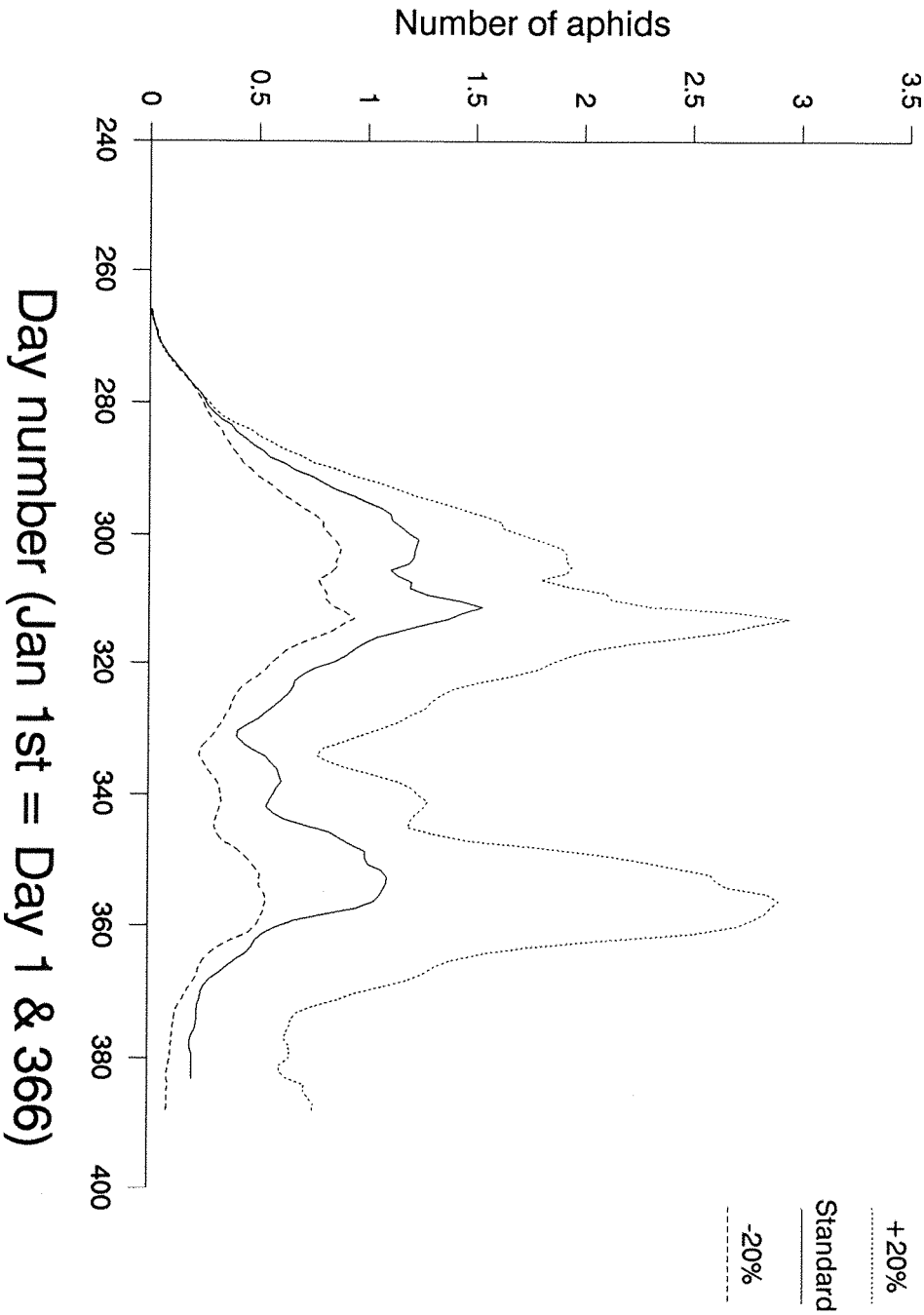


Figure 41. The effect of changes in instar duration on density of *R. padi*, 1986/7.

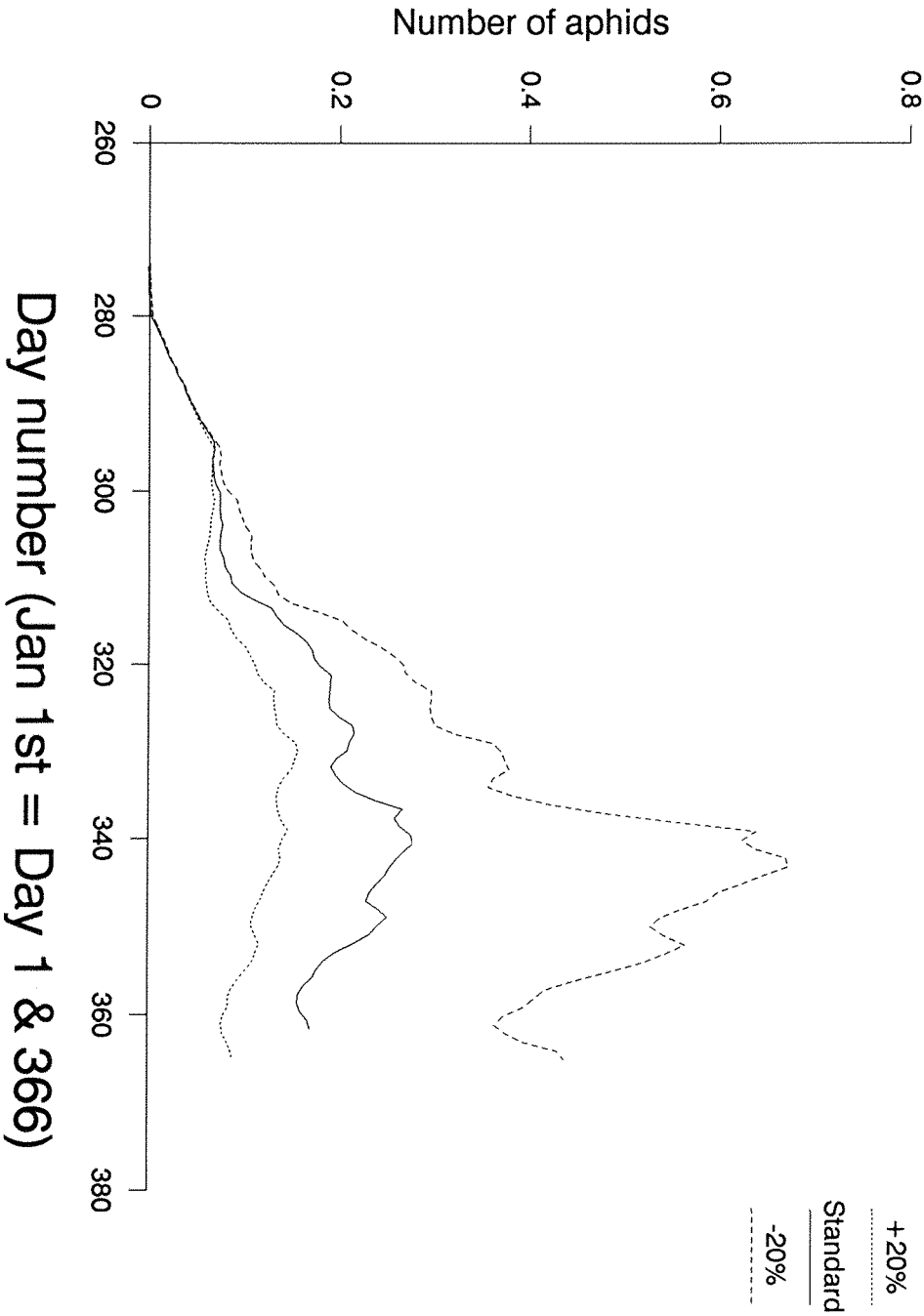


Figure 42. The effect of changes in instar duration on density of *R. padi*, 1987/8.

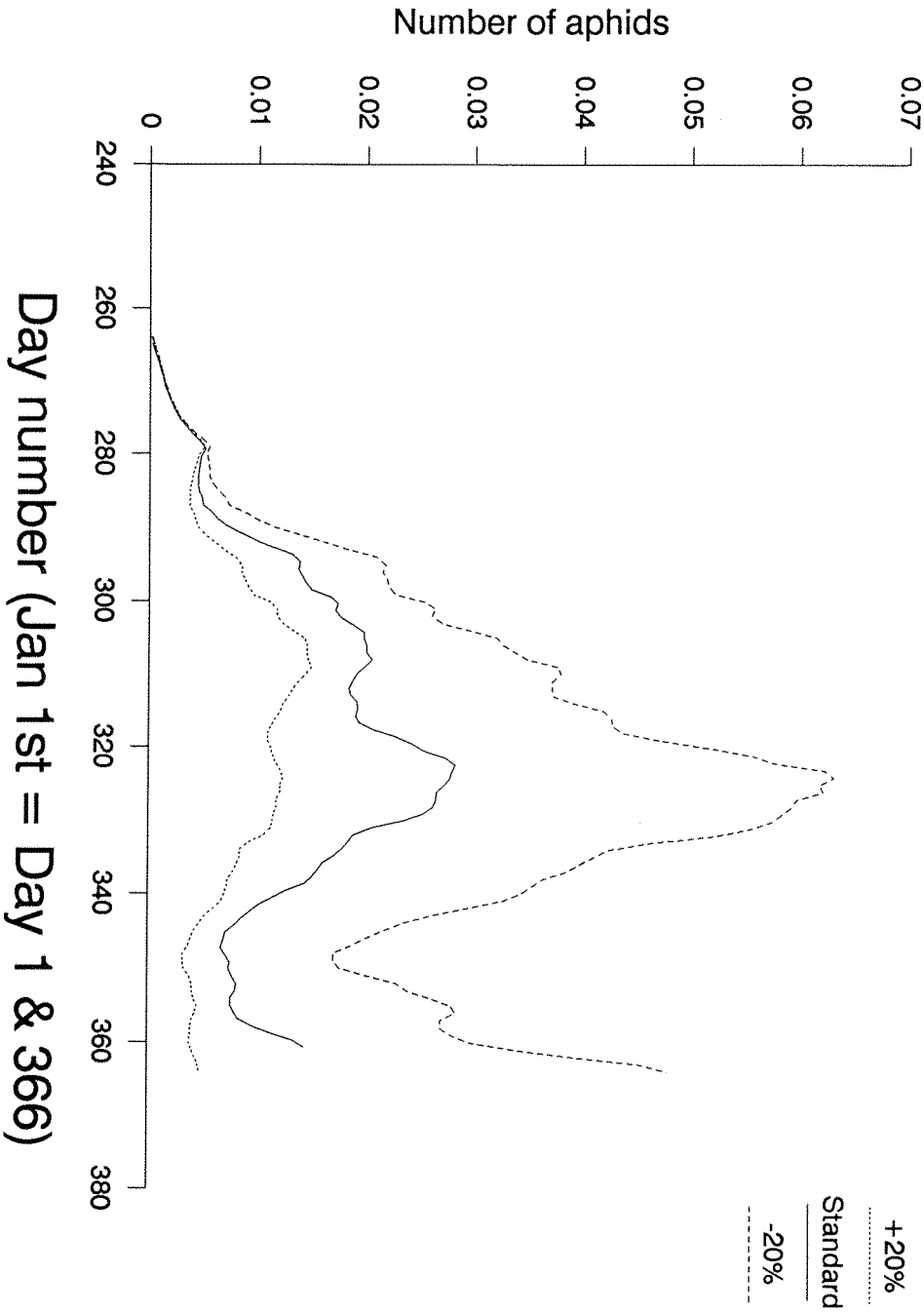


Figure 43. The effect of changes in survival on density of *R. padi*, 1985/6.

Figure 44. The effect of changes in survival on density of *R. padi*, 1987/8.

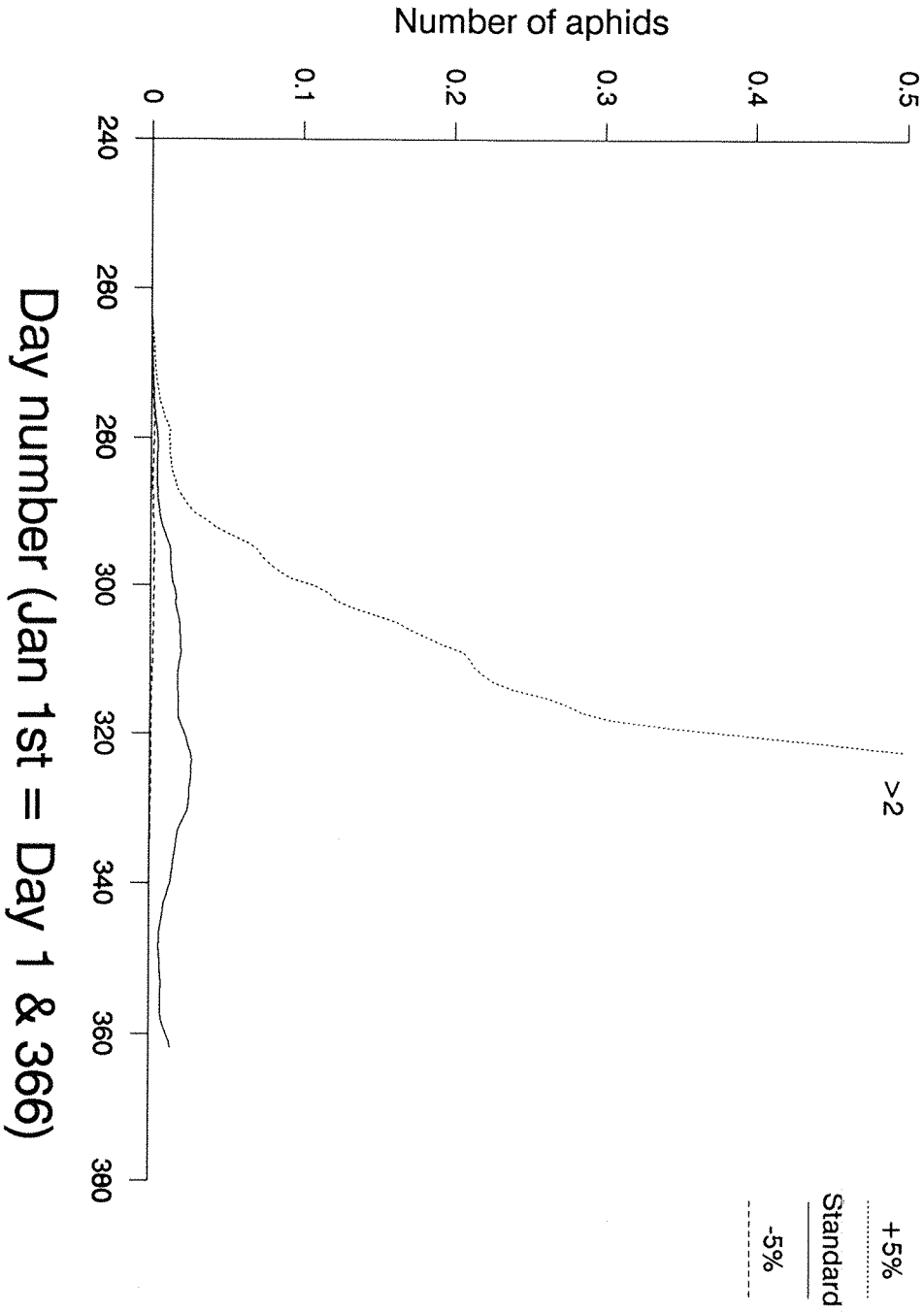


Figure 45. The effect of changes in fecundity on density of *R. padi*, 1985/6.

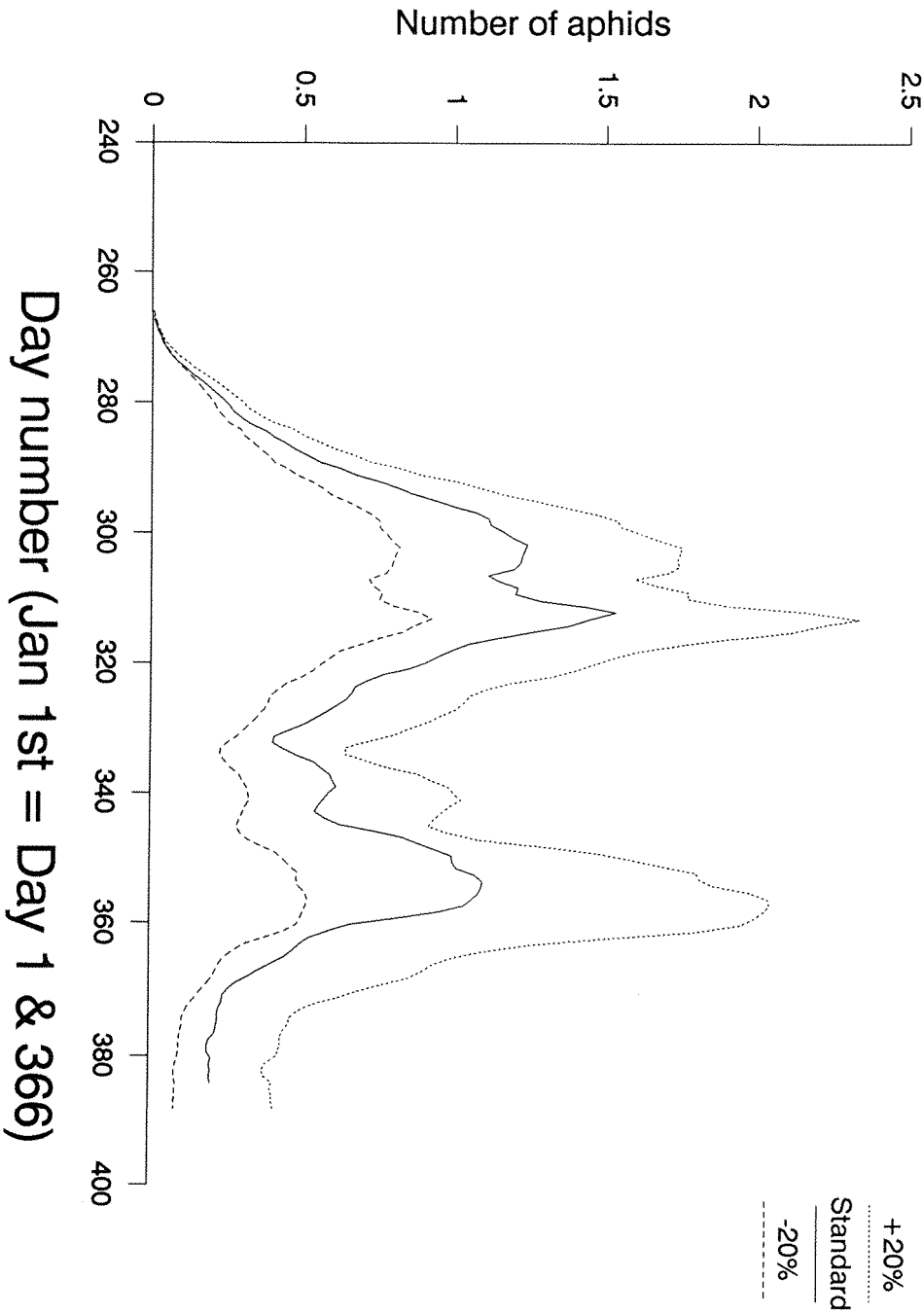


Figure 46. The effect of changes in fecundity on density of *R. padi*, 1986/7

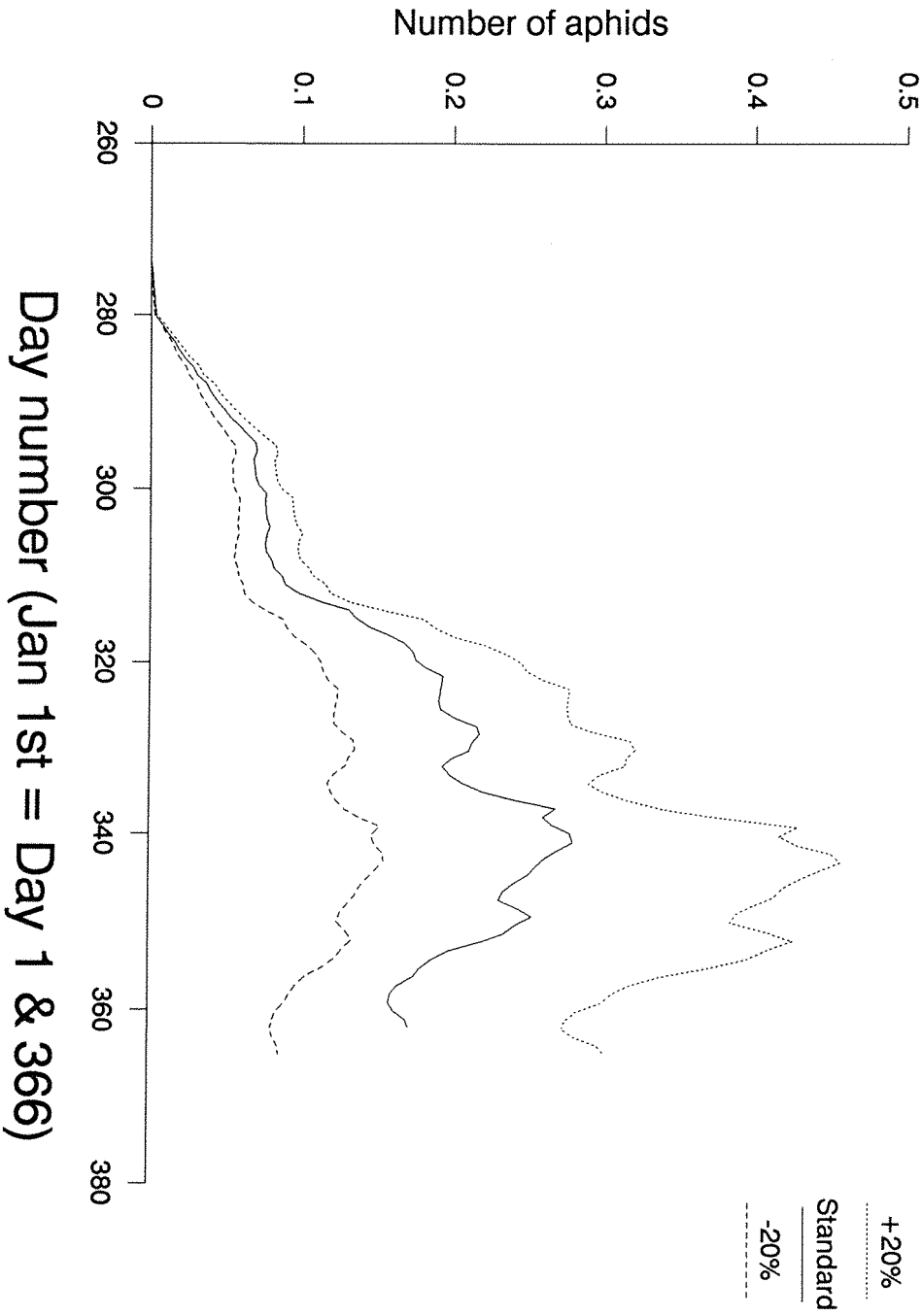
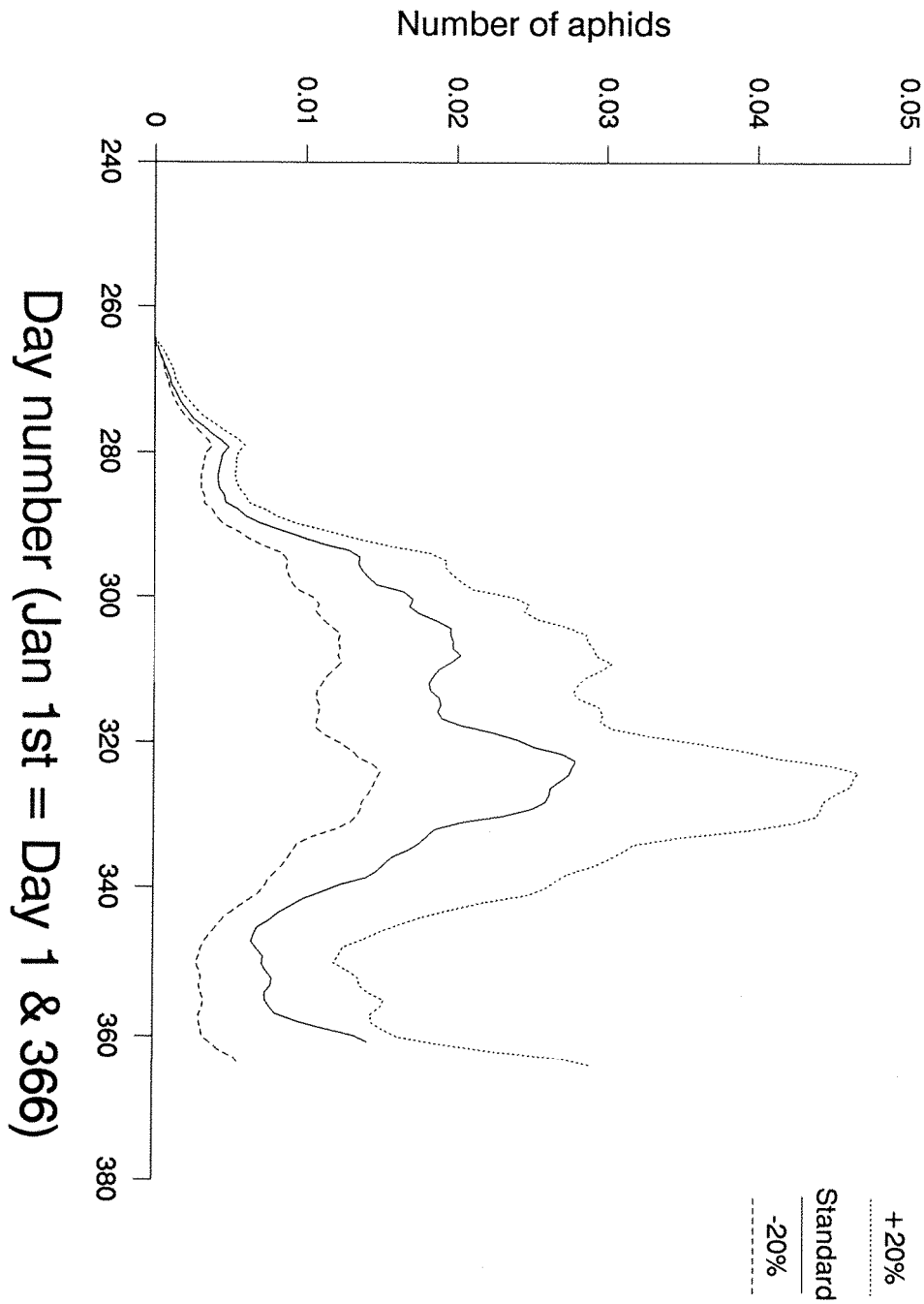


Figure 47. The effect of changes in fecundity on density of *R. padi*, 1987/8.



factors such as cultivar, nutritional status and growth stage of the host plant and aphid age should be considered.

7.2.6 Immigration

Alterations to the immigration rate have a proportional and symmetrical effect on peak aphid densities (Figures 48,49,50). The changes have a 1:1 response on the peak and so it need not be known as acurately as survival and reproductive rates, which have super-proportional effect of peak densities.

7.2.7 Morph Determination

Changing the proportion of nymphs that become alate adults did not affect the shape or the timing of the population curves (Figures 51,52,53). The proportion was changed by 50% in 1985/6 and 20% in 1986/7 and 1987/8 and even the 50% change resulted in a response of less than 1% in the peak density (Figure 51). This was probably because aphid densities are so low that the density-dependent inducement of alatiform nymphs rarely occurs and so their numbers contribute little to the total population density. Therefore, there was no need to know the proportion of alatiform nymphs accurately.

Figure 48. The effect of changes in immigration on density of *R. padi*, 1985/6.

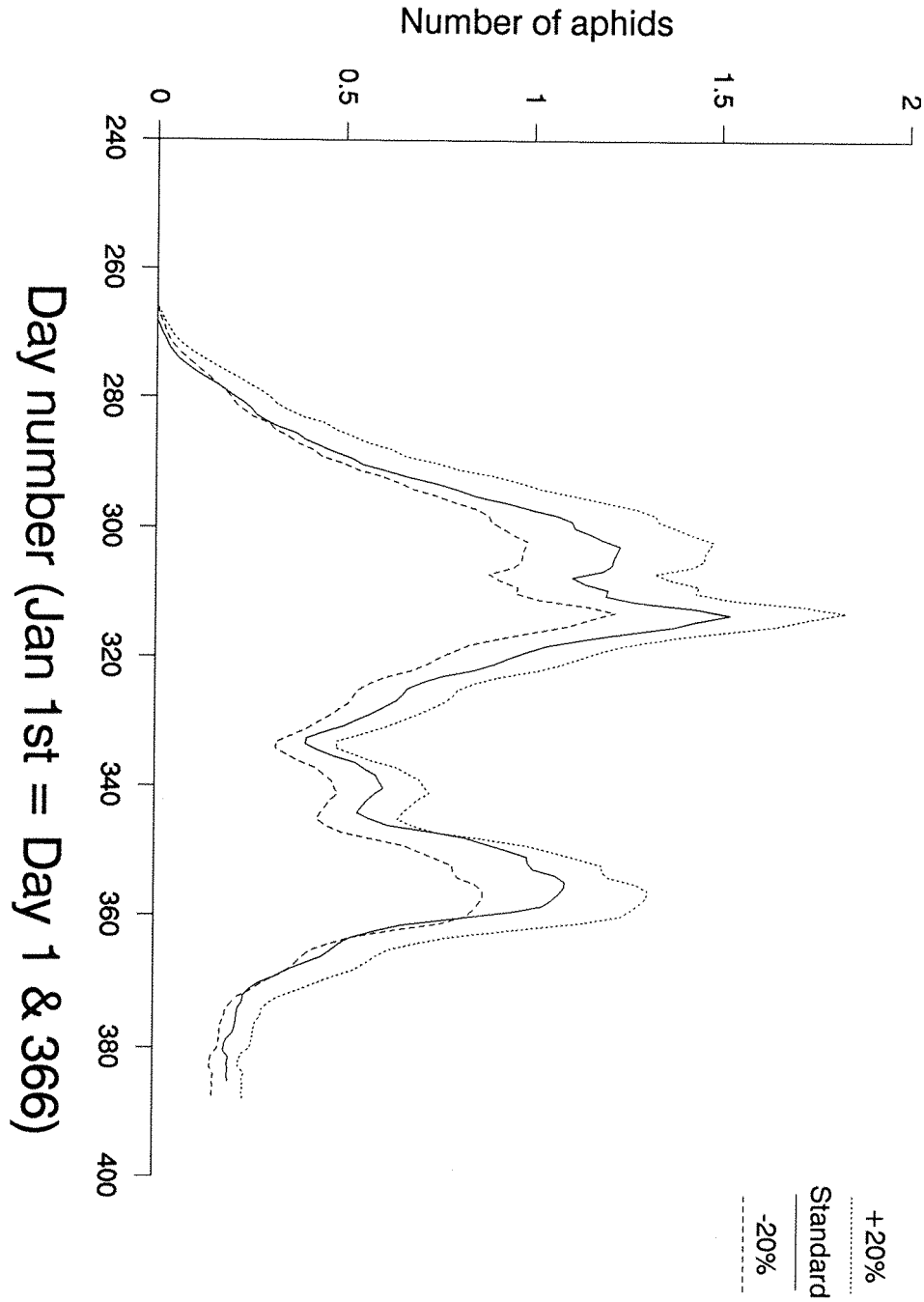


Figure 49. The effect of changes in immigration on density of *R. padi*, 1986/7.

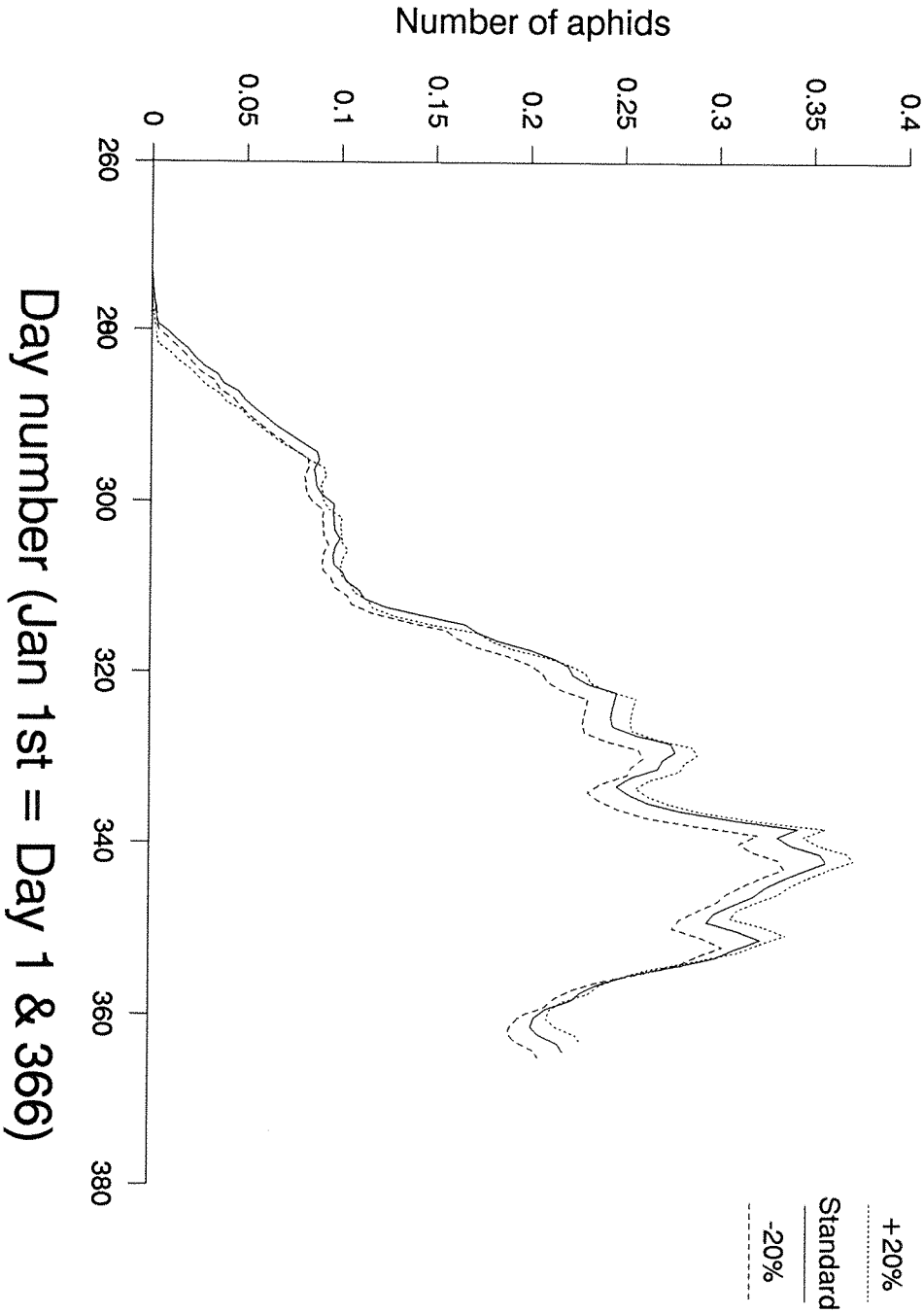


Figure 50. The effect of changes in immigration on density of *R. padi*, 1987/8.

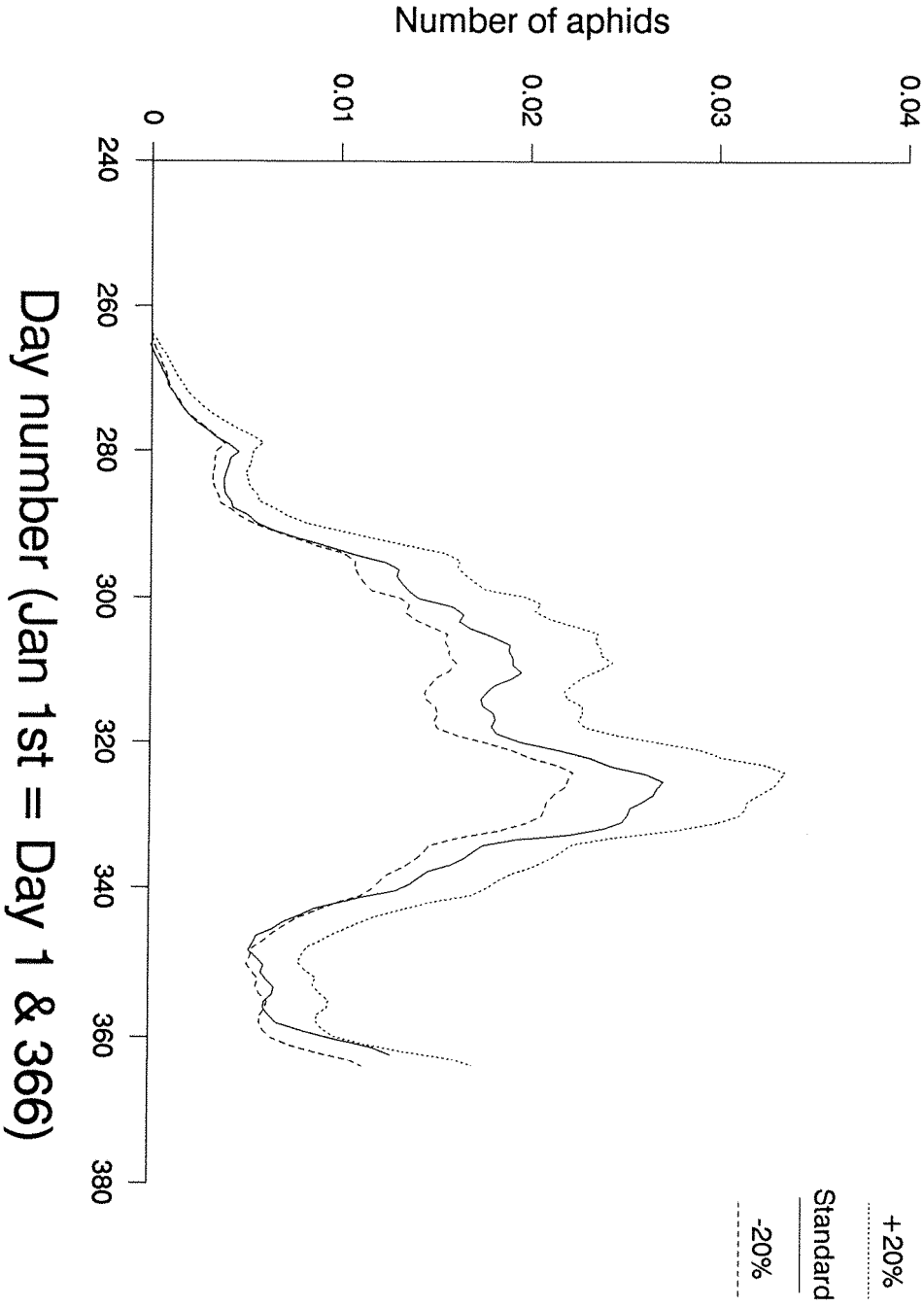


Figure 51. The effect of changes in morph determination on density of *R. padi*, 1985/6.

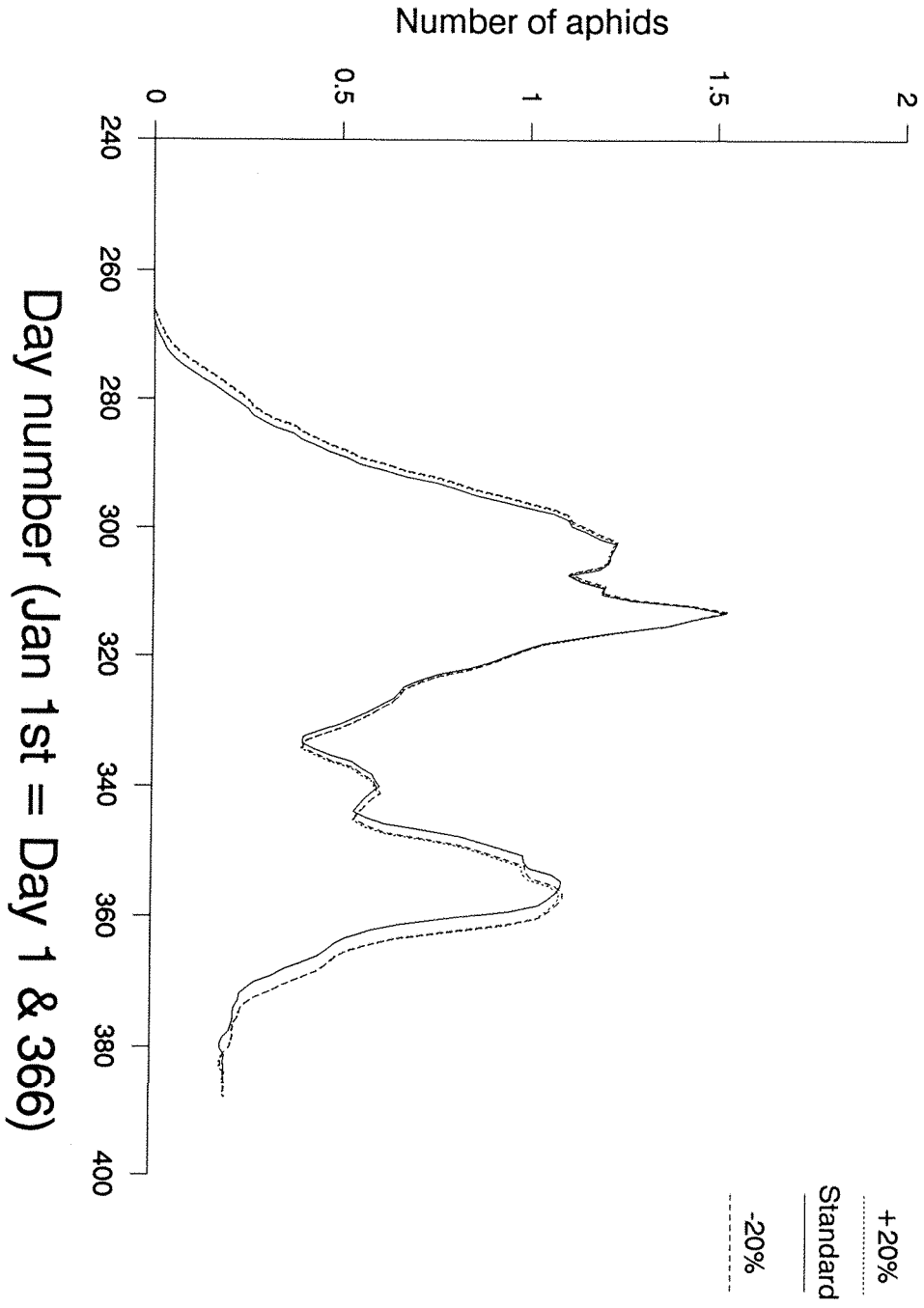


Figure 52. The effect of changes in morph determination on density of *R. padi*, 1986/7.

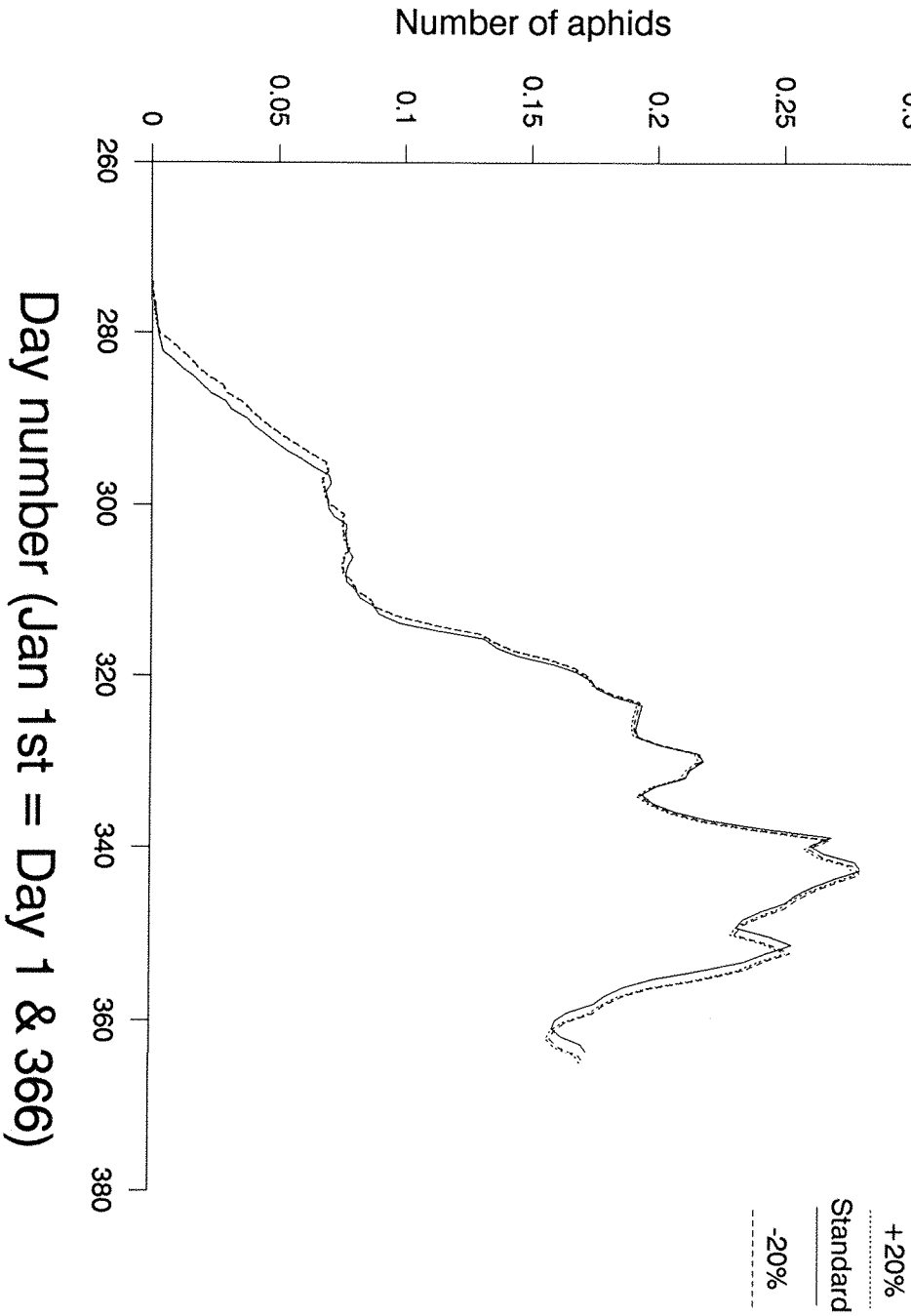
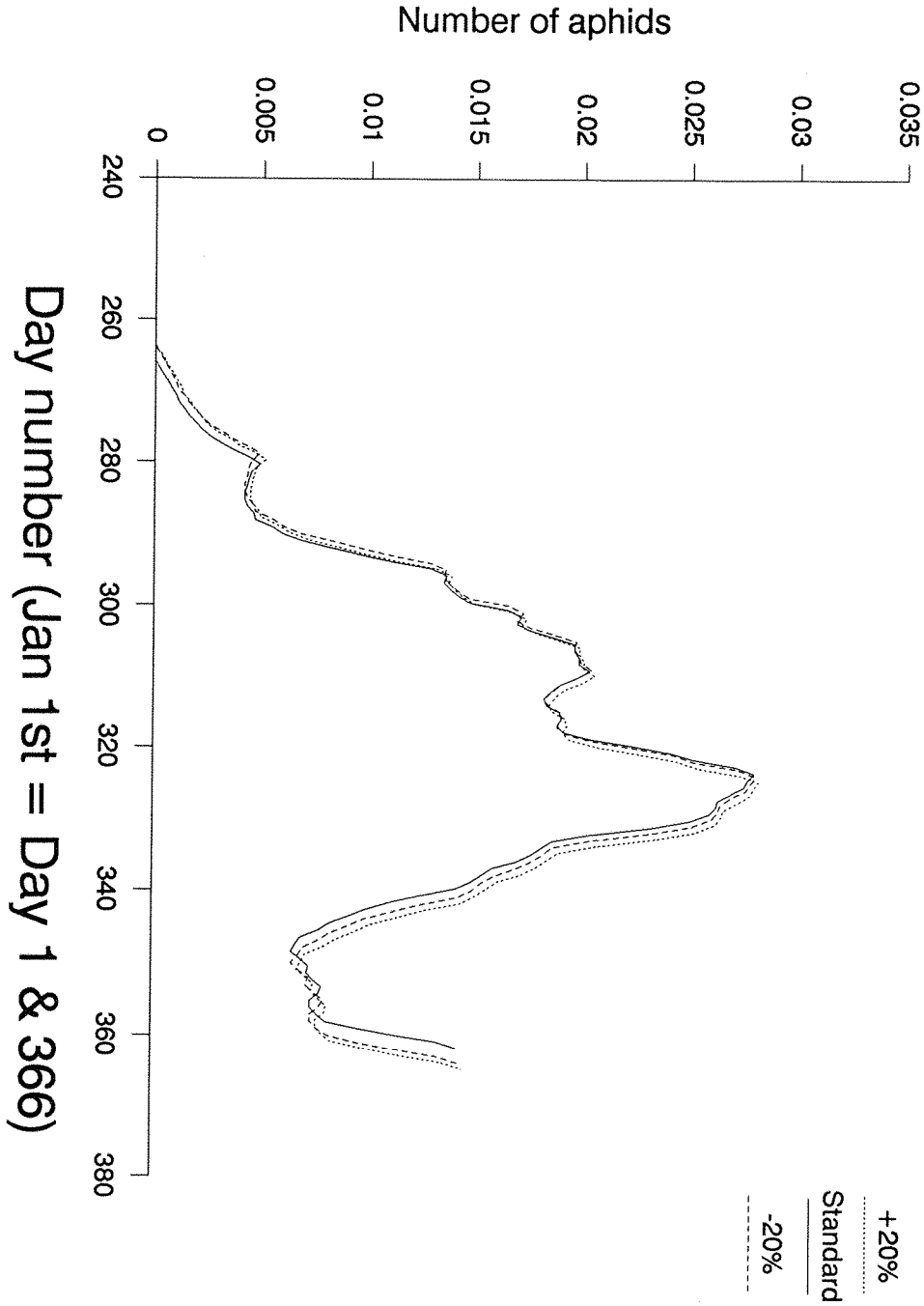


Figure 53. The effect of changes in morph determination on density of *R. padi*, 1987/8.



8. Virus Subsystem

The model consisted of four sections;
(i) Initialisation and data input.
(ii) Infection by alate aphids.
(iii) Infection by apterous aphids.
(iv) Output.

8.1 Initialisation and Data Input

Arrays used in the model were dimensioned and their elements set to zero. The parameters needed to run the model were entered into the program at the start of each simulation. These were :
(i) sensitivity factors, to change parameter values;
(ii) start and finish days of the simulation;
(iii) plant density (number/m^2) and latent period of the virus in the plant (days);
(iv) start and finish of immigration;
(v) the estimates of daily numbers of viruliferous virginoparae caught in a suction trap (See Section 4.2.1);
(vi) daily maximum and minimum temperature (^oC);
(vii) initial decimal crop growth stage;
(viii) daily rainfall (mm); and
(ix) predicted daily density of *R. padi* (number/plant).

8.2 Infection by Immigrant Alate Adults

8.2.1 Submodel

The number of previously healthy plants/m^2 that became infected with virus by viruliferous alate adults each day (ALINF) was calculated by multiplying an estimate of the number of viruliferous virginoparae caught in the nearest RIS 12.2m suction trap (VIMM(DAY)) by a deposition factor

ALINF=VIMM(DAY)*0.0237

8.2.2 Data

The method used in Section 6.2.1 was used to estimate the numbers of virginoparae caught in the 12.2m suction trap. Alates were assumed to have a density gradient of -1.0, a flight time of 2h and to land randomly in the crop (Taylor & Palmer, 1972; Section 6.2.1) and so for each alate caught in a suction trap, 0.0237 would land per square metre.

Virus infectivity trials of alate aphids caught live in a 1.5m suction trap (Section 6.2.2) were used to estimate the proportion of aphids carrying virus.

8.3 Infection by Apterous Aphids

Two versions describing the spread of virus by apterous aphids were developed.

8.3.1 Version 1

8.3.1.1 Submodel

It was assumed in the submodel that new inoculations were aggregated around the original foci. The negative binomial distribution was used to calculate the number of previously healthy plants (HLTHY) that became infected each day (APINF)

$$APINF=HLTHY*(1-(k/(k+INOC/PLANTS))^{k})$$

where INOC was the number of inoculations made each day, PLANTS the total number of plants present and k the negative binomial distribution parameter.

In a negative binomial distribution with a mean of x per sample (here the number of infected plants/m^2) and a parameter, k, the probability of drawing a sample with nothing in it (no infected plants) is

$$k/(k+x)^{k}$$

Assuming that there is no distinction between an aphid inoculating a healthy or an infected plant, x becomes

$$INOC/PLANTS$$

so that the probability of a plant receiving one or more inoculations on a given day is

$$1-(k/(k+INOC/PLANTS)^{k})$$

Multiplying this by the number of healthy plants available gives the number that become infected each day (APINF).

The number of inoculations made each day was calculated using

INOC=PLDEN(DAY)*PROBAC

where PLDEN(DAY) was the density of aphids predicted by the aphid subsystem DEN(DAY), number per plant, multiplied by 300, the crop sowing rate, so that aphid density was in terms of numbers per square metre and PROBAC is the probability that an aphid sampled at random from the popualtion would be viruliferous.

PROBAC was calculated by dividing the number of source plants on the previous day (SORC(DAY-1)), a source plant being one in which the virus had replicated and been translocated throughout the plant so that a feeding aphid could acquire sufficient virus to transmit to a healthy plant, by the product of the number of plants per square metre (PLANTS) and the proportion of plants infested with one or more aphids (INC).

PROBAC=SORC(DAY-1)/(PLANTS*INC)

How SORC(DAY-1) was calculated will be discussed later. INC was estimated using the Nachman model (Nachman, 1981; Perry, 1987) which related the proportion of empty sample units (Γ_0, the proportion of uninfested plants) to the mean density of aphids (π)

$$\log_e(-\log_e(\Gamma_0))=\log_e(\alpha)+ß*\log_e(\pi)$$

where α and ß are the intercept and the regression coefficient, respectively. Hence

$$\Gamma_0=EXP(-EXP(\log_e(\alpha)+ß*\log_e(\pi)))$$

and

$$INC=1-\Gamma_0$$

The negative binomial distribution parameter, k, was estimated (KPARA) using the equation (Taylor, Woiwod & Perry, 1979)

$$KPARA=1/(a*m^{b-2}-m^{-1})$$

where a and b were estimated from the power-law (variance(s^2)=a*mean(m)^b) relationship (Taylor, 1961)

$$\log_{10}(s^2)=\log_{10}(a)+b*\log_{10}(m)$$

It was assumed that each plant in a field was in one of three conditions. It was either: 1. healthy (it contained no virus), 2. latent (the virus was replicating and translocating within the plant

but its titre was too low for a probing aphid to acquire sufficient virus to infect a healthy plant), or, 3. source (when the virus titre was sufficiently high for acquisition by an aphid and transmission to a new host plant).

It was assumed that the day after a plant was infected it entered the latent condition (LTNT(DAY)) and after it had undergone a latent period (LP) it became a source plant (SORC(DAY)). Therefore the number of latent plants was calculated using

$$LTNT(DAY) = \sum_{i=j}^{l} INFEC(i)$$

where j and l are the limits of the summation (j=DAY-LP+1 and l=DAY-1) and INFEC(i) is the sum of the number of plants infected by immigrant alates and the number infected by apterous aphids, the number of source plants using

$$SORC(DAY) = \sum_{i=1}^{n} INFEC(i)$$

where n is the upper limit of the summation (n=DAY-LP), the number of healthy plants using

HLTHY=PLANTS-LTNT(DAY)-SORC(DAY)

The percentage of plants infected with virus (PCINF) was calculated by subtracting the number of healthy plants from the total number of plants, dividing by the total number of plants and multiplying by 100.

9.3.1.2 Data

Estimates of $\Gamma_0(P_0)$ and $\pi(t)$ from field samples of naturally occurring populations of *R. padi* at Rothamsted in 1985/6, 1986/7 and 1987/8 (Chapter 4) were used to fit a linear regression between Γ_0 and π

P_0=EXP(-EXP(-0.7259+0.8751*$\log_e$(t))) r=0.899 df=55 p<0.001

INC was calculated by substituting the predicted aphid density (DEN(DAY)) for t in the above equation and subtracting the result from 1.0

INC=1.0-(EXP(-EXP(-0.7259+0.8751*ALOG(DEN(DAY)))))

Parameter values for a and b were estimated by fitting a linear regression to field estimates of s^2 and m collected at Rothamsted in 1985/6, 1986/7 and 1987/8 (Chapter 4)

$$\log_{10}(s^2)=-0.907+1.288*\log_{10}(m) \quad r=0.768 \quad df=55 \quad p<0.001$$

Therefore, k was estimated (KPARA) using the values of a and b, from the above equation, with the predicted aphid density (DEN(DAY))

$$KPARA=1/(0.124*(DEN(DAY))^{-0.712}-(DEN(DAY))^{-1})$$

The latent period of BYDV between 15°C and 20°C is 6 days (van der Broek & Gill, 1980).

8.3.1.3 Appraisal of Version 1

When fitting the power-law variance-mean relationship (Taylor, 1961) the mean sample densities were always greater than the variances. This contradicted a basic assumption of the negative binomial distribution, the mean density must always be less than the variance and so indicated that the negative binomial distribution was inappropriate and should not be used.

8.3.2 Final Version

It was decided that a measure of the rate of BYDV spread should be used rather than trying to use a probability distribution to describe the spread.

8.3.2.1 Submodel

The number of previously healthy plants that became infected each day because of apterous vectors (APINF) was calculated by multiplying the density of vectors (PVECS), a transmission coefficient (with a value of 0.0166 newly infected plants per vector per day), the proportion of aphids that were knocked off a plant by rainfall (RKNOC) and a coefficient of shelter provided by the crop growth stage from the effects of the rain (PKNOC)

$$APINF=PVECS*0.0166*(1+RKNOC)*PKNOC$$

PVECS was calculated using the same method used in Section 7.3.1.1 to calculate the number of inoculations made each day (INOC).

The effect of rainfall (RAIN(DAY)) on disturbing aphids feeding on plants was investigated experimentally and two equations were used to describe the effect

RKNOC=0.0208*RAIN(DAY) If RAIN(DAY)<3.6mm

RKNOC=0.05+0.0167*RAIN(DAY) If RAIN(DAY)≥3.6mm

It was found that aphids on young plants at decimal growth stage 11 (Zadoks, Chang & Konzak, 1974), were dislodged from the plants by rain on 50% more occasions than aphids on older plants at decimal growth stage 21 because the latter sheltered the aphids more from the rain. Two equations were used to estimate a coefficient of shelter (PKNOC) with crop growth stage represented by accumulated physiological time (DDP, DDP=430 was equivalent to a decimal growth stage of 21)

PKNOC=1.5 If DDP<430

PKNOC=1.0 If DDP≥430

The numbers of infections by alate and apterous adults, latent, source and healthy plants and percentage infection were calculated by the same procedures as used in Section 7.3.1.1.

8.3.2.2 Data

8.3.2.2.1 Calculation of Transmission Coefficient

An experiment to determine the transmission coefficient was carried out in a glasshouse (University of Southampton) between 25 April and 21 May, 1986.

8.3.2.2.1.1 Materials and Methods

Maximum and minimum temperatures were 37°C and 9°C, respectively, under a natural light regime.

Winter barley, cv Sonja, was sown in trays (55x55x5cm) in five rows each with eleven plants, with 5cm between plants and 10cm between rows (a sowing rate of 300 seeds per square metre). When the seedlings had reached the two leaf stage, decimal growth stage 12 (Zadoks, Chang & Konzak, 1974), alate aphids, which had been given an acquisition feed of 72h on BYDV-infected plants, were clip-caged onto the central seedling, one aphid per tray. The clip-cages and adults

were removed after 48h and the nymphs, numbering between seven and ten, were allowed to move freely.

Six trays were sampled on each of three occasions, eight, fifteen and twenty-two days after the removal of the adults and clip-cages. All aphids were collected and each tray was sprayed with a synthetic pyrethroid to kill any remaining aphids to prevent further virus spread. The aphids collected were placed individually onto virus-sensitive oat seedlings, cv Maris Tabbard, for 2 days. After which the aphids were removed and the plants grown for a further 14 days to determine the number of virulent aphids (Plumb, 1983). The plants in the trays were grown for a further fourteen days after which a visual inspection of virus symptoms was made to determine the number of plants infected.

8.3.2.2.1.2 Results

The number of infective aphids (w) increased with time (t) and the number of healthy plants (X_t) decreased exponentially from the initial number of healthy plants (X_0)

$$X_t=X_0*EXP(-a*w*t)$$

$$\log_e(X_t/X_0)=-a*w*t$$

where "a" was the coefficient of virus transmission efficiency (Nakasuji, Miyai, Kawamoto & Kiritani, 1985; here shortened to 'transmission coefficient').

The absolute value of the slope from regressing $\log_e(X_t/X_0)$ against w*t (constraining the regression through the origin) gave an estimate of "a", 0.0166 newly infected plants per vector per day (Figure 54; $r=-0.75$ $df=16$ $p<0.001$).

8.3.2.2.2 Determining the Effect of Rainfall

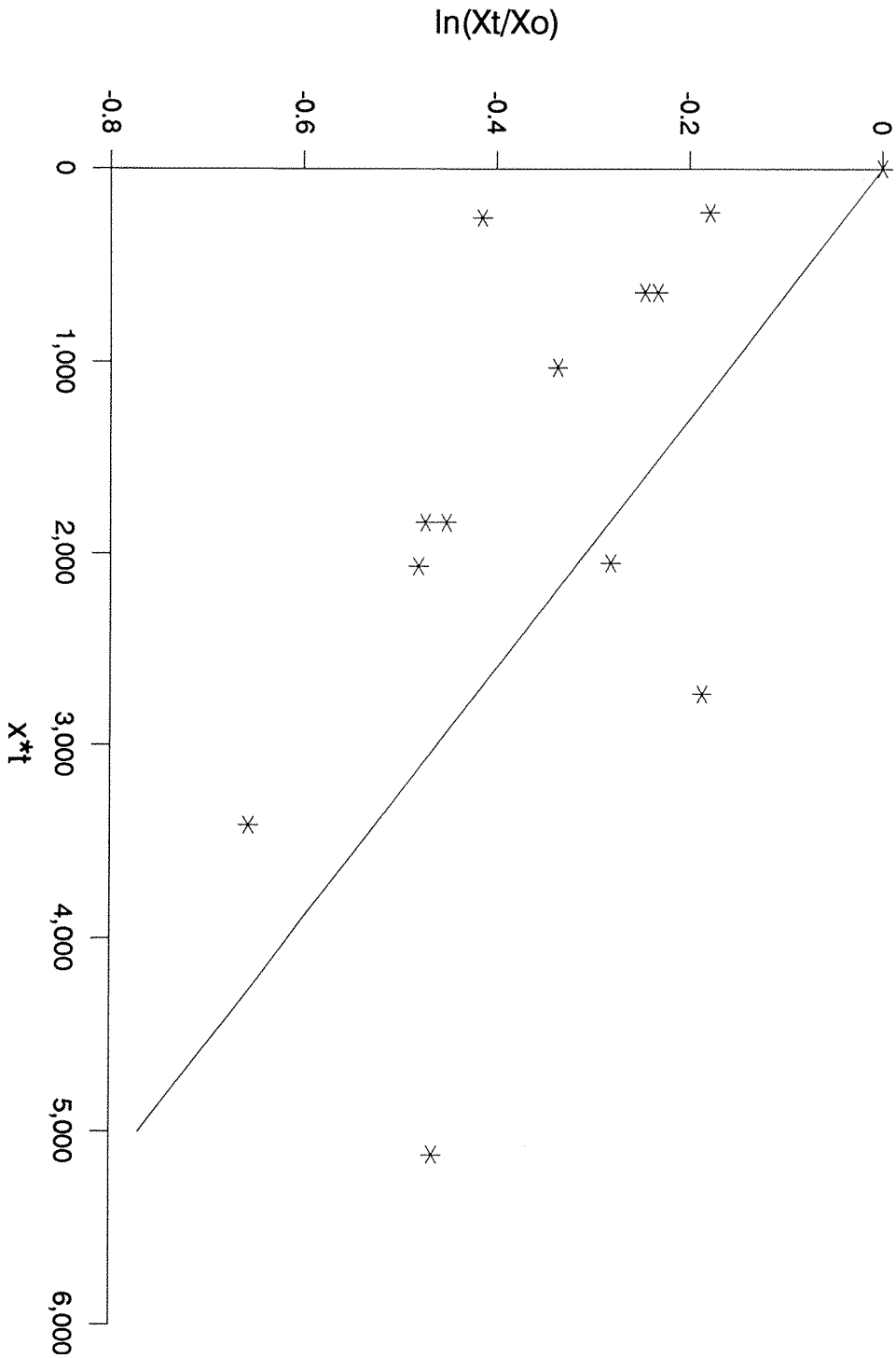
The effects of rainfall on aphid dispersal were determined by laboratory experiments. Although field investigations would have been more realistic the measurements would have been more difficult and there would have been less control. The experiments were aimed at answering three questions:

(i) Did rainfall cause aphids to behave differently in different age classes?

(ii) Did different plant growth stages offer different degrees of protection to the aphids from the rain?

(iii) Did rainfall intensity affect the number of aphids dislodged?

Figure 54. Regression of l $og_e(X_t/X_0)$ against w*t(X_t is number of healthy plants after time t, X_0 is initial number of healthy plants, w is the number of vectors and t is time in days).



8.3.2.2.2.1 Materials and Methods

Winter barley seedlings, cv Sonja, were grown individually in 10cm pots. When either the plants had one leaf fully expanded, decimal growth stage 11, or had began tillering, decimal growth stage 21, six fourth instar or apterous adult aphids were clip-caged onto the first fully expanded leaf of the main shoot of each plant and left to settle. After two days the clip-cages were removed and the numbers of aphids feeding, and their instars, were recorded. Sixteen pots, arranged in a 4x4 square, were placed at the bottom of a rain tower (See Fitt et al., 1986). Rain fell on the pots at a rate of $1cm^3$/min for 5min or $20cm^3$/min for 15min, after which the pots were carefully removed and the plants examined to determine how many ahids had been dislodged. The rainfall rate was measured by placing a glass measuring cylinder beneath a stream of rain drops before and after each experiment for 1min and recording the volume of water collected (Fitt et al., 1986).

The effect of rainfall on dislodging aphids in different age-classes was investigated by removing the fourth instar and apterous aphids with the clip-cages and leaving a similar number of first and second instar nymphs.

8.3.2.2.2.2 Results

The proportions of aphids dislodged by the rain are given in Table 8. The proportion of first or second instar nymphs dislodged was not significantly different from that of fourth instar or apterous adults ($p>0.05$). Therefore, no distinction was made between aphids of different ages in their response to rain.

Tillering plants gave significantly more protection to aphids from the rain than plants with only one leaf (Table 8, $p<0.001$). An aphid on a one leaf plant was 50% more likely to be dislodged than an aphid on a older plant.

The more rain that fell the greater the number of aphids that were dislodged (Table 8, $p<0.001$). Rainfall recorded in the field is measured as the height of water which falls (in mm), the volume of water that had fallen in the experiment was converted into height (height=volume/internal area of the glass cylinder, area=π*(internal diameter of the cylinder)^2) so that the data could be entered into the model. It was assumed that if no rain fell then no aphids would be dislodged from their plants. Therefore, two linear regressions were used to calculate the proportion of aphids knocked off (RKNOC) by rainfall (RAIN(DAY))

Table 8. The effects of aphid instar, plant growth stage and rainfall intensity on aphid dispersal caused by rainfall.

	Aphid instar		Growth stage		Rain	
	Young	Old	1 leaf	1 tiller	Light	Heavy
mean	0.80	0.74	0.36	0.74	0.74	0.15
s.e.	0.06	0.05	0.10	0.05	0.05	0.05

RKNOC=0.0208*RAIN(DAY) If RAIN(DAY)<3.6mm

RKNOC=0.05+0.0167*RAIN(DAY) If RAIN(DAY)≥3.6mm

where 3.6mm is the amount of rain that fell with a rainfall rate of $1cm^3/min$ for 5min.

Since these equations were fitted using data involving tillering plants and it had been shown that rainfall had a greater effect on aphids feeding on younger plants the relationships had to be modified to take this into account. Therefore, the degree of shelter provided to aphids by plants (PKNOC) was calculated. Crop growth stage was represented by accumulated physiological time (DDP). Field observations of crop growth had shown that a plant required at least 430 day-degrees above 5^oC from the time when it was sown before it began tillering. Therefore, if DDP was less than 430, equivalent to a growth stage less than 21, than the aphids were 50% more likely to be dislodged than if DDP was greater than, or equal to, 430 for the same amount of rainfall

PKNOC=1.5 If DDP<430

PKNOC=1.0 If DDP≥430

9. Model Predictions of Virus Spread

9.1 Introduction

The model was validated by a visual comparison of output with field results. The model was considered accurate if its predictions fell within one standard error of the observed results (standard errors are given as vertical bars on all graphs).

9.2 Results and Discussion

9.2.1 1985/6 - Sowing date : 13th September

The model predicted the peak percentage of infected plants accurately but underestimated the amount of virus found in mid-November (Figure 55). In autumn 1985 volunteers, plants from the previous cereal crop, were present in the field. These could have acted as reservoirs of virus, and aphids feeding on them could have acquired the virus and increased the pressure of virus inoculum on the crop. Therefore the disease could have been introduced into the experimental plots by means other than infectious alate aphids colonizing the crop. If virus reservoirs were present, a higher incidence of virus would probably have occurred at an earlier stage than expected. It was also possible that factors such as high wind or natural enemy disturbance of aphids, forced them to move and could have been responsible for the higher incidence of virus found early on in the field.

9.2.2 1985/6 - Sowing date : 23rd September

The model underestimated the amount of virus found in the field (Figure 56). Cereal volunteers acting as reservoirs for the virus could have been responsible again for this, although the aphid sub-model also underestimated the number of aphids found in the field (Figure 13). The virus sub-model might, therefore, have under-estimated virus spread because too few aphids were predicted. Asensitivity ananlysis altering vector density will be carried to investigate this.

9.2.3 1985/6 - Sowing date : 4th October

More virus was found in the experimental plots than predicted by the model (Figure 57). This difference cannot be attributed to the aphid sub-system underestimating the number of aphids found in the field (Figure 16). This could, therefore, implicate other factors not currently considered in the model, such as local virus reservoirs,

Figure 55. Observed and predicted BYDV incidence in 1985/6, sowing date 13 Sept.

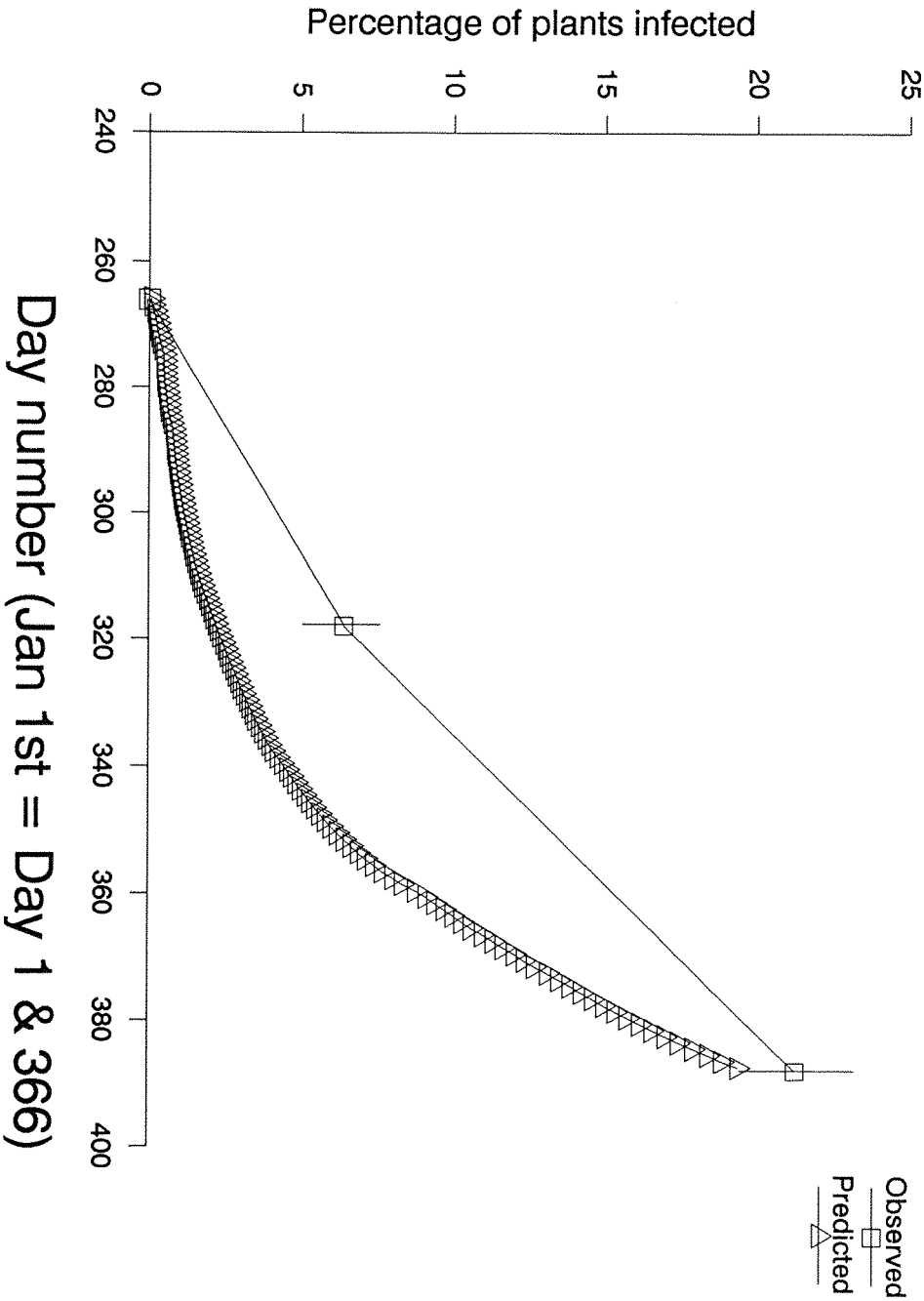


Figure 56. Observed and predicted BYDV incidence in 1985/6, sowing date 23 Sept.

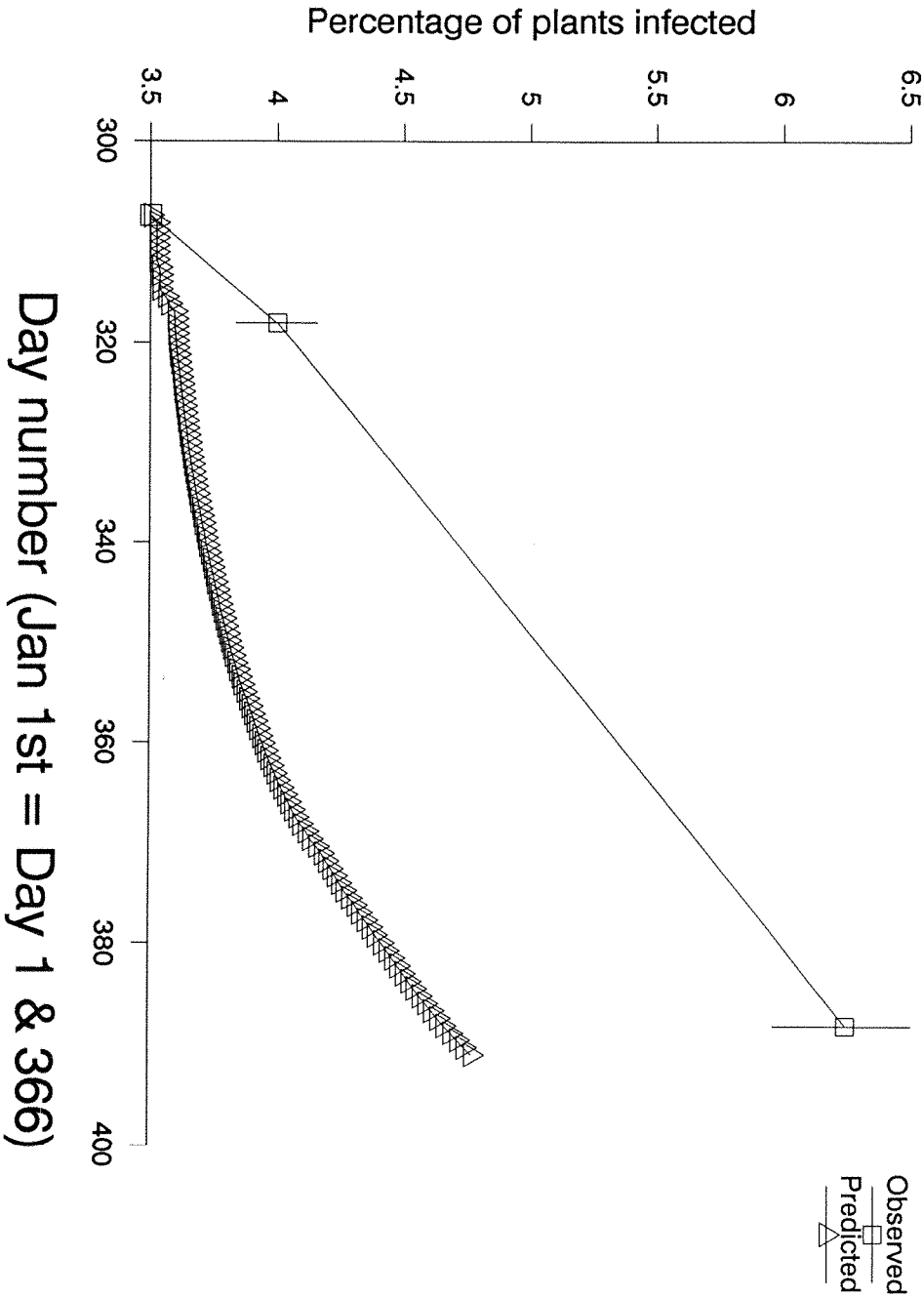
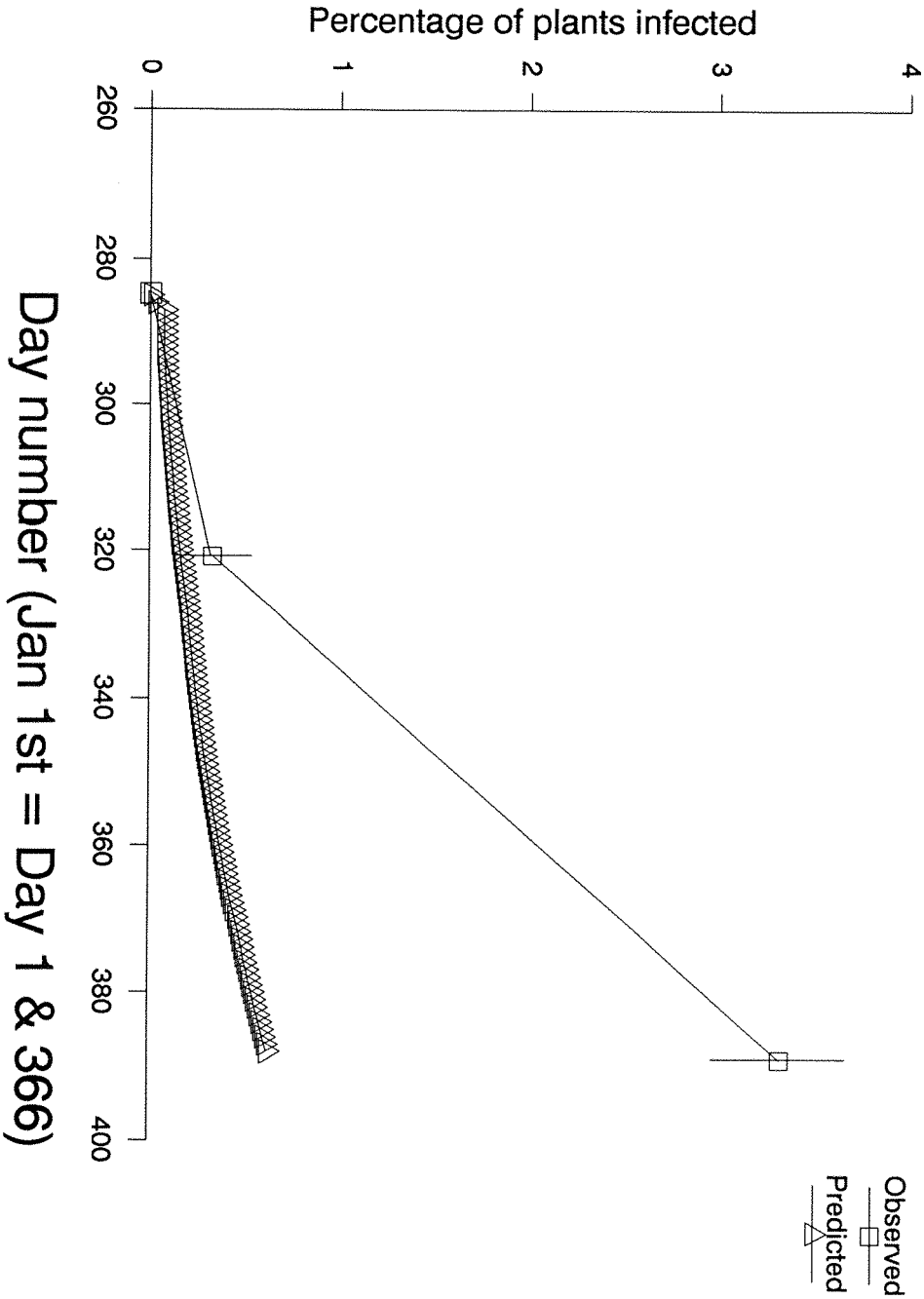


Figure 57. Observed and predicted BYDV incidence in 1985/6, sowing date 4 Oct.



strong winds or natural enemies, which may cause vectors to disperse.

9.2.4 1986/7 - Sowing date : 12th September

The model overestimated the incidence of the very small amount of virus found in the field (Figure 58). Virus incidence was determined by visual inspections of the crop and no infected plants were found, although the model predicted a maximum of 0.13% of the plants to be infected. However the difference between observed and expected incidence was very small, equivalent to only 0.36 infected plants/m^2.

The virus incidence in later sown plots was so small that it was decided not to carry out simulations for these.

9.2.5 1987/8 - Sowing date : 10th September

The model predicted that 0.12 infected plants/m^2 would be found although no infected plants were found in the field (Figure 59). The difference between observed and predicted virus spread was less than found in 1986/7 probably because fewer aphids were predicted by the aphid sub-system in 1987/8. Virus incidence was low in later sown plots and no further simulations were carried out.

9.3 Discussion

Validation of the virus sub-system has revealed that further work was required to improve the accuracy of its predictions. In 1985/6 the model underestimated the amount of virus in mid-November in all three sowings. One possible explanation was that the presence of cereal volunteers, which acted as virus and vector reservoirs and could have caused more virus to be introduced to the crop earlier and in greater quantities than predicted from immigrant infections by alate aphids. A possible solution to this would be to create a procedure within the model whereby field recordings of numbers of volunteers are input into the model. However, the importance of these reservoirs in virus spread, considering virus strain, distance of volunteers from crop plants, growth stage of volunteers and vector populations need to be determined. Also improving the predictions of aphid numbers by the aphid sub-system could improve the fit of the predictions from the virus sub-system. This could be achieved by developing a procedure in which the number of aphids observed in the field would be 'driving' the model.

The effects of wind and natural enemies dislodging aphids and the influence of crop growth stage increasing the contact with

Figure 58. Observed and predicted BYDV incidence in 1986/7, sowing date 12 Sept.

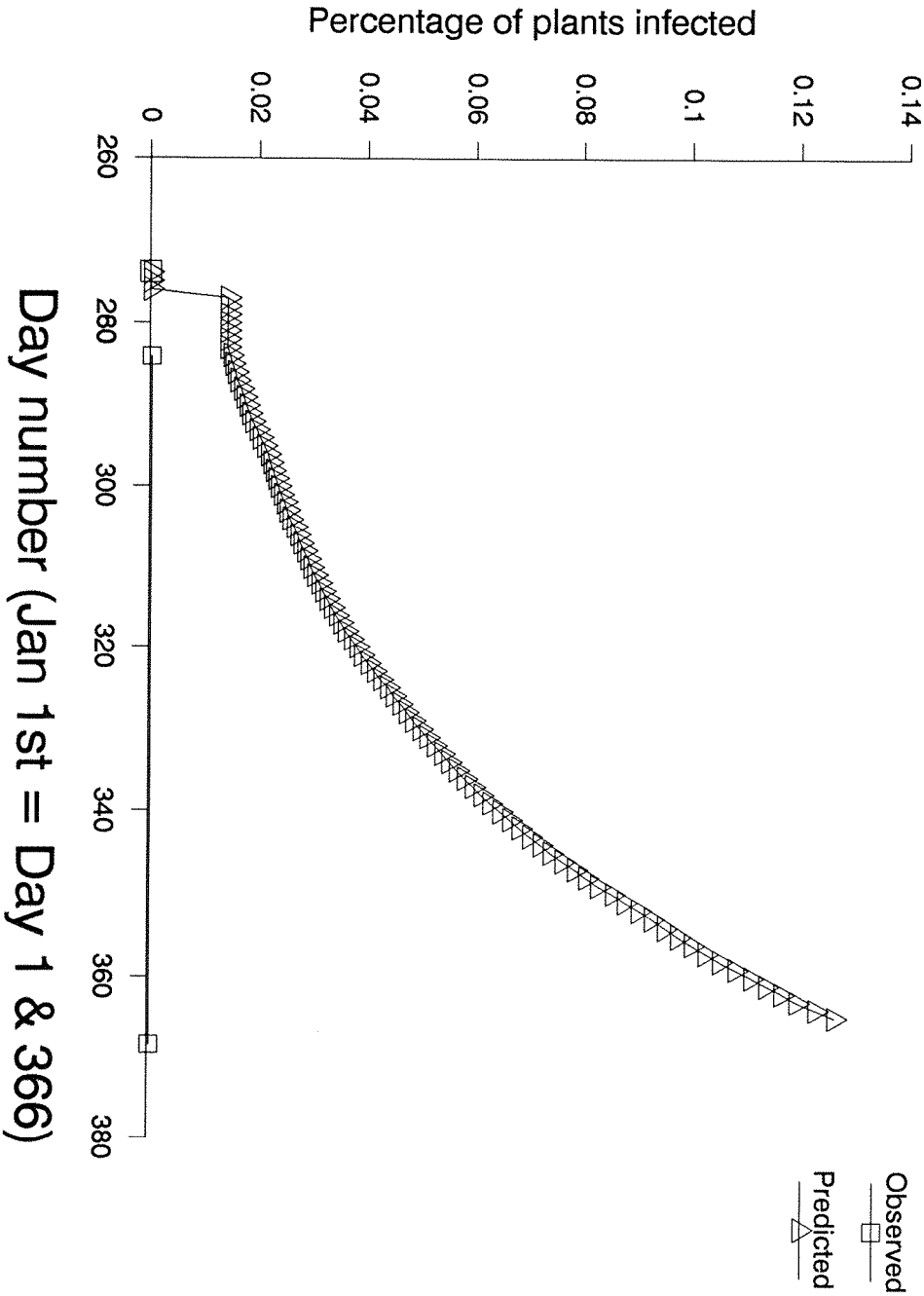
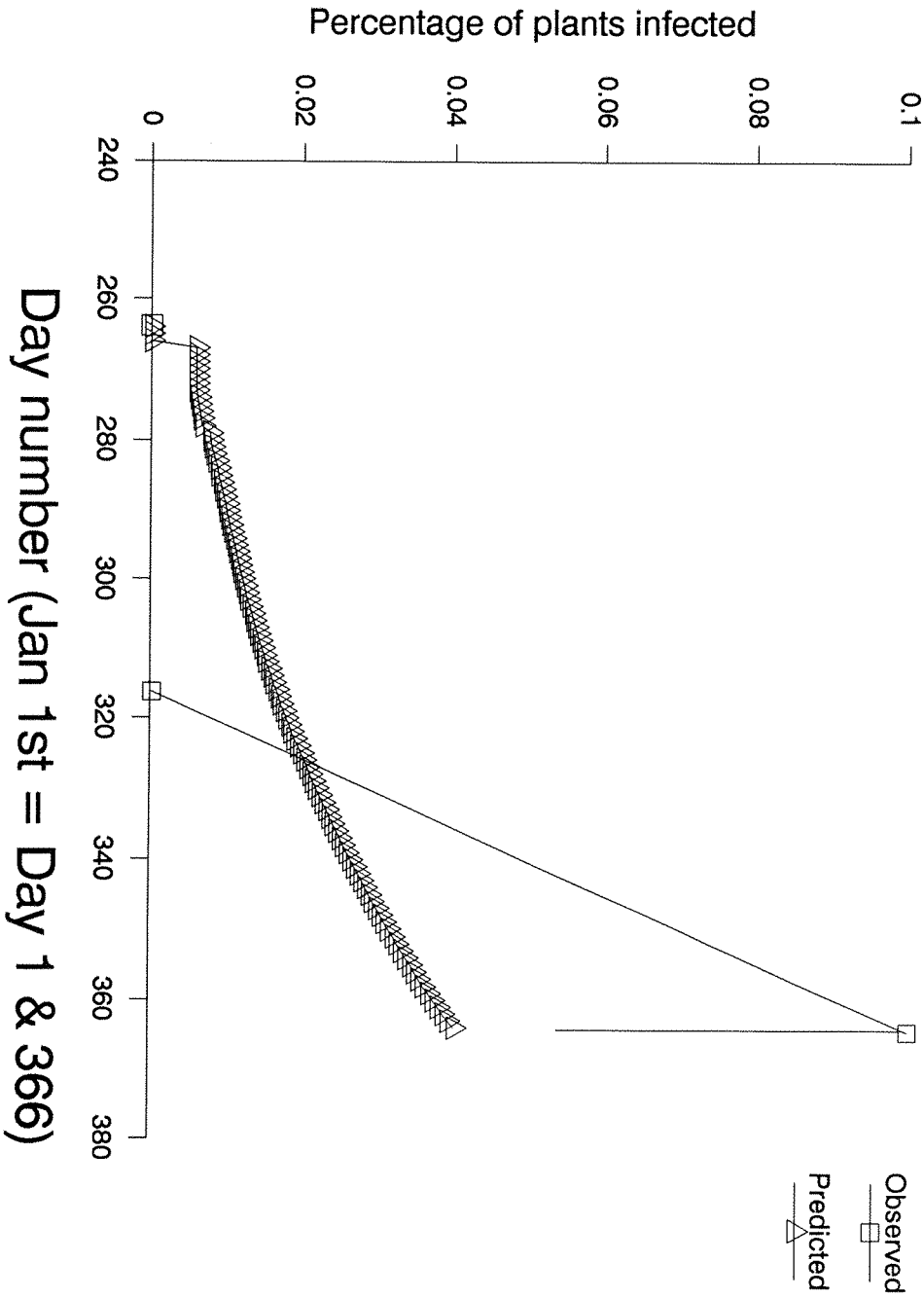


Figure 59. Observed and predicted BYDV incidence in 1987/8, sowing date 10 Sept.



neighbouring plants and on aphid dispersal could have increased the amount of virus spread. Roitberg, Myers & Frazer (1979) found that the prescence of coccinellids increased the dispersal of pea aphids, *A. pisum*, and hypothesised that this could, inadvertently, increase virus spread as dislodged aphids colonise new healthy host plants.

10. Sensitivity Analysis of Virus Sub-model

10.1 Introduction

A sensitivity analysis was carried out to determine the relative influence of the processes considered in the sub-model. Data from the first and second sowings in 1985/6 were used in the analysis as insufficient virus was present in the other sowings and years to produce interpretable results.

Small changes were made to the latent period of the virus in the plant (±2 days), initial crop growth stage (±2), rainfall (±20%) and aphid density (±20%).

10.2 Results

10.2.1 Latent Period

Changing the length of the latent period of the virus in a host plant had a proportional effect on the percentage of plants infected. Increasing the period by 2 days led to a 32% decrease in virus incidence in the first sowing (Figure 60) and a 22% decrease in the second sowing (Figure 61), while a decrease of 2 days increased the percentage infection by 21% and 16% in the first and second sowings respectively. Increasing the latent period decreased the virus spread by delaying the availability of virus to feeding aphids, reducing the numbers of vectors present and resulting in a lower number of infected plants.

10.2.2 Initial Crop Growth Stage

Alterations to the initial crop growth stage had a super-proportional effect on virus spread. Increasing the growth stage by 2 decreased virus incidence 3% in the first sowing (Figure 62) and by 4% in the second sowing (Figure 63), while decreasing it by 2 increased virus spread by 4% and 7% in the first and second sowings respectively. Increasing the initial crop growth stage broughtforward the onset of tillering which decreases the number of aphids forced to move by rain and infect new hosts earlier in the season. Hence virus spread was reduced.

10.2.3 Rainfall

Changing the amount of rain by 20% had very little influence on virus incidence, altering it by only 2% in both the first (Figure 64) and second (Figure 65) sowings. Therefore, rainfall need not be

Figure 60. The effect of changes in latent period on virus incidence in 1985/6, sowing date 13 Sept.

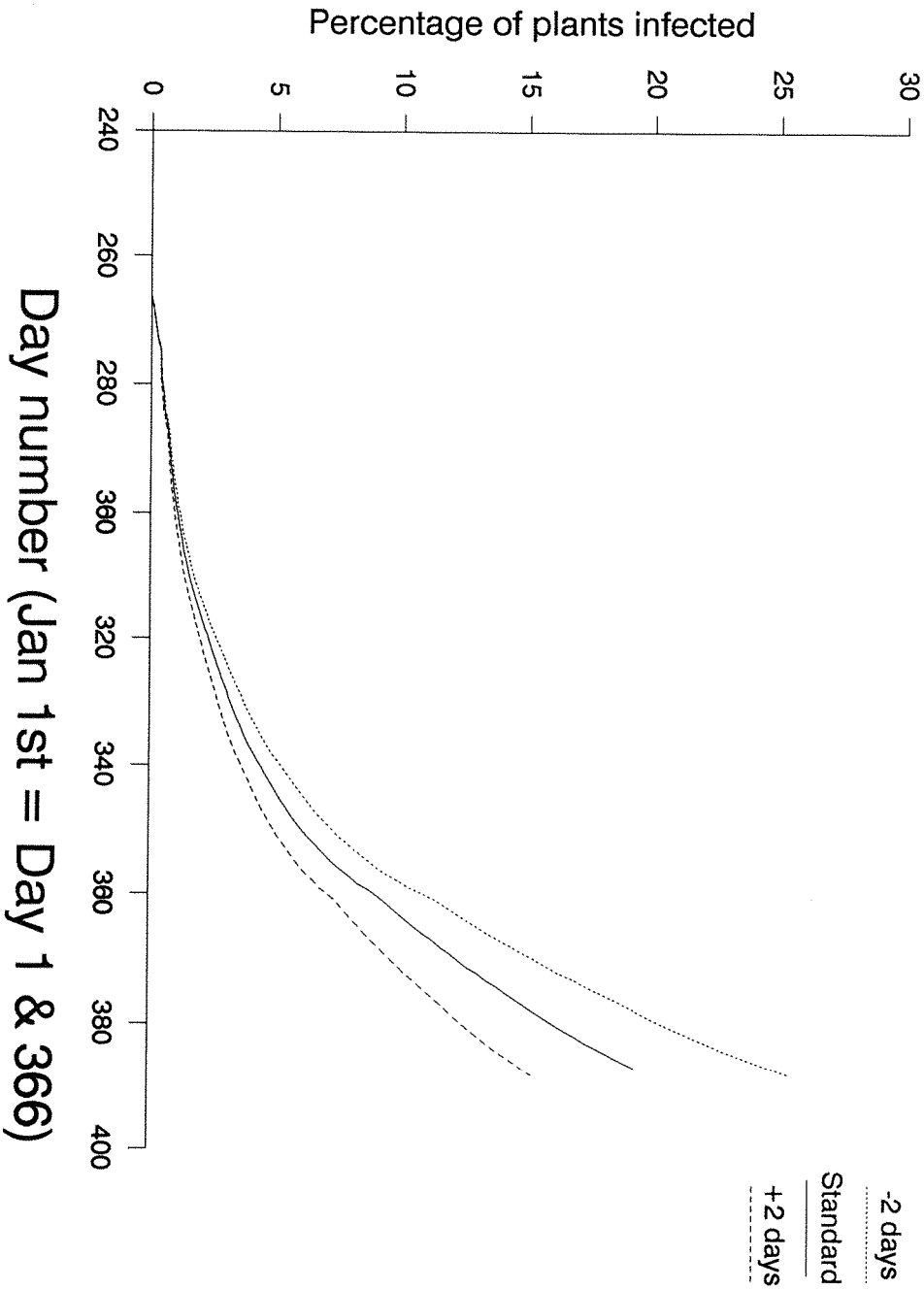


Figure 61. The effect of changes in latent period on virus incidence in 1985/6, sowing date 23 Sept.

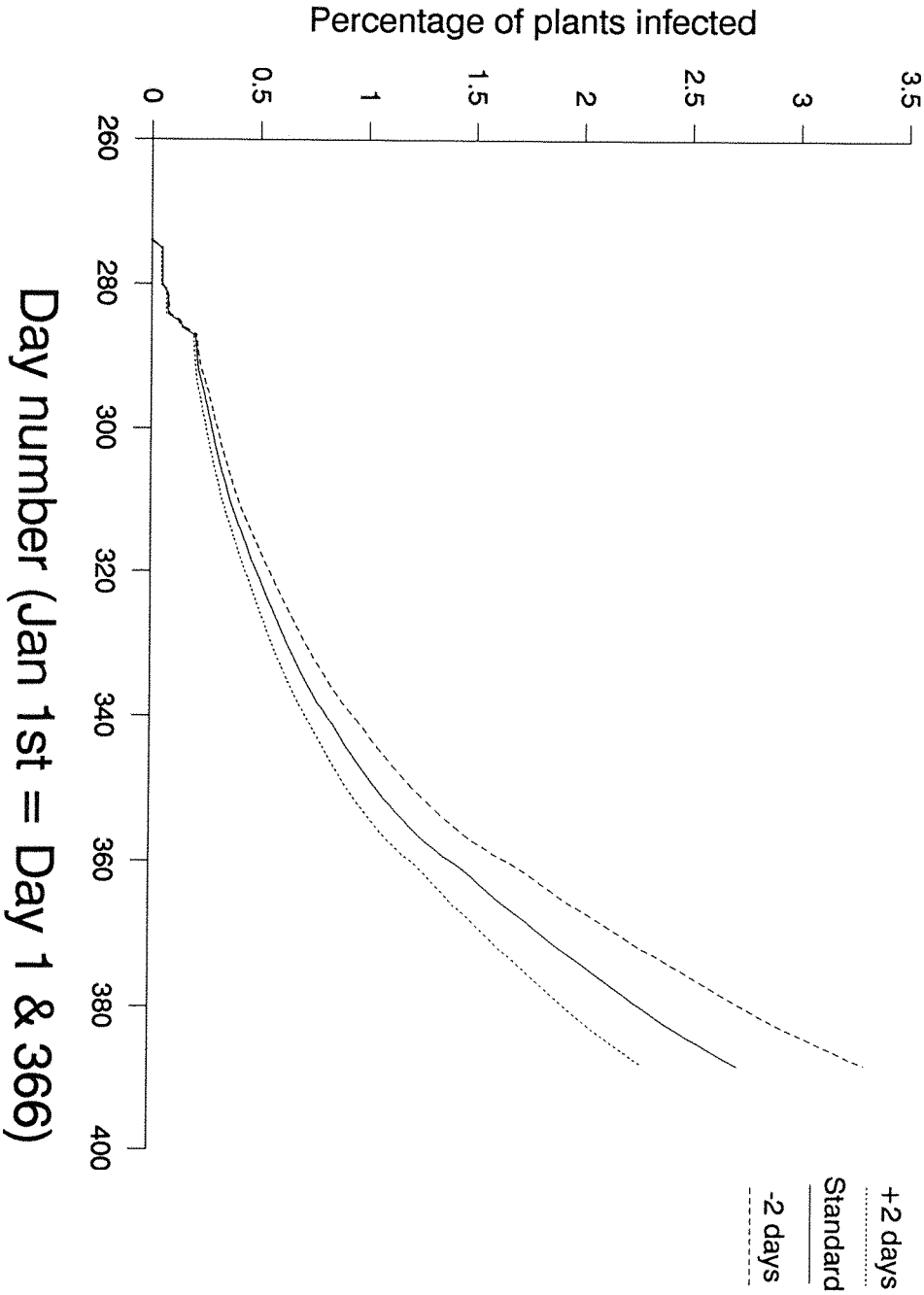


Figure 62. The effect of changes in growth stage on virus incidence in 1985/6, sowing date 13 Sept.

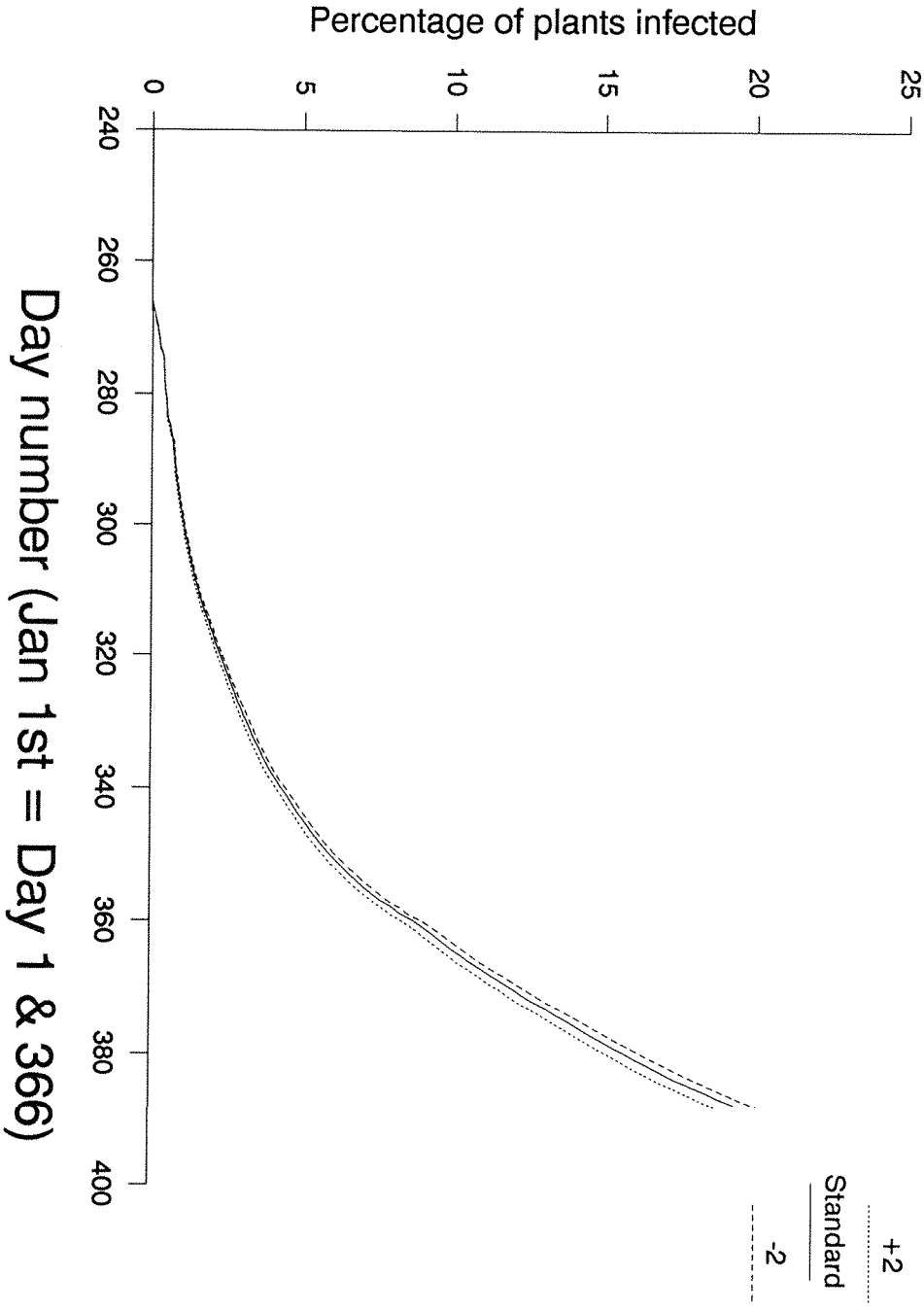


Figure 63. The effect of changes in growth stage on virus incidence in 1985/6, sowing date 23 Sept.

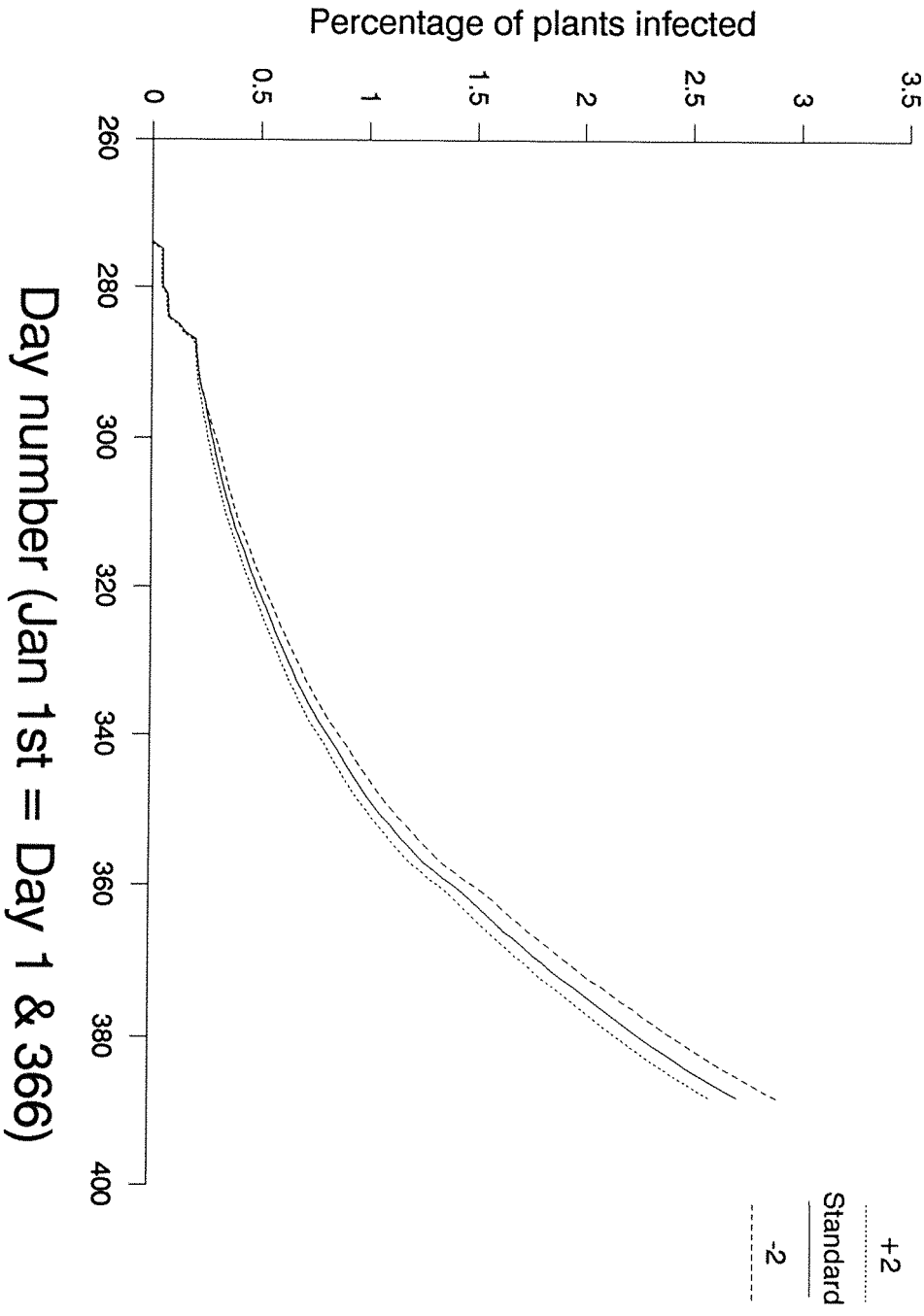


Figure 64. The effect of changes in rainfall on virus incidence in 1985/6, sowing date 13 Sept.

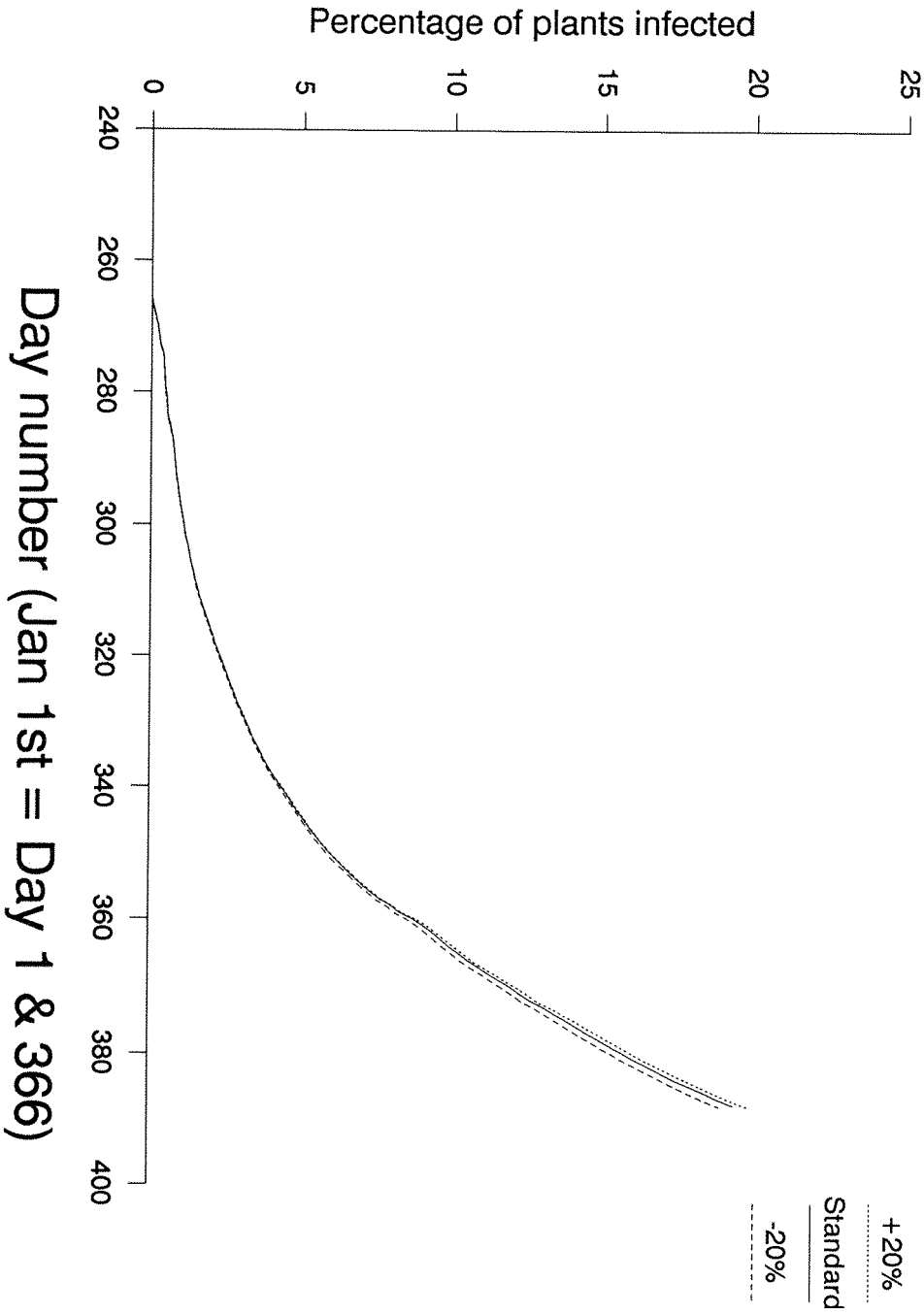
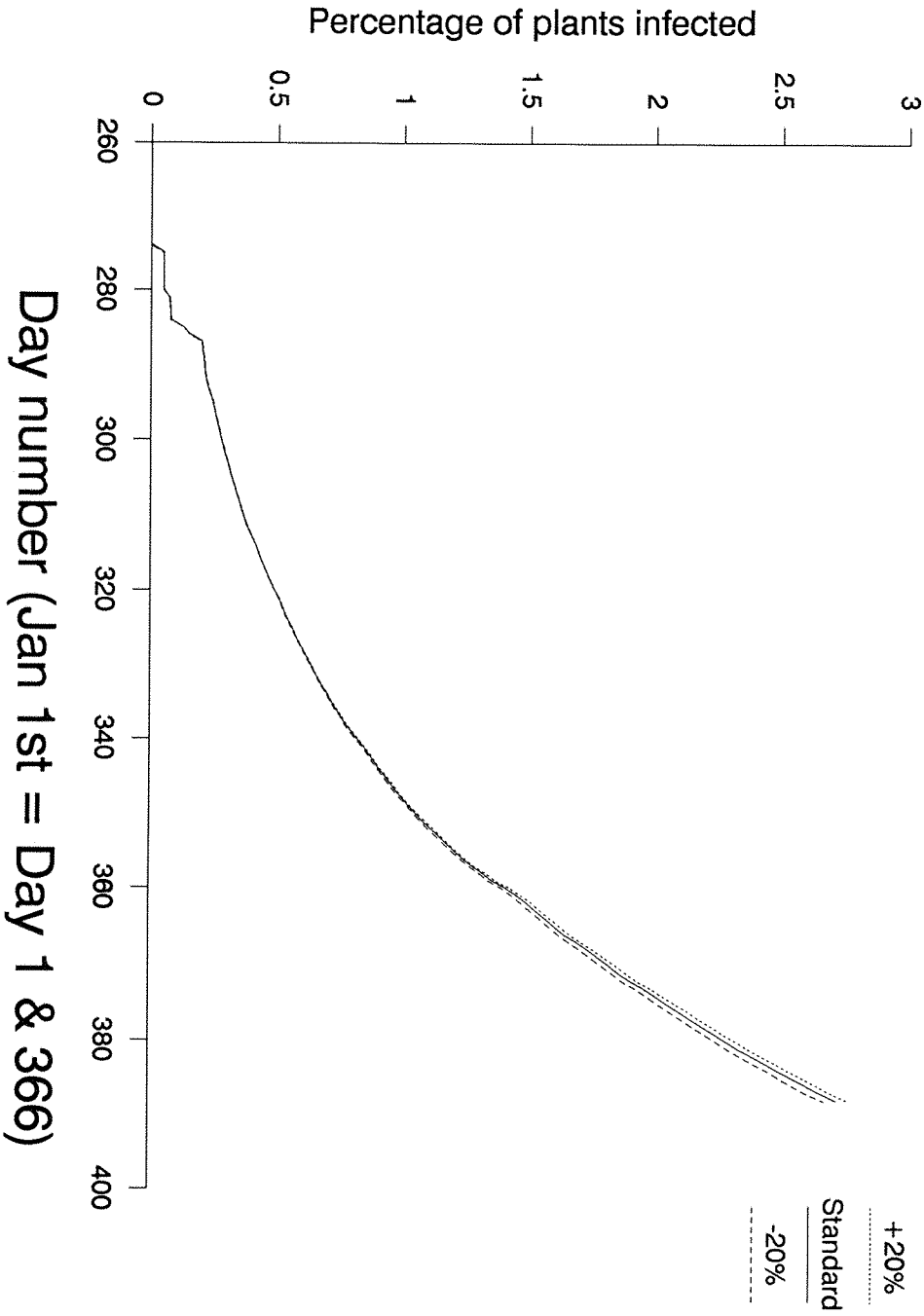


Figure 65. The effect of changes in rainfall on virus incidence in 1985/6, sowing date 23 Sept.



determined as accurately as the virus latent period, which has a super-proportional effect on virus spread, although rainfall is an important component.

10.2.4 Aphid Density

Altering the density of aphids on the crop had a sub-proportional effect on virus spread, changing it by 20% changed virus incidence by 16% on the first sowing (Figure 66) and by 8% on the second (Figure 67). It appeared that as total aphid density increased, and so the number of vectors present, more plants were infected.

10.3 Discussion

Of the processes examined in the sensitivity analysis, changes to the latent period of the virus in the host plant had the greatest effect on virus spread. Increasing the period by 2 days decreased virus incidence by 32% in the first sown plots in 1985/6. Frazer (1977) found that by increasing the latent period, in a sensitivity analysis of his model, of alfalfa mosaic virus (AMV) lowered the spread of the disease. It appears that the latent period is important in the epidemiology of a virus and should be measured accurately under a range of natural conditions.

The number of aphids on the crop needs to be known accurately as no virus submodel, however sophisticated, would predict virus incidence accurately if the numbers of vectors, used as input into the virus submodel, was predicted inaccurately. Frazer (1977) found that if aphid number were reduced, by increased predation in his model, then less AMV was spread. Similar results were observed for two rice virus diseases. Isimototo & Yamada (1986) found that decreasing the numbers of the small brown plant hopper, *L. striatellus*, reduced the infection rate of rice stripe virus. Sasabata et al. (1973) found that insecticide applications that reduced the number of spiders in a rice paddy allowed green rice leafhopper, *N. cincticeps*, populations to increase and more rice dwarf virus to be spread.

Initial crop growth stage and rainfall had less dramatic effects on virus than latent period and aphid densities. Increasing the initial crop growth stage decreased the percentage of plants infected with virus as the change was equivalent to bringing forward the timing of tillering of the plants and therefore the number of aphids knocked off by rain was reduced earlier in the season.

Figure 66. The effect of changes in vector density on virus incidence in 1985/6, sowing date 13 Sept.

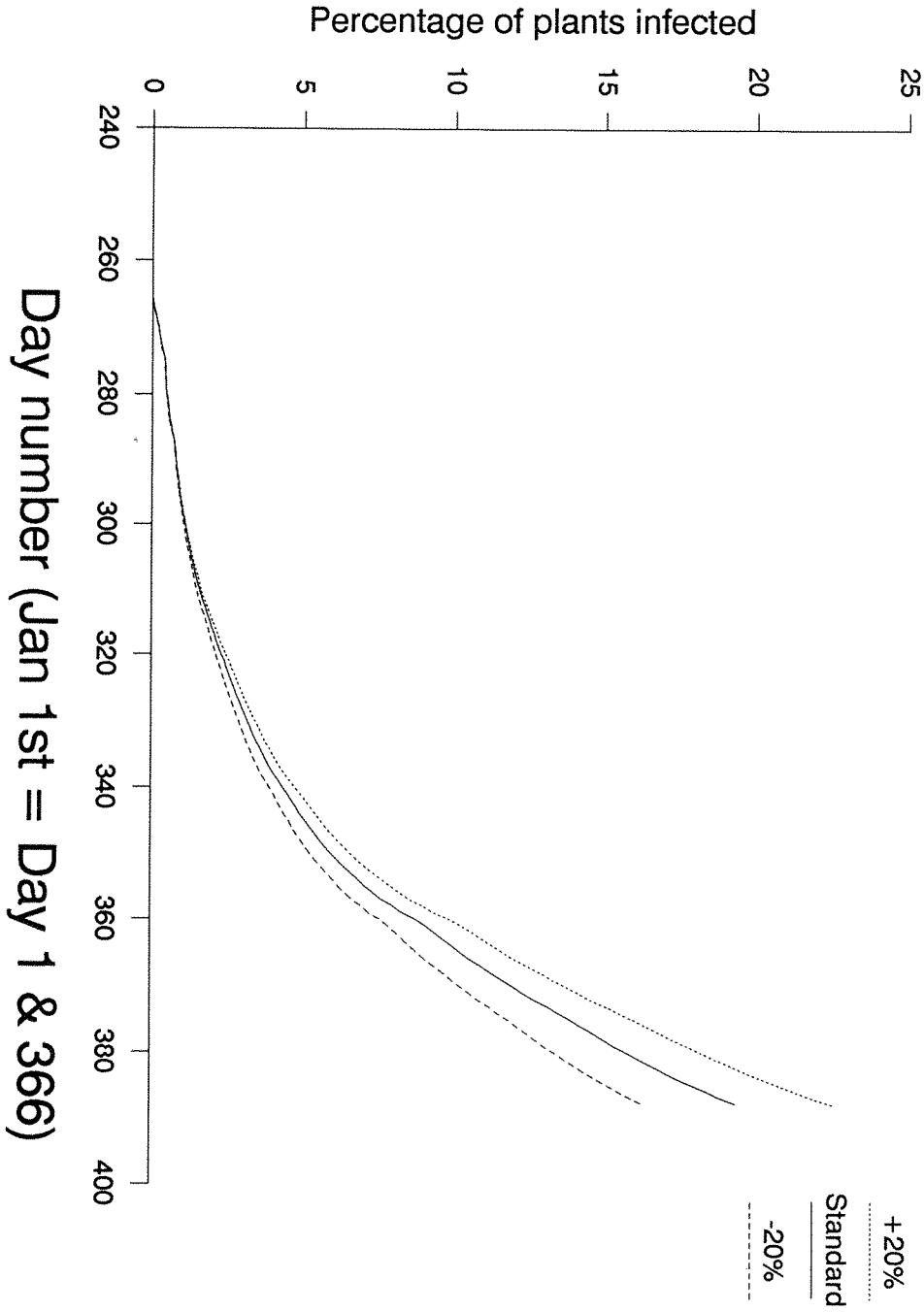
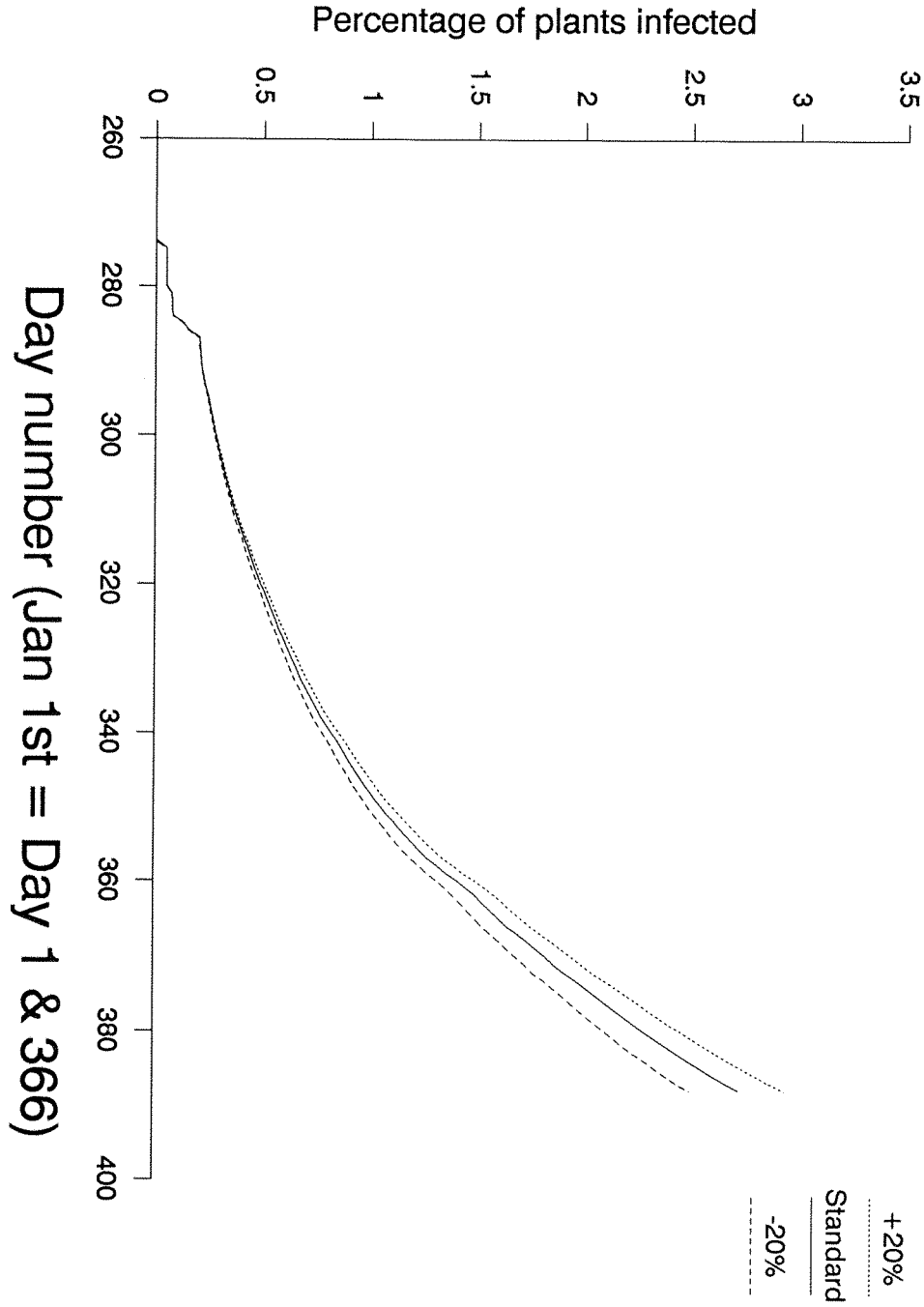


Figure 67. The effect of changes in vector density on virus incidence in 1985/6, sowing date 23 Sept.



Reducing the amount of rain that fell meant that less aphids were dislodeged to move and infect new hosts.

11. DISCUSSION

This research has combined field and laboratory studies with computer models to investigate the population dynamics of *R. padi* and spread of BYDV. The field studies involved monitoring the population development of *R. padi* (the main vector of BYDV in the autumn) and the spread of the disease in experimental plots which were sown on five dates in each of three years. Most *R. padi* were found on the earliest sown plots and numbers declined with later sowing dates. Most aphids were found in 1985/6, with peak densities about five times those found in 1986/7 and about fifty-two times those found in 1987/8. This was because of the greater number of migrant aphids flying in the autumn of 1985 (Figure 2) which had higher reproductive and survival rates in the warmer, drier autumn (Figure 3) allowing the aphid population to increase more rapidly than in either 1986 or 1987. Virus incidence was also highest in 1985/6 and in the earliest sown plots in all of the years. However, the relationship between virus incidence (y) and crop sowing date (x) in this year was not significant ($y=262.0-0.95*x$, $r=0.94$, d.f.=1, $p>0.05$). There was a significant relationship between virus incidence (y) and peak aphid density (z)($y=0.428+13.384*z$, $r=0.99$, d.f.=6, $p<0.001$), implying that conditions favouring aphid population growth would also facilitate virus spread.

Laboratory studies concentrated on the bionomics and movement of *R. padi*. Experiments were carried out to investigate the effect of temperature on the development, survival and reproduction of *R. padi*. These produced data similar to those found by Dean (1974), although the different methods used to rear aphids in both studies are known to affect aphid physiology (Adams, 1946; MacKinnon, 1961). Other authors have described the relationship between temperature and development and reproductive rates of aphids linearly (Hughes, 1963; Hughes & Gilbert, 1971) although it has been shown that the relationship is curveilinear close to both the upper and lower thresholds (Stinner, Gutierrez & Butler, 1974). Therefore, it would have been more realistic to have used curvilinear expressions to describe the relationship between development and temperature.

A series of experiments were carried out to study the movement of *R. padi*. A number of techniques have been used to tag aphids to follow their movement, including radioactive labels (Petterson, 1968) and paint (Muir, unpublished results). Both of these methods were rejected in favour of using virus spread as an indirect marker of aphid dispersal. Therefore, viruliferous aphids were allowed to move freely in arenas of healthy plants and the number of plants subsequently infected was related to numbers of vectors and time. The

measure of dispersal obtained was used with the model describing the population dynamics of *R. padi* and the predictions were found to underestimate the amount of virus spread recorded in the field. Therefore, other factors, thought to stimulate aphid movement, were investigated. Itô (1960) showed that apterous *R. padi* emigrated from plants, if colonies reached sufficiently high numbers, to overcome the reduction in nutrient availability caused by overcrowding. Even though the aphids faced additional mortalities by moving between plants these may be outweighed by the gain of a high nutrient source. However, the aphid densities which stimulated apterous emigration were completely unrealistic in comparison with field densities of *R. padi* in the autumn. Thus, it appears that apterous emigration, stimulated by overcrowding, is more likely to occur in the summer, when aphid densities can reach several hundred per plant (Cannon, 1984), than winter. Therefore, aphid dispersal caused by overcrowding was not considered in the model. The amount of rainfall was found to influence aphid movement at different crop growth stages. Aphids, that were feeding on plants at two growth stages, were exposed to two intensities and amounts of rain and it was found that more aphids were dislodged from seedlings under heavy rainfall than from more mature plants under lighter rain. The rain knocked the aphids off the plants which provided more shelter at later growth stages.

Data from published sources and the experiments were used to construct the model. The aphid sub-system followed a design previously adopted by Carter et al. (1982). The sub-system is deterministic with a step length of one day. A stochastic approach would have been biologically more realistic (Carter, 1986) but the advantages gained by doing so would not make up the extra demands made on computer resources (Gilbert & Hughes, 1971). A number of procedures are available to optimise the length of the time step (Rabbinge, 1976) but it was felt that it would be more practical, in computing terms, to set it to one day. The virus sub-system was also deterministic and had a step length of one day. The rate of virus spread was predicted from the number of aphids in the crop, predicted by the aphid sub-system, and how much they moved. Extensive verification was carried to ensure no errors occurred in the computer code and that the program functioned as it was intended (Jeffers, 1978).

The predictions from the model of the population development of *R. padi* were similar to those observed in the field (Chapter 6). However, there were differences but these could have been the result of factors not considered currently in the model. For example, because of the low aphid densities found in the field, the maximum being 1.5 aphids per plant in the three years (Chapter 6), a single

aphid that was accidentally missed during sampling would have had a large bias to the results. This effect becomes more serious the lower the densities.

A sensitivity analysis was carried out to estimate the importance of the processes considered in the model and the accuracy to which they needed to be known. Temperature, nymphal instar duration, survival and reproductive rate were found to have a super-proportional effect on aphid density. These results are similar to those found by Carter et al. (1982). Temperature was recorded with a standard 1m Stevenson screen situated approximately 300m from the experiments. Therefore, it would reasonable to assume that the temperatures recorded would not necessarily be the same as those experienced by the aphids feeding on the crop (Baker, 1980) although the extent of the difference is not known. An improvement would be to use portable meteorological recording equipment and to place the sensors close to the feeding aphids.

The duration of the nymphal instar age-classes used in the model was determined in the laboratory under constant temperatures. Messenger (1964), working with the spotted alfalfa aphid, *Therioaphis maculata*, and Siddiqui, Barlow & Randolph (1973), working with the pea aphid, *Acrythrosiphum pisum*, found that the aphid development rates were higher under low fluctuating temperatures, like those likely to be experienced in the field, than under constant low temperatures. Therefore development rates should be determined under fluctuating temperatures although it would be more difficult and costly to maintain these conditions.

Altering the survival rate had a dramatic effect on aphid densities. Survival was quantified by relating the difference between a predicted and observed aphid density between two successive field samples to the temperature accumulated, below a threshold, over the sample interval. The predicted density on the second sample date was calculated by using a logistic relationship for growth of the population from the first sample. This assumption was based on the population dynamics of cereal aphids during the summer in S. E. England (Cannon, 1984; Carter, 1978; Dewar & Carter, 1984). Although studies have related aphid survival to low temperature, both in the laboratory (Williams, 1980; Bale et al., 1988) and in the field (Williams, 1980), the data published were not in a suitable format to use in the model. Therefore, further research, with much shorter sampling intervals, is required.

The reproductive rate used in the model was determined under constant temperatures using leaf discs as a source of nutrient (Dean,

1974). However, Messenger (1964) found that fluctuating temperatures affected the fecundity of *T. maculata* and this should be considered in determining aphid reproductive rates. Also Leather (1982) and Leather & Dixon (1981) found that the reproductive rate of apterous *R. padi* varied with the growth stage of the host plant. This effect was not considered in the model as the growth stages that influenced the reproductive rate were not those found in the autumn and winter at Rothamsted. Leather (1982) and Leather & Dixon (1981) used plants of greatly differing growth stage and it may be possible that small differences in physiological age of the host plant may have significant effects on aphid fecundity. Therefore further research is required to investigate this.

Adult longevity and immigration had a proportional effect on aphid densities and so appeared less important. However, both should be measured carefully, especially immigration. It is the number of aphids landing on the crop that initiates population development. If deposition is miscalculated then the size of the population of *R. padi* will also be inaccurate, irrespective of the accuracy of the other processes in the model. Immigration was calculated by assuming that the aphids had a density-height gradient of -1.0 and a flight time of 2h. These were the average values for aphids in the summer in S. E. England (Taylor & Palmer, 1972), no data were available for aphids in the autumn and winter. Data to verify these assumptions could be obtained from repeating the experiment of Taylor & Palmer (1972) in the autumn and winter.

The predictions from the virus sub-system were similar to those found in the field in 1985/6 but not those found in 1986/7 and 1987/8. A number of possible reasons for the differences were suggested. The first was that reservoirs of aphids and virus were present in the experimental area prior to the emergence of the crop. The volunteer plants had grown from seed shed from the previous cereal crop and could have been infected by immigrant viruliferous alate aphids. Aphid colonies accumulating on these plants would have acquired the virus. These reservoirs would then have increased the inoculum pressure on the crop and hence more virus spread may have occurred than expected from a clean seed bed. There was no data available to describe the relationship between aphid and virus reservoirs and virus incidence of a crop and so it was not possible to construct a computer procedure simulating this. Future research is necessary that includes sampling volunteers for aphids and testing them for virus.

Predators increase the between-plant movement of aphids by disturbing individuals feeding in a colony forcing them to fall from

their original host plant to find another (Roitberg et al., 1979). This would increase virus spread if the aphids that were disturbed were viruliferous and found new, healthy hosts on which they began to feed. Frazer (1977) that, at high aphid densities, increased coccinellid behaviour disturbed and dislodged more pea aphids than they eat and, therefore, more AMV spread occured. No data were recorded, in either of the three years, of natural enemy numbers in this study. However, casual observations, made while sampling for aphids, suggested that predators were more numerous in 1985 than in either 1986 or 1987. Further field studies are required not only on the effect of natural enemies upon virus spread but also upon the effect on the virus vectors.

It could also be possible that as a crop matures and plants overlap with neighbouring plants the 'leaf-bridges' formed would provide aphids with a more convenient route to move between plants than walking across the soil. Hence more virus spread could occur. There is no evidence to confirm this. Proving this experimentally would be complicated because as the crop ripens it provides more shelter to aphids from rainfall and so less involuntary movement would occur (Chapter 9) confounding the measurement of movement across leaf-bridges.

The differences between predicted and observed aphid densities and virus incidence in this study do not detract from the usefulness of employing a system analysis approach to study virus epidemiology. These steps involved in this study have exposed areas in our knowledge of BYDV that were weak. The study began with the definition of which processes in the system were thought to be important. Published data were used to quantify these processes and it soon became apparent that no data were available in some areas. Therefore experiments were carried out to fill these gaps and verify the existing data. The predictions from the model were compared with results from field trials. The differences between the predicted and observed results questioned the philosophy behind the construction of the model and the data used to develop and validate it. Hence, further experiments have been stimulated to provide data to improve the model.

One of the major strengths of simulation modelling is that it highlights areas of a system on which further research is required. Using the model simulating the epidemiology of BYDV has suggested that more work is needed on the bionomics of *R. padi*, especially nymphal instar age-class duration, low temperature survival and fecundity, and its dispersal characteristics, particularly the influence of crop growth stage, natural enemies and environmental

conditions. If research on these topics is pursued the data provided which will improve the accuracy of the model's predictions and eventually it might be possible that the model could be used in an advisory capacity, rationalising spray applications aimed at controlling BYDV, as well as being used as an educational and research tool.

REFERENCES

A'BROOK J. 1968. The effect of plant spacing on the numbers of aphids trapped over the groundnut crop. Annals of Applied Biology. 61. 289-294.

A'BROOK J. 1981. Some observations in west Wales on the relationships between numbers of alate aphids and weather. Annals of Applied Biology. 97. 11-15.

ADAMS J. B. 1967. A physiologic difference in aphids (Homoptera) raised on excised leaves and on intact plants. Canadian Journal of Zoology. 45. 588-590.

AJAYI O. 1981. Interactions between barley yellow dwarf virus aphids and *Cladosporium* on cereals Ph.D. Thesis University of London

BAGNAL R.H. & BRADLEY R.H.E. 1958. Resistance to virus Y_o in potato. Phytopathology. 48. 121-125.

BAKER C. R. B. 1980. Some problems in using meteorological data to forecast the timing of insect life cycles. EPPO Bulletin. 10. 83-91.

BALE J. S. HARRINGTON R. & CLOUGH M. S. 1988. Low temperature mortality of the peach-potato aphid *Myzus persicae*. Ecological Entomology. 13. 121-129.

BARLOW N. & DIXON A. F. G. 1980. Simulation of lime aphid population dynamics. Pudoc. 171pp.

BENNETT C.W. 1969. Seed transmission of plant viruses. Advances in Virus Research. 14. 221-261.

BROADBENT L. CORNFORD C.E. HULL R. & TINSLEY T.W. 1949. Overwintering of aphids especially *Myzus persicae* (Sulzer) in root clamps. Annals of Applied Biology. 36. 513-524.

BROEK van der L. J. & GILL C. C. 1980. The median latent periods for three isolates of barley yellow dwarf virus in aphid vectors. Phytopathology. 70. 644-646.

CANNON R. J. C. 1984. The development rate of *Metopolophium dirhodum* (Walker)(Hemiptera:Aphididae) on winter wheat. Bulletin of Entomological Research. 74. 33-46.

CARTER N. 1978. Simulation study of the English grain aphid. Ph.D. Thesis, University of East Anglia.

CARTER N. 1986. Simulation modelling. In 'Plant Virus Epidemics' eds. G. D. McLean R. G. Garrett & W. G. Ruesink. Academic Press. 193-215.

CARTER N. DIXON A. F. G. & RABBINGE R. 1982. Cereal aphid populations : biology, simulation and prediction. Pudoc. 97pp.

CARTER N. MCLEAN I. F. G. WATT A. D. DIXON A. F. G. 1980. Cereal aphids : a case study and review. Applied Biology. 5. 271-348.

CHAMBERS R.J. & ADAMS T.H.L. 1986. Quantification of the impact of hoverflies (Diptera:Syrphidae) on cereal aphids in winter wheat : an analysis of field populations. Journal of Applied Ecology. 23. 895-904.

CHAMBERS R.J. SUNDERLAND K.D. STACEY D.L. & WYATT I.J. 1985. Control of cereal aphids in winter wheat by natural enemies : aphid-specific

predators parasitoids and parthengenic fungi. Annals of Applied Biology. 108. 219-231.

CHAMBERS R.J. SUNDERLAND K.D. WYATT I.J. & VICKERMAN G.P. 1983. The effects of predator exclusion and caging on cereal aphids in winter wheat. Journal of Applied Ecology. 20. 209-224.

CHUA T. H. 1978. A model of an aphid-parasite-hyperparasite system with reference to timing of attack. Malaysian Agricultural Journal. 375-386.

CONWAY G. R. 1978. Pest and Pathogen Control. Wiley. 488pp.

COON B. F. 1959. Grass hosts of cereal aphids. Journal of Economic Entomology. 52. 994-996.

DEAN G. J. W. 1973. Bionomics of aphids reared on cereals and some Graminae. Annals of Applied Biology. 73. 127-135.

DEAN G. J. W. 1974. Effect of temperature on the cereal aphids *Metopolpphium dirhodum Rhopalosiphum padi* and *Macrosiphum avenae*. Bulletin of Entomological Research. 63. 401-409.

DEAN G. J. W. 1974. The overwintering and abundance of cereal aphids. Annals of Applied Biology. 76. 1-7.

DEAN G. J. W. & LUURING B. B. 1970. Distribution of aphids in cereal crops. Annals of Applied Biology. 80. 130-132.

DEAN G. J. W. & WILDING N. 1971. Entomophthora infecting the cereal aphids *Metopolophium dirhodum* and *Sitobion avenae*. Journal of Invertebrate Pathology. 18. 169-176.

DEAN G. J. W. & WILDING N. 1973. Infection of cereal aphids by the fungus Entomophthora. Annals of Applied Biology. 74. 133-138.

DEWAR A. M. 1977. Morph determination and host alternation in the apple-grass aphid *Rhopalosipuhum insertum* (Wlk.). Ph.D. Thesis University of Glasgow

DEWAR A. M. & CARTER N. 1984. Decision trees to assess the risk of cereal aphid (Hemiptera:Aphididae) outbreaks in summer in England. Bulletin of Entomological Research. 74. 387-398.

DIXON A. F. G. 1971. The life-cycle and host prefences of the bird cherry-oat aphid *Rhopalosiphum padi* and their bearing on the theories of host alternation in aphids. Annals of Applied Biology. 68. 135-14-7.

DIXON A. F. G. & DEWAR A. M. 1974. The time of determination of gynoparae and males in the bird cherry-oat aphid *Rhopalosiphum padi*. Annals of Applied Biology. 78. 1-6.

DIXON A. F. G. & GLEN D. M. 1971. Morph determination in the bird cherry-oat aphid *Rhopalosiphum padi*. Annals of Applied Biology. 68. 11-21.

DOODSON J. K. & SAUNDERS P. J. W. 1969. Observations on the effects of some systemic chemicals applied to cereals in trials at the NIAB. Proceedings of 5th British Insecticide and Fungicide Conference. 1. 1-7.

DRY W. W. & TAYLOR L. R. 1970. Light and temperature thresholds for take-off by aphids. Journal of Animal Ecology 39. 493-504.

ELAMIN E. M. 1975. Studies on the ecology of aphids on spring cereals and maize. Ph.D. Thesis, University of London.

EVENHUIS H.H. 1968. The natural control of the apple-grass aphid *Rhopalosiphum insertum* with remarks on the control of apple aphids in the Netherlands in general. Netherlands Journal of Plant Pathology. 74. 106-117.

FITT B.D.L. GREGORY P.H. TODD A.D. MCCARTNEY H.A. & MACDONALD O.C. 1987. Spore dispersal and plant disease gradients : a comparison between empiracle models. Journal of Phytopathology 118. 227-242.

FOXE M.J. & ROCHOW W.F. 1975. Importance of virus source leaves in vector specificity of barley yellow dwarf virus. Phytopathology. 65. 1124-1129.

FRAZER B.D. 1977. Plant virus epidemiology and computer simulation of aphid populations. In 'Aphids as Virus Vectors'. eds. K.F. Harris and K. Maramorosch. Academic Press. 413-431.

FRAZER B.D. & GILBERT N. 1976. Coccinellids and aphids : a quantitative study of the impact of adult ladybirds (Coloeptera:Coccinellidae) preying on filed populations of pea aphids (Homoptera:Aphididae). Journal of the Entomological Society of British Columbia. 73. 33-56.

GAIR R. 1953. Observations on grass aphids in Derbyshire 1950-52. Plant Pathology. 2. 117-121.

GILBERT N. & GUTIERREZ A.P. 1973. A plant-aphid-parasite relationship. Journal of Animal Ecology. 42. 323-340.

GILBERT N. & HUGHES R.D. 1971. A model of an aphid population - three adventures. Journal of Animal Ecology. 40. 525-534.

GILDOW F.E. 1980. Increased production of alate aphids by aphids reared on oats infected with barley yellow dwarf virus. Annals of the Entomological Society of America. 73. 343-347.

GILL C.C. 1969. Cyclical transmissibility of barley yellow dwarf virus from oats with increaesing age of infection. Phytopathology. 59. 23-28.

GILL C.C. 1970. Aphid nymphs transmit an isolate of barley yellow dwarf virus more effectively than adults. Phytopathology. 60. 1747-1752.

GUTIERREZ A.P. HAVENSTEIN D.E. NIX H.A. & MOORE P.A. 1974. The ecology of *Aphis craccivora* Koch and subterranean clover stunt virus in southeast Australia. II. A model of cowpea aphid populations in temperate pastures. Journal of Applied Biology. 11. 1-20.

HANIOTAKIS G.E. & LANGE W.H. 1974. Beet yellows virus resistance in sugar beets : mechanisms of resistance. Journal of Economic Entomology. 67. 25-28.

HARRISON B.D. FINCH J.T. GIBBS A.J. HOLLINGS M. SHEPERD R.J. VALENTA V. & WETTER C. 1971. Sixteen groups of plant viruses. Virology. 45. 356-363.

HEATHCOTE J.D. & BYFORD W.J. 1975. Surveys of sugar-beet seed crops mangold clamps and weeds in England for aphids and viruses 1963-1973. Journal of Agricultural Science Cambridge. 84. 87-95.

HODEK I. HAGEN K.S. & van EMDEN H.F. 1972. Methods of studying effectiveness of natural enemies. In "Aphid Technology". ed. H.F. van Emden. Academic Press. 147-188.

HODEK I. & HODKOVA M. 1988. Multiple role of temperature during insect diapause : a review. Entomologia Experimentalis et Applicata. 49. 153-165.

HUGHES R.D. 1963. Population dynamics of the cabbage aphid *Brevicoryne brassicae* (L.). Journal of Animal Ecology. 32. 393-424.

HUGHES R.D. & GILBERT N. 1968. A model of an aphid population - a general statement. Journal of Animal Ecology. 37. 553-563.

HUGHES R.D. & WOOLCOCK L.T. 1965. A modification of Johnson's method of rearing aphids for ecological studies. New Zealand Journal of Agricultural Research. 8. 728-736.

HULL R. 1969. Alfalfa mosaic virus. Advances in Virus Research. 15. 365-433.

ITO Y. 1960. Ecological studies on population increase and habitat segregation among barley aphids. Bulletin of the National Institute of Agricultural Science Hishigahara. 11. 45-127.

JEDLINSKI H. & BROWN C.M. 1965. Cross protection and mutual exclusion by three strains of barley yellow dwarf virus in *Avena sativa*(L.). Virology. 26. 613-621.

JEFFERS J.N.R. 1978. An Introduction to Systems Analysis. Edward Arnold. 198pp

JONES M.G. 1972. Cereal aphids their parasites and predators caught in cages over oat and winter wheat crops. Annals of Applied Biology. 72. 13-25.

KENDALL D.A. & SMITH B.D. 1981. The significance of aphid monitoring in improving barley yellow dwarf virus control. In 'Proceedings of the 1981 British Crop Protection Conference'. 399-403.

KIRITANI K. 1979. A systems approach to pest mamagement of the green rice leafhopper Pest Management. Proceeding of an International Conference. 229-252.

KISIMOTO R. & YAMADA Y. 1986. A planthopper-rice virus epidemiology model : rice stripe and small brown planthopper, *Laodelphax striatellus* Fallen. In "Plant Virus Epidemics" eds. G. D. McLean R. G. Garrett & W. G. Ruesink. Academic Press. 327-344.

KNIGHT J.D. BALE J.S. FRANKS F. MATHIAS S.F. & BAUST J.G. 1986. Insect cold hardiness : super-cooling points and pre-freeze mortality. Cryoletters. 7. 194-203.

KNUTSON K.W. & BISHOP G.W. 1964. Potato leafroll virus-effect of date of inoculation on percent infection and symptom expression. American Potato Journal. 41. 227-238.

LEATHER S.R 1982. Preliminary studies on the effect of host age and aphid generation on the reproduction and survival of the bird cherry-oat aphid *Rhopalosiphum padi*. Annals Agriculture Fennicae. 21. 13-19.

LEATHER S.R. 1980. Egg survival in the bird cherr-oat aphid *Rhopalosiphum padi*. Entomologia Experimentalis et Applicata. 27. 96-97.

LEATHER S.R. & DIXON A.F.G. 1981. The effect of cereal growth stage and feeding site on the reproductive activity of the bird cherry aphid *Rhopalosiphum padi*. Annals of Applied Biology. 97. 135-141.

MACKINNON J.P. 1961. Preference of aphids for excised leaves to whole plants. Canadian Journal of Zoology. 39. 445-447.

MARKKULA M. & LAUREMA S. 1964. Change in the concentration of free amino acids in plants induced by virus diseases and the reproduction of aphids. Annals Agriculture Fennicae. 3. 265-271.

MESSENGER P.S. 1964. The influence of rhythmically fluctuating temperatures on the devlopment and reproduction of the spotted alfalfa aphid *Therioaphis maculata*. Journal of Economic Entomology. 57. 71-76.

MILLER J.W. & COON B.F. 1964. The effect of barley yellow dwarf virus on the biology of its vector the English grain aphid *Macrosiphum avenae*. Journal of Economic Entomology. 57. 970-974.

MIYAI S. KIRITANI K. & NAKASUJI F. 1986. Models of epidemics of rice dwarf. In "Plant Virus Epidemics" eds. G. D. McLean R. G. Garrett & W. G. Ruesink. Academic Press. 429-482.

NACHMAN G. 1987. Systems analysis of acarine predator-prey interactions. I. A stochastic simulation model of spatial processes. Journal of Animal Ecology. 56. 247-266.

NAKASUJI F. KIRITANI K. 1972. Descriptive models for the system of the natural spread of infection of rice dwarf virus (RDV) by teh green rice leaf hopper *Nephotettix cincticeps* UHLER (Hemiptera:Deltocephalidae). Research on Popualtion Ecology. 14. 18-35.

NAKASUJI F. KIRITANI K. & TOMIDA E. 1975. A computer simulation of the epidemiology of the rice dwarf virus. Research on Population Ecology. 16. 245-251.

NAKASUJI F. MIYAI S. KAWAMOTO H. & KIRITANI K. 1985. Mathematical epidemiology of rice dwarf virus transmitted by green rice leaf hoppers : a differential equation model. Journal of Applied Ecology. 22. 839-847.

OSWALD J.W. & HOUSTON B.R. 1953. The yellow-dwarf virus disease of cereal crops. Phytopathology. 43. 128-136.

PERRY J.N. 1987. Host-parasitoid models of intermediate complexity. American Naturalist. 130. 955-957.

PETTERSSON J. 1968. Tagging aphids. Opuscula Entomologia. 33. 219-22-9.

PLUMB R.T LENNON E.A & GUTTERIDGE R.A 1988. Forecasting barley yellow dwarf virus by monitoring vector populations and infectivity. In 'Plant Virus Epidemics'. eds. G.D. McLean R.G. Garrett & W.G. Ruesink. Academic Press. 457pp.

PLUMB R.T. 1983. Barley yellow dwarf virus - a global problem. In 'Plant Virus Epidemiology'. eds. R.T. Plumb & J.M. Thresh. Blackwells. 185-198.

POTTS G. R. & VICKERMAN G.P. 1974. Studies on the cereal ecosystem. Advances in Ecological Research. 8. 107-197.

RABBINGE R. 1976. Biological control of the fruit-tree red spider mite. Pudoc. 243pp.

RABBINGE R. ANKERSMIT G.W. & PAK G.A. 1979. Epidemiology and simulation of population development of *Sitobion avenae* in winter wheat Netherlands. Journal of Plant Pathology. 85. 197-220.

RASMUSSON D.C. & SCHALLER C.W. 1959. The inheritance of resistance in barley to the yellow-dwarf virus. Agronomy Journal. 51. 661-664.

RAUTAPAA J. 1976. Population dynamics of cereal aphids and method of predicting population trends. Annals Agriculture Fennicae. 15. 272-293.

ROCHOW W.F. 1970. Barley yellow dwarf virus : phenotypic mixing and vector specificity. Science. 167. 875-878.

ROCHOW W.F. 1979. Field variants of barley yellow dwarf virus : detection and fluctuation during twenty years. Phytopathology. 69. 655-660.

ROCHOW W.F. 1982. Dependent transmission by aphids of barley yellow dwarf luteoviruses from mixed infections. Phytopathology. 72. 302-305.

ROCHOW W.F. & CARMICHAEL L.E. 1979. Specificity among barley yellow dwarf viruses in enzyme lineked immunosorbent assays. Virology. 95. 415-420.

ROCHOW W.F. & JEDLINSKI H. 1970. Variants of barley yellow dwarf virus collected in New York and Illinois. Phytopathology. 60. 1030-1-035.

ROGERSON J.P. 1948. The oat bird-cherry aphis, *Rhopalosiphum padi* L., and comparison with *R. crataegellum* Theo (Hemiptera,Aphididae). Bulletin of Entomological Research. 38. 157-176.

ROITBERG B.D. MYERS J.H. & FRAZER B.D. 1979. The influence of predators on the movement of apterous pea aphids between plants. Journal of Animal Ecology. 48. 111-122.

RUESINK W.G. & IRWIN M.E. 1986. Soybean mosaic virus epidemiology : a model and some implications. In 'Plant Virus Epidemics' eds. G.D. McLean R.G. Garrett & W.G. Ruesink. Academic Press. 295-314.

SASABA T. & KIRITANI K. 1975. A systems model and computer simulation of the green rice leafhopper populations in control programmes. Research on Population Ecology. 14. 231-244.

SASABA T. KIRITANI K. & URAPE T. 1973. A preliminary model to simulate the effect of insecticides on a spider-leafhopper system in the paddy field. Research on Population Ecology.. 15. 9-22.

SCHELLER H.V. & SHUKLE R.H. 1986. Feeding behaviour and transmission of barley yellow dwarf virus by *Sitobion avenae* on oats. Entomologia Experimentalis et Applicata. 40. 189-195.

SHANKS C.H. 1965. Seasonal populations of the strawberry aphid and transmission of strawberry viruses in the field in relation to virus control in western Washington. Journal of Economic Entomology. 58. 316-322.

SHANNON R.E. 1975. "Systems Simulation". Prentice-Hall 387pp

SIDDIQUI W.H. BARLOW C.A. & RANDOLPH P.A. 1973. Effects of some constant and alternating temperatures on population growth of the pea aphid *Acrytosiphon pisum* (Homoptera:Aphididae). Canadian Entomologist. 105. 145-156.

SIGVALD R. 1986. Forecasting the incidence of potato virus Y_o. In "Plant Virus Epidemics" eds. G.D. McLean R.G. Garrett & W.G. Ruesink. Academic Press. 419-442.

SOPP P. & CHIVERTON P. 1987. Autumn predation of cereal aphids by polyphagous predators in southern England : a first look using ELISA. Bulletin WPRS/SROP. 10 103-108.

STIMMANN M.W. & SWENSEN K.G. 1967. Aphid transmission of cucumber mosaic virus affected by temperature and age of infection in diseased plants. Phytopathology. 57. 1074-1076.

STINNER R.E. GUTIERREZ A.P. & BUTLER G.D. 1974. An algorithm for temperature dependent growth rate simulation. Canadian Enomologist. 106. 519-524.

SUNDERELAND K.D. 1988. Quantitative methods for the detecting invertebrate predation occuring in the field. Annals of Applied Biology. 112. 201-224.

SWENSON K.G. 1969. Plant susceptibility to virus infection by insect transmission. In "Virus Vectors and Vegetation" ed. K. Maramorosch. Wiley. 143-157.

SYLVESTER E.S. 1965. The latent period of pea-enation mosaic virus in the pea aphid *Acyrthosiphon pisum* (Harris) - an approach to its estimation. Virology. 25. 62-67.

TATCHELL G.M. PLUMB R.T. & CARTER N. 1988. Migration of alate morphs of the bird cherry aphid (*Rhopalosiphum padi*) and implications for the epidemiology of barley yellow dwarf virus. Annals of Applied Biology. 112. 1-11.

TAYLOR L.R. 1977. Aphid forecasting and the Rothamsted Insect Survey. Journal of the Royal Agricultural Society of England. 138. 75-97.

TAYLOR L.R. 1983. EURAPHID : synoptic monitoring for migrant vector aphids. In 'Plant Virus Epidemiology' eds. R.T. Plumb & J.M. Thresh. Blackwells. 133-146.

TAYLOR L.R. & PALMER J.M.P. 1972. Aerial sampling. In 'Aphid Technology'. ed. H.F. van Emden. Academic Press. 189-234.

TAYLOR L.R. WOIWOD I.P. & PERRY J.N. 1979. The negative binomial distribution as a dynamic ecological model and the density-dependence of k. Journal of Animal Ecology. 48. 289-304.

VICKERMAN G.P. & SUNDERELAND K.D. 1975. Arthropods in cereal crops : nocturnal activity vertical distribution and aphid predation. Journal of Applied Ecology. 12. 755-766.

VICKERMAN G.P. & WRATTEN S.D. 1979. The biology and pest status of cereal aphids (Hemiptera:Aphididae) in Europe : a review. Bulletin of Entomological Research. 69. 1-32.

VORLEY W.T. & WRATTEN S.D. 1985. A simulation model of the role of parasitoids in the population development of *Sitobion avenae* (Hemiptera:Aphididae) on cereals. Journal of Applied Ecology. 22. 813-823.

WATSON M.A. 1967. Transmission of cucumber mosaic virus from plants infected with two strains. Report of Rothamsted Experimental Station 1966. p120.

WATT A.D. DIXON A.F.G 1981 The effect of cereal growth stages and overcrowding of aphids on the induction of alatae in *Sitobion avenae* and its consequences for population growth. Ecological Entomology. 6. 441-447.

WIKTELIUS S. & PETTERSON J. 1985. Simulations of bird cherry-oat aphid population dynamics : a tool for developing strategies for breeding aphid-resistent plants. Agriculture Ecosystems and the Environment. 14. 159-170.

WILLIAMS C.T. 1980. Low temperature mortality of cereal aphids. Bulletin SROP/WPRS. 3. 63-66.

WIT de C.T. & GOUDRIAAN J. 1978. Simulation of ecological processes. Pudoc. 175pp.

WRATTEN S.D. 1977. Reproductive starategy of winged and wingless morphs of the aphids *Sitobion avenae* and *Metopolophium dirhodum*. Annals of Applied Biology. 85. 319-331.

ZADOKS J.C. CHANG T.T. & KONZAK C.F. 1974. A decimal code for the growth stage of cereals. Weed Research. 14. 415-421.

ZITTER T.A. 1977. Epidemiology of aphid-borne viruses. In 'Aphids as Virus Vectors' eds. K.F. Harris & K. Maramorosch. Academic Press. 385-412.

Appendix 1. FORTRAN listing of the aphid sub-model.

```
C
C*****1.INITIALISATION
C
      REAL NEWNY,NWNY,MXTT,MNTT,MORT,LOWT,MEANT,INSAL,INSAP,NP,NL,
     *INOC,INFEC,LTNT,LP,K,INC0,INC,MR,MD
      DIMENSION INSAL(2,50),INSAP(2,50),APTER(2,150),ALATE(2,150),
     *IMM(450),MNTT(450),MXTT(450)
C
C INPUT ARRAY SIZES
C
      DATA INSTP,ADSTP/50,150/
      OPEN(UNIT=8,STATUS='NEW',CARRIAGECONTROL='LIST')
C
C ZERO ALL INSTARS AND VARIABLES
  309 TOTAD=0.0
      TOTALA=0.0
      TOTPRD=0.0
      TOTLRD=0.0
      TOTFOR=0.0
      TOTALF=0.0
      TOTTHI=0.0
      TOTALT=0.0
      TOTSEC=0.0
      TOTALS=0.0
      TOTFIR=0.0
      TOTALP=0.0
      TOTAPN=0.0
      TOTALN=0.0
      TOTALE=0.0
      TOTDEN=0.0
      DO 400 I=1,50
      DO 401 J=1,2
      INSAL(J,I)=0.0
      INSAP(J,I)=0.0
  401 CONTINUE
  400 CONTINUE
      DO 402 I=1,150
      DO 403 J=1,2
      APTER(J,I)=0.0
      ALATE(J,I)=0.0
  403 CONTINUE
  402 CONTINUE
      DO 404 I=1,450
      IMM(I)=0.0
      MNTT(I)=0.0
      MXTT(I)=0.0
  404 CONTINUE
C
C
C*****2.DATA INPUT
C
C INPUT START AND FINISH DAYS AND SKIP VALUE FOR APHID INPUT
C
      READ*,ISTART,IFINIS
C
C SENSITIVITY FACTORS:SEN1=TEMP,SEN2=ADULT LONG,SEN3=INSTAR LENGTH,
C SEN4=SURVIVAL,SEN5=REPROD,SEN6=IMM,SEN7=TRANSMISSION COEFICIENT,
C SEN9=ALATE NYMPHS
C
      READ*,SEN1,SEN2,SEN3,SEN4,SEN5,SEN6,SEN7,SEN9,SEN10,SEN11
C
C TEMP DATA, MAXS THEN MINS
C
      READ (*,*)(MXTT(I),I=ISTART-1,IFINIS)
      READ (*,*)(MNTT(I),I=ISTART,IFINIS+1)
C
```

```
C ADD SENSITIVITY FACTOR
C
      DO 10 I=ISTART-1,IFINIS+1
      MXTT(I)=MXTT(I)+(SEN1)
      MNTT(I)=MNTT(I)+(SEN1)
   10 CONTINUE
C
C INPUT OF INITIAL CROP GROWTH STAGE AND NOS PLANTS/M
C
      READ*,TILERS
C
C CALCULATE NOS OF ALATES LANDING PER PLANT FOR EACH ALATE
C CAUGHT IN SUCTION TRAP
C
      TAYPAL=0.0237/TILERS
C
C IMMIGRATION DATA
C
C START AND FINISH DAYS OF IMMIGRATION AND CONC FACTOR
C
      READ*,IMSTAR,IMFINI,INCONF
C
C NOW THE ACTUAL DATA
C
      READ (*,*)(IMM(I),I=IMSTAR,IMFINI)
C
C DATA INPUT FINISHED
C
C HEADINGS ARE NOW PRINTED
C
      WRITE(2,600)
  600 FORMAT(1H1,'IDAYY   I-APT   II-APT   III-APT   IV-APT   V-APT
     *I-ALT   II-ALT   III-ALT   IV-ALT   V-ALT   TOTYN'//)
      WRITE(3,601)
  601 FORMAT(1H0,'GSTAGE   PRD-AD   ALTIM   TOTALE   TOTAL DENSITY
     *AFIDUN   TOTDDG TOTDEV'///)
C
C
C
C ********MODEL BEGINS******
C
C
      DO 107 IDAYY=ISTART,IFINIS
C
C
C****4.IMMIGRATION
C
C BASIC DATA HAS ALREADY BEEN INPUT. FIRST SKIP STATEMENT
C
      IF(IDAYY.GE.IMSTAR.AND.IDAYY.LE.IMFINI.AND.IMM(IDAYY).
     *NE.0.0)THEN
C
      ALTIM=IMM(IDAYY)*INCONF*TAYPAL*SEN6
      ELSE
      ALTIM=0.0
      END IF
      ALATE(1,1)=ALTIM
      TOTALA=TOTALA+ALATE(1,1)
C
C
C*****5.DEVELOPMENT AND SURVIVAL
C
C
C CALCULATE MEAN TEMP
C
      UPPT=MXTT(IDAYY)
      LOWT=MNTT(IDAYY)
      MEANT=(UPPT+LOWT)/2.0
C
```

```
C ADULT LIFESPAN APPROX 17 DAYS. RATE OF DEVELOP CONSTANT
C INCREASES BY SAME AMOUNT EACH DAY IRRESPECTIVE OF TEMP
C
      DEVAD=0.0605
C
C***CALCULATE DAILY DEVELOPMENT RATE USING A LOGISTIC RELATIONSHIP
C FOR NYMPHS, BEGINING WITH APTEROUS MORPHS
C
      AP=-0.01513
      NP=0.28998
      BP=-0.13761
      CP=16.91035
C
      DAPMAX=AP+(NP/(1+EXP(BP*(UPPT-CP))))
      DAPMIN=AP+(NP/(1+EXP(BP*(LOWT-CP))))
      DAPMEAN=AP+(NP/(1+EXP(BP*(MEANT-CP))))
C
      APDEV=(DAPMAX+DAPMIN+DAPMEAN)/3.0
C
C ALATE NYMPHS
C
      AL=-0.01413
      NL=0.25793
      BL=-0.13591
      CL=16.82999
C
      DALMAX=AL+(NL/(1+EXP(BL*(UPPT-CL))))
      DALMIN=AL+(NL/(1+EXP(BL*(LOWT-CL))))
      DALMEAN=AL+(NL/(1+EXP(BL*(MEANT-CL))))
C
      ALDEV=(DALMAX+DALMIN+DALMEAN)/3.0
C
C******LOW TEMPERATURE MORTALITY
C
C DECLARE LIMIT FOR DAY-DEGREES
C
      SURLIM=2.8
C
C CALL DAY-DEGREE SUBROUTINE
C
      CALL DAYDEG(UPPT,LOWT,SURLIM,DDAS,DDBS)
C
C CALCULATE SURVIVAL
C
      SURV=(0.9511-0.0173*DDBS)*SEN4
C
C DECLARE THE PROPORTION EACH INSTAR OCCUPIES IN TOTAL
C DEVELOPMENT. ORDER IS APTEROUS PRD, APTEROUS 4THS,....
C
      APPLIM=0.9463
      APFLIM=0.6985
      APTLIM=0.4800
      APSLIM=0.2571
C
      ALPLIM=0.9522
      ALFLIM=0.6211
      ALTLIM=0.4265
      ALSLIM=0.2289
C
C CALL UP DEVELOPMENT AND SURVIVAL SUBROUTINE
C
C APTEROUS ADULTS
C
      IF(APTER(1,ADSTP).NE.0.0)GO TO 1001
      IF(TOTAD.NE.0.0)CALL ADDEV(APTER,ADSTP,TOTAD,OLDAPH,SURV,
     *DEVAD,SEN2)
C
C ALATE ADULTS
C
```

```
      IF(ALATE(1,ADSTP).NE.0.0)GO TO 1001
      IF(TOTALA.NE.0.0)CALL ADDEV(ALATE,ADSTP,TOTALA,OLDAPH,SURV,
     *DEVAD,SEN2)
C
C APTEROUS NYMPHS
C
      IF(INSAP(1,INSTP).NE.0.0)GO TO 1001
      IF(TOTAPN.NE.0.0)CALL INSDEV(INSAP,INSTP,APTER(1,1),TOTPRD,
     *TOTFOR,TOTTHI,TOTSEC,TOTFIR,SURV,APDEV,APPLIM,APFLIM,APTLIM,
     *APSLIM,SEN3)
C
C ALATE NYMPHS
C
      IF(INSAL(1,INSTP).NE.0.0)GO TO 1001
      IF(TOTALN.NE.0.0)CALL INSDEV(INSAL,INSTP,ALATED,TOTLRD,
     *TOTALF,TOTALT,TOTALS,TOTALP,SURV,ALDEV,ALPLIM,ALFLIM,ALTLIM,
     *ALSLIM,SEN3)
C
C TOTAL UP ADULTS
C
      TOTAD=TOTAD+APTER(1,1)
      TOTALA=TOTALA+ALATE(1,1)
      TOTALE=TOTALE+ALATED
C
C******6.REPRODUCTION AND MORPH DETERMINATION
C
C DECLARE PARAMETERS IN THE LOGISTIC RELATIONSHIP
C USED IN REPRODUCTION
      AR=-0.03568
      CR=5.92466
      BR=0.31851
      MR=12.03233
C
C CALCULATE THE MAXIMUM, MINIMUM AND MEAN REPRODUCTIVE
C RATES FOR APTEROUS ADULTS
C
      FECUP=AR+(CR/(1+EXP(-BR*(UPPT-MR))))
      IF(FECUP.LT.0.0)FECUP=0.0
      FECLO=AR+(CR/(1+EXP(-BR*(LOWT-MR))))
      IF(FECLO.LT.0.0)FECLO=0.0
      FECME=AR+(CR/(1+EXP(-BR*(MEANT-MR))))
      IF(FECME.LT.0.0)FECME=0.0
C
C CALCULATE MEAN REPRODUCIVE RATE FOR APTEROUS ADULTS
C
      FEC=((FECUP+FECLO+FECME)/3.0)*SEN5
C
C CALCULATE REPRODUCTIVE RATE FOR ALATE ADULTS
C ASSUMING THEY ARE 1.3X LESS FECUND
C
      ALFEC=FEC/1.3
C
      NEWNY=0.0
      NWNY=0.0
C
C AGE-SPECIFIC PROCEDURE - THE REPRODUCTION POTENTIAL
C OF AN ADULT DEPENDS ON ITS AGE IE THE OLDER AN ADULT
C IS THE LESS FECUND IT IS
C BEGIN WITH APTEROUS ADULTS
C
      DO 50 I=1,ADSTP
C
      FCTO=0.0117+1.1375*APTER(2,I)
      NEWNY=NEWNY+(FEC*FCTO*APTER(1,I))
C
      ALFCTO=0.0117+1.1375*ALATE(2,I)
      NWNY=NWNY+(ALFEC*ALFCTO*ALATE(1,I))
C
   50 CONTINUE
```

```
C
C DECIDE THE PROPORTION OF NYMPHS WHICH ARE ALATIFORMS
C
      AD=0.0021
      CD=0.9911
      BD=0.0757
      MD=67.4164
      ALPROP=(AD+(CD/(1+EXP(-BD*(TOTDEN-MD)))))*SEN9
      IF(ALPROP.GT.1.0)ALPROP=1.0
      IF(ALPROP.LT.0.0)ALPROP=0.0
      INSAL(1,1)=(NWNY+NEWNY)*ALPROP
      IF(INSAL(1,1).LT.0.0)INSAL(1,1)=0.0
C
C NOW DECIDE THE NUMBER WHICH ARE APTERIFORM
C
      INSAP(1,1)=NEWNY+NWNY-INSAL(1,1)
      IF(INSAP(1,1).LT.0.0)INSAP(1,1)=0.0
C
C
C CALCULATE TOTALS AND SET AGES
C
      TOTFIR=TOTFIR+INSAP(1,1)
      TOTALP=TOTALP+INSAL(1,1)
      INSAL(2,1)=0.0
      INSAP(2,1)=0.0
C
C TOTAL NYMPHS
C
      TOTAPN=TOTFOR+TOTTHI+TOTSEC+TOTFIR
      TOTALN=TOTALF+TOTALT+TOTALS+TOTALP
C
C TOTAL UP DENSITY
C
      TOTDEN=TOTAD+TOTALA+TOTPRD+TOTFOR+TOTALF+TOTTHI+
     *TOTALT+TOTSEC+TOTALS+TOTFIR+TOTALP
C
C TOTAL UP  POTENTIAL VECTORS
C
      PVECS=(TOTAD+TOTPRD+TOTFOR+TOTALF+TOTTHI+TOTALT+
     *TOTSEC+TOTALS+TOTFIR+TOTALP)*TILERS
C
C*******8.OUTPUT
C
C
      TOTYN=TOTFIR+TOTSEC+TOTTHI+TOTALP+TOTALS+TOTALT
      WRITE(2,132)IDAYY,TOTFIR,TOTSEC,TOTTHI,TOTFOR,TOTAD,
     *TOTALP,TOTALS,TOTALT,TOTALF,TOTALA,TOTYN
  132 FORMAT(I4,11F10.4)
      WRITE(3,39)IDAYY,TOTPRD,ALTIM,TOTALE,TOTDEN,
     *AFIDUN,TOT,DEVTOT
   39 FORMAT(I4,7F10.4)
 1000 CONTINUE
  107 CONTINUE
      GO TO 1003
C
C WARNING MESSAGE WHEN AN ARRAY OVERFLOWS
C
 1001 WRITE(2,1002)
 1002 FORMAT(1H1,'ARRAY EXCEEDED')
 1003 CONTINUE
C
C
C*****10.INPUT VARIABLES PRINTED
C
C
 1004 CONTINUE
      WRITE(2,1010)
 1010 FORMAT(1H0,'CONC FACTORS AND SUCTION TRAP DATA'/)
      WRITE(2,1012)INCONF,IMSTAR,IMFINI
```

```
 1012 FORMAT(3I4)
      WRITE(2,1013)(IMM(I),I=IMSTAR,IMFINI)
 1013 FORMAT(10I4)
      WRITE(2,1019)
 1019 FORMAT(1H0,'SENSTIVITY ANALYSIS FACTORS')
WRITE(2,1114)SEN1,SEN2,SEN3,SEN4,SEN5,SEN6,SEN9,SEN10,SEN11
 1114 FORMAT(1H0,9F5.2)
      WRITE(2,1022)TILERS,TAYPAL
 1022 FORMAT(1H0,'TILLERS PER SQM=',F8.2,'ALATES PER TILLER
     *PER SUCTION TRAP APHID=',F10.8)
      WRITE(2,9996)
 9996 FORMAT(1H0,'MAX TEMPS'/)
      WRITE(2,9995)(MXTT(I),I=ISTART-1,IFINIS)
 9995 FORMAT(15F7.2)
      WRITE(2,9994)
 9994 FORMAT(1H0,'MIN TEMPS'/)
      WRITE(2,9993)(MNTT(I),I=ISTART,IFINIS+1)
 9993 FORMAT(15F7.2)
C
C
C******THE END*******
      STOP
      END
      SUBROUTINE ADDEV(ADULTS,ADSTP,TOTAL,OLDAPH,SURV,DEVOP,SEN2)
      DIMENSION ADULTS(2,ADSTP)
      TOTAL=0.0
      OLDAPH=0.0
C
C UPDATING WITH OLDEST AGE CLASS
C
      DO 109 I=ADSTP,2,-1
C SKIP STATEMENT IF ELEMENT IS EMPTY
      IF(ADULTS(1,I-1).NE.0.0)THEN
C MOVE INTO NEXT AGE CLASS WITH SOME DEATHS
      ADULTS(1,I)=ADULTS(1,I-1)*SURV
C AGE IS UPDATED
      ADULTS(2,I)=ADULTS(2,I-1)+DEVOP
C ZEROING TAKES PLACE
      ADULTS(1,I-1)=0.0
      ADULTS(2,I-1)=0.0
C COMPARE WITH LONGEVITY
      IF(ADULTS(2,I).GE.1.0*SEN2)THEN
      OLDAPH=OLDAPH+ADULTS(1,I)
      ADULTS(1,I)=0.0
      ADULTS(2,I)=0.0
      END IF
C TOTAL UP
      TOTAL=TOTAL+ADULTS(1,I)
      END IF
  109 CONTINUE
      RETURN
      END
      SUBROUTINE INSDEV(APHIDS,INSTP,ADULTS,PRD,FOURS,THIRS,SECS,
     *FIRS,SURV,DEVOP,PLIM,FLIM,TLIM,SLIM,SEN3)
      DIMENSION APHIDS(2,INSTP)
      PRD=0.0
      FOURS=0.0
      THIRS=0.0
      SECS=0.0
      FIRS=0.0
C UPDATING STARTING WITH OLDEST AGE CLASS
      DO 110 I=INSTP,2,-1
C SKIP STATEMENT IF ELEMENT IS EMPTY
      IF(APHIDS(1,I-1).NE.0.0)THEN
C MOVE APHIDS INTO NEXT AGE CLASS WITH SOME DEATHS
      APHIDS(1,I)=APHIDS(1,I-1)*SURV
C AGE UDPATED
      APHIDS(2,I)=APHIDS(2,I-1)+DEVOP
C ZEROING
```

```
      APHIDS(1,I-1)=0.0
      APHIDS(2,I-1)=0.0
C CHECK AGAINST LONGEVITY TO SEE WHICH INSTAR APHID IS IN
      IF(APHIDS(2,I).GE.1.0*SEN3)THEN
      ADULTS=ADULTS+APHIDS(1,I)
      APHIDS(1,I)=0.0
      APHIDS(2,I)=0.0
      ELSE IF(APHIDS(2,I).GE.PLIM)THEN
      PRD=PRD+APHIDS(1,I)
      ELSE IF(APHIDS(2,I).GE.FLIM)THEN
      FOURS=FOURS+APHIDS(1,I)
      ELSE IF(APHIDS(2,I).GE.TLIM)THEN
      THIRS=THIRS+APHIDS(1,I)
      ELSE IF(APHIDS(2,I).GE.SLIM)THEN
      SECS=SECS+APHIDS(1,I)
      ELSE
      FIRS=FIRS+APHIDS(1,I)
      END IF
      END IF
  110 CONTINUE
      RETURN
      END
      SUBROUTINE DAYDEG(TMAX,TMIN,TLIM,DDA,DDB)
      TMEAN=(TMAX+TMIN)/2.0
      IF(TMIN.GE.TLIM)GO TO 10
      IF(TMAX.LE.TLIM)GO TO 20
      IF((TMAX-TLIM).GE.(TLIM-TMIN))GO TO 30
C
C TMAX>TLIM, TMIN<TLIM, TMAX-TLIM<TLIM-TMIN
C
      DDA=(TMAX-TLIM)/4.0
      DDB=(TLIM-TMIN)/2.0-(TMAX-TLIM)/4.0
      RETURN
   10 CONTINUE
C
C TMIN GE TLIM
C
      DDA=TMEAN-TLIM
      DDB=0.0
      RETURN
   20 CONTINUE
C
C TMAX LE TLIM
C
      DDA=0.0
      DDB=TLIM-TMEAN
      RETURN
   30 CONTINUE
C
C TMAX>TLIM, TMIN<TLIM, TMAX-TLIM GE TLIM-TMIN
C
      DDA=(TMAX-TLIM)/2.0-(TLIM-TMIN)/4.0
      DDB=(TLIM-TMIN)/4.0
      RETURN
      END
```

Appendix 2. FORTRAN listing of the virus sub-model.

```
C
C ******* 1. INITITALISATION
C
      REAL LTNT,INC,LP,INFEC
      INTEGER DAY
      DIMENSION DEN(390),VIMM(390),LTNT(390),SORC(390),PLDEN(390),
     *INFEC(390),RAIN(390),TMAX(390),TMIN(390)
      CHARACTER*1 DUMMY
C
C ZERO ARRAYS AND VARIABLES
C
      ALINF=0.0
      INC=0.0
      PROBAC=0.0
      APINF=0.0
      PCINF=0.0
      DO 10 I=1,390
      DEN(I)=0.0
      VIMM(I)=0.0
      LTNT(I)=0.0
      SORC(I)=0.0
   10 CONTINUE
C
C ******** 2. DATA INPUT
C
C READ DENSITIVITY ANALYSIS VARIABLES
C FOR STANDARD RUN SET TO 0 OR 1
C
      READ*,SEN1,SEN2,SEN3,SEN4
C
C READ START AND FINISH DAYS OF SIMULATION
C
      READ*,ST,FIN
C
C NOS PLNATS PER SQ. M. AND LATENT PERIOD OF VIRUS IN PLANT
C
      READ*,PLANTS,LP
      LP=LP+SEN1
C
C READ INITIAL CROP GROWTH STAGE
C
      READ*,GS
      GS=GS+SEN2
C
C CALCULATE CROP PHYSIOLOGICAL AGE
C
      DDP=(0.047-SQRT(0.00221+(0.00006*(0.57-GS))))/0.00003
C
C READ START AND FINSIH DAYS OF IMMIGRATION
C
      READ*,STIMM,FINIMM
C
C READ NOS OF INFECTIVE ALATES CAUGHT IN SUCTION TRAP
C
      READ(*,*)(VIMM(I),I=STIMM,FINIMM)
C
C READ IN APHID DENSITY
C READ PAST TITLE LINES OF INPUT FILE
C
      DO 5 I=1,4
      READ(1,7)DUMMY
    7 FORMAT(A1)
    5 CONTINUE
C
C READ IN ACTUAL DATA
C
```

```
      READ(1,12)(DEN(I),I=ST,FIN)
   12 FORMAT(34X,F10.4)
C
      DO 17 I=ST,FIN
      DEN(I)=DEN(I)*SEN4
   17 CONTINUE
C
C READ IN DAILY RAINFALL
C
      READ(*,*)(RAIN(I),I=ST,FIN)
      DO 15 I=ST,FIN
      RAIN(I)=RAIN(I)*SEN3
   15 CONTINUE
C
C READ IN DAILY MAX AND MIN TEMPS
C
      READ(*,*)(TMAX(I),I=ST,FIN)
      READ(*,*)(TMIN(I),I=ST,FIN)
C
C DATA INPUT FINISHED
C
C WRITE HEADINGS OF OUTPUT FILE
C
      WRITE(2,13)
   13 FORMAT(//' DAY     ALINF     PLDEN       INC     PROBAC
     *PVECS    APINF     INFEC      LTNT      SORC     HLTHY
     *PCINF'//)
C ------------------------------------------------------------
C MODEL LOOP BEGINS
C ------------------------------------------------------------
C
      DO 999 DAY=ST,FIN
C
C CALCULATE NUMBER OF INFECTIONS CAUSED BY ALATES
C
      ALINF=VIMM(DAY)*0.0237
C
C CALCULATE DENSITY OF APHIDS PER PLANT
C
      PLDEN(DAY)=DEN(DAY)*PLANTS
C
C CALCULATE THE INCIDENCE OF APHIDS GIVEN THE ABOVE DENSITY
C
      INC=1-EXP(-EXP(-0.7259+0.8751*ALOG(DEN(DAY))))
C
C CALCULATE THE PROBABILITY OF ACQUIRING VIRUS
C
      PROBAC=SORC(DAY-1)/PLANTS
C
C CACULATE THE DENSITY OF POTENTIAL VECTORS
C
      PVECS=PLDEN(DAY)*PROBAC
C
C CALCULATE THE EFFECT OF RAINFALL KNOCKING APHIDS OFF PLANTS
C
      IF(RAIN(DAY).LT.12.0)THEN
      RKNOC=0.0208*RAIN(DAY)
      ELSE
      RKNOC=0.05+0.0167*RAIN(DAY)
      ENDIF
C
C CALCULATE THE SHELTER PROVIDED BY CROP GROWTH STAGE
C
      IF(DDP.LT.430.0)THEN
      PKNOC=1.5
      ELSE
      PKNOC=1.0
      ENDIF
C
```

```
C UPDATE CROP GROWTH STAGE
C
      PLIM=1.0
      CALL DAYDEG(TMAX(DAY),TMIN(DAY),PLIM,DD)
      DDP=DDP+DD
C
C CALCULATE THE NUMBER OF INFECTIONS CAUSED BY APTARAE
C
      APINF=PVECS*0.00166*(1+RKNOC)*PKNOC
C
C TOTAL UP THE NUMBER OF INFECTIONS FROM ALATES AMD APTERAE
C
      INFEC(DAY)=ALINF+APINF
C
C CALCULATE THE NUMBER OF LATENT PLANTS
C
      DO 100 I=DAY-LP+1,DAY-1
      LTNT(DAY)=LTNT(DAY)+INFEC(I)
  100 CONTINUE
C
C CALCULATE THE NUMBER OF SOURCE PLANTS
C
      DO 200 I=1,DAY-LP
      SORC(DAY)=SORC(DAY)+INFEC(I)
  200 CONTINUE
C
C CALCULATE THE NUMBER OF HEALTHY PLANTS
C
      HLTHY=PLANTS-LTNT(DAY)-SORC(DAY)
C
C CALCULATE THE PERCENTAGE INFECTION
C
      PCINF=((PLANTS-HLTHY)/PLANTS)*100.0
C
C OUTPUT THE RESULTS
C
      WRITE(2,111)DAY,ALINF,PLDEN(DAY),INC,PROBAC,PVECS,APINF,
     *INFEC(DAY),LTNT(DAY),SORC(DAY),HLTHY,PCINF
  111 FORMAT(I4,11F10.4)
  999 CONTINUE
C
C MODEL LOOPE ENDS
C
C
C OUTPUT OF INPUT CONDITIONS FOR EASE OF VERIFICATION
C
      WRITE(2,222)
  222 FORMAT(//' START AND FINSIH DAYS '/)
      WRITE(2,223)ST,FIN
  223 FORMAT(2F6.0)
      WRITE(2,333)
  333 FORMAT(//' SOWING RATE AND PLANT LATENT PERIOD '/)
      WRITE(2,334)PLANTS,LP
  334 FORMAT(2F6.0)
      WRITE(2,444)
  444 FORMAT(//' INFECTIVE ALATE IMMIGRATION '/)
      WRITE(2,445)(VIMM(I),I=STIMM,FINIMM)
  445 FORMAT(10F5.2)
      WRITE(2,555)
  555 FORMAT(//' SKIP VALUE AND PROB OF MOVING '/)
      WRITE(2,556)ISKIP,PROBMV
  556 FORMAT(I4,F5.2)
      WRITE(2,666)
  666 FORMAT(//' APHID DENSITY '/)
      WRITE(2,667)(DEN(I),I=ST,FIN)
  667 FORMAT(10F10.4)
      STOP
      END
C
```

```
      SUBROUTINE DAYDEG(TMX,TMN,TLM,DDA)
      TMN=(TMX+TMN)/2.0
      IF(TMN.GE.TLM)GO TO 10
      IF(TMX.LE.TLM)GO TO 20
      IF((TMX-TLM).GE.(TLM-TMN))GO TO 30
C
C TMX>TLM, TMN<TLM, TMX-TLM<TLM-TMX
C
      DDA=(TMX-TLM)/4.0
      RETURN
   10 CONTINUE
C
C TMN≥TLM
C
      DDA=TMN-TLM
      RETURN
   20 CONTINUE
C
C TMX≤TLM
C
      DDA=0.0
      RETURN
   30 CONTINUE
C
C TMX>TLM, TMN<TLM, TMX-TLM≥TLM-TMN
C
      DDA=(TMX-TLM)/2.0-(TLM-TMN)/4.0
      RETURN
      END
```