Annu. Rev. Phytopathol. 1990. 28:393–424

EPIDEMIOLOGY OF BARLEY YELLOW DWARF: A Study in Ecological Complexity

Michael E. Irwin

University of Illinois and Natural History Survey, 607 East Peabody Drive, Champaign, Illinois 61820

J. Michael Thresh

Overseas Development Natural Resources Institute, Chatham Maritime, Kent ME4 4TB, England

KEY WORDS: persistently transmitted virus, virus vectors, aphid movement BYDV pathosystem, integrated pest management

INTRODUCTION

Most pathosystems encompass intermeshed biological relationships between a pathogen and its hosts in a shared environment. If additional biological entities such as vectors of the pathogen are integral components, as with many plant virus systems, the complexity increases substantially. When the virus is widespread in several crop and perennial plant species, has a number of distinct variants, or the variants are spread selectively by several vector species, the complexity of the ecological interactions is even greater. All of these elements, when interacting concurrently, create exceptionally complex ecological pathosystems. The disease complex known as barley yellow dwarf epitomizes such systems.

Investigating virus variants, their plant hosts and vectors, and elucidating the confounding resultant interactions in diverse and fluctuating environments in different regions of the world is an extremely difficult task, but one that must be undertaken to gain an understanding of the pathosystem. To do so

0066-4294/90/0901-0393$02.00

requires a major multidisciplinary effort involving scientists investigating plant/virus, virus/vector, and plant/vector interactions (Figure 1). The team must also include researchers that understand physical environments and their influence on biological processes and those able to analyze, interpret, and model these associations. Through such efforts, scientific principles that govern epidemics of barley yellow dwarf can be deduced and placed in appropriate ecological and economic contexts to facilitate forecasting and management of such epidemics on local and regional scales.

Literature on barley yellow dwarf virus (BYDV) and its vector species is extensive—arguably greater than for any other plant virus pathosystem. Much of the known data concerning BYDV ecology is widely quoted and has been discussed in previous reviews, including a recent edited book (14). Conflicting views are not uncommon.

This article is not meant to be a comprehensive reevaluation of barley yellow dwarf epidemiology; instead it focuses on some of the more controversial issues and attempts to put many of the known facts into an epidemiological context. Because we believe that vector movement has been inadequately studied and has not been incorporated into the foundation of barley yellow dwarf epidemiology, this article emphasizes, but is by no means restricted to, the role of vectors.

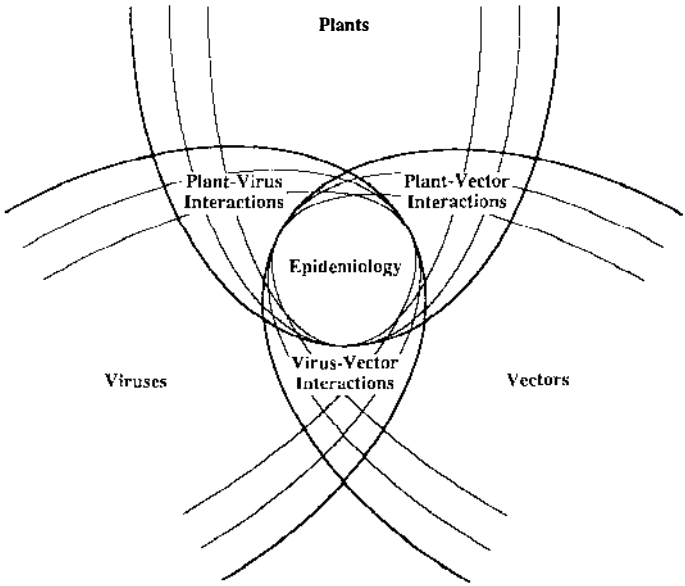

Figure 1 Conceptual diagram of the component parts and disciplines involved in the study of barley yellow dwarf epidemiology, all interacting in a shared environment.

COMPONENTS OF THE PATHOSYSTEM

The barley yellow dwarf pathosystem has three biotic elements: the luteoviruses that form the BYDV complex, the different aphid species that are virus vectors, and the grass and other plant species that are hosts of both the viruses and their vectors. To understand the epidemiology of barley yellow dwarf, the biotic components must be understood.

BYDV Variants, Their Host Plants, and Their Vectors

Barley yellow dwarf is the most widespread and economically important virus disease of cereals, worldwide (87). This disease affects over 100 species in the family Poaceae, including barley, wheat, oats, sorghum, rye, triticale, maize, rice, and many wild grasses (103). Wild annual and perennial grasses, graminaceous weeds, and volunteer cereals play an important role in the epidemiology of barley yellow dwarf, serving as hosts and thus reservoirs of this virus complex.

Barley yellow dwarf is caused by what might best be considered a continuous, often overlapping, range of luteoviruses, only some of which are closely related serologically and share common aphid vectors (87). The various isolates are grouped into strains on the basis of their transmissibility by over 20 species of aphids, in a circulative, persistent manner. Once the virus is acquired, the vector is potentially infective for life; however, BYDV is inefficiently transmitted when access periods for vector inoculation are relatively short, i.e. less than 24 hours. Thus, barley yellow dwarf epidemics in cereals are due almost exclusively to aphid species that colonize (i.e. aphids that feed for a considerable amount of time, usually become established and reproduce), rather than by itinerants that pass through and probe the crop while in transit.

Based upon the principal aphid species transmitting different isolates of BYDV, Rochow (93) characterized and designated five strains found in New York State and gave each an acronym from the initial letters of its principal vector species. Transmitted specifically are MAV by *Sitobion avenae* (previously placed in the genus *Macrosiphum*); RPV by *Rhopalosiphum padi;* RMV by *Rhopalosiphum maidis;* and SGV by *Schizaphis graminum*. The PAV strain is transmitted nonspecifically by *R. padi,* an efficient vector, and by *S. avenae,* a somewhat less efficient vector.

These strain designations have been adopted almost universally by many investigators of barley yellow dwarf. However, because isolates of these strains vary to some degree, it has become commonplace to use the suffix "-like," e.g. RPV-like, when referring to isolates that fit a particular designation but have not been fully characterized. A well-characterized isolate is usually designated by "-locality," e.g. RPV-IL for a specific isolate of RPV from the state of Illinois, which resembles the strain designation of Rochow

(95). In this paper, we use the term "variant" as an adjective to modify some strain designations. Thus an "RPV variant" is an isolate of BYDV that fits most closely the criteria for the RPV strain but is not an isolate that has been well characterized, nor is it among the isolates upon which Rochow based the original designation.

The major vectors that colonize cereals have dissimilar biologies (10). *S. avenae* is monoecious and holocyclic, developing entirely on grasses. *R. padi* and *Metopolophium dirhodum* are heteroecious and holocyclic, colonizing cereals and other Poaceae as secondary hosts after overwintering on their primary woody hosts, *Prunus padus* and *Rosa* spp., respectively; *M. dirhodum* can, however, overwinter anholocyclically in warmer climates (10). *S. graminum* infests barley, wheat, sorghum, maize, and many grass species, and reproduces anholocyclically in North America, where it overwinters mainly in the southern states (52). However, it can overwinter holocyclically on graminaceous hosts in parts of northern Europe and perhaps even North America (10). *R. maidis* is entirely anholocyclic, developing on a wide range of grasses, including barley, maize, and sorghum, but in central North America it does not overwinter north of southern Illinois and Arkansas (112).

Although most isolates of BYDV infect many graminaceous species, there is evidence that a few isolates of BYDV are adapted to particular hosts. Rochow et al (94, 97), for instance, noted that virus isolates transmitted nonspecifically cause more severe symptoms than do isolates transmitted specifically. Furthermore, Baltenberger et al (7) observed that a cultivar responded differently to an RPV variant and a PAV variant. Additionally, mixed infections abound in the field, suggesting that cross protection is not an important factor limiting superinfection. For example, a survey in Pennsylvania by Gildow et al (39) determined that 16% of the BYDV-infected plants contained more than one strain of BYDV. Baltenberger et al (7) concluded that dual infection by RPV and PAV variants caused more severe symptoms than that by either one alone. When more than one strain of BYDV was spreading through fields, A. Comeau (personal communication) observed that a greater proportion of the plants contained mixed infections during severe than during mild epidemics. This could have important economic as well as epidemiological consequences.

In susceptible plants, systemic movement of BYDV occurs between one and three days of infection, depending on the length of the inoculation-access period (16, 40). Plant species, cultivar, and temperature all affect the rate of systemic movement; rates of translocation are greater in sensitive than in more tolerant cultivars (53). The rate of systemic movement may also depend to some extent on the BYDV variant involved.

Temporal changes in virus concentration during the course of infection can vary with BYDV strain. Skaria et al (101) found that concentrations of PAV-P

antigen during a 30-day period, in paired cultivars of wheat, barley, and oats, differ slightly with plant species, but invariably reach peak titer 12 days after inoculation. As indirect evidence of differences in virus concentration between BYDV isolates, Gill (41) found that aphid transmission of an MAV variant in oats fluctuated cyclically over a 38-day period, whereas transmission of an SGV-nonspecific variant from oats over a 33-day period reached a single peak 9–14 days after inoculation (42).

Plant age at time of virus inoculation changes the likelihood and course of infection (107). For example, virus antigen concentrations in oats inoculated at the one- to two-leaf stage with a severe PAV variant reached maximum levels in roots after seven to eight days, and concentrations were three to four times greater than in leaves. Concentrations in similar oat plants inoculated at the four- to five-leaf stage reached maximum virus titer in roots after ten days and in leaves after 18 days, but the concentration in leaves was two to five times greater than in roots (30).

Time of infection relative to plant phenology has economic implications by restricting the time available for an epidemic to develop. Gildow & Frank (38) confirmed that early infections of oats with a PAV variant limit yields significantly more than do slightly later infections. Time of infection, however, appears to be governed more by the timing of vector activity and time of sowing than by virus/plant interactions.

Changes in infectious virus concentration are likely to influence epidemics. Infectious virus titer could certainly alter the probability of a vector acquiring and then transmitting a virus isolate. However, the importance of this factor in the progress of disease, compared with the many others that regulate epidemics, is presumably quite small. This is because, as long as a minimum titer is present and the vector feeds for an adequate length of time, the probability of successful acquisition and transmission of that isolate is high, particularly with efficient vectors (A. D. Hewings & C. E. Eastman, personal communication). Thus, feeding, movement, and other vector-related behavioral activities appear to be of greater importance in generating epidemics than are the intricate virus/host interrelationships.

Luteoviruses have a high degree of vector specificity. Data suggest that virus-recognizing receptors located on cell membranes of the salivary glands may determine which luteoviruses can be transmitted by which aphid species. Because of the intimate association of luteoviruses with aphid tissues, these viruses are totally dependent on aphid behavior for survival and spread (36).

The Status of BYDV Strain Designation

Based on serology, cytopathology (43), and dsRNA "fingerprints" (37), BYDV strains can be separated into two groups: Group 1 contains PAV, MAV, and SGV variants, and Group 2 contains RPV and RMV variants.

Because of the chemical and genetic integrity of these two groups, formal designation as distinct luteoviruses appears valid and fundamental to future investigations.

Other than these two distinct luteoviruses, the validity of the classification of the current strains, based on vector specificity, is equivocal. Apart from the four aphid species mentioned above, at least 19 additional species can transmit one or more isolates of BYDV (1). When more vector species are tested against the innumerable BYDV isolates around the world, what will be the impact on the current classification of BYDV strains? Based on the current criterion used for strain designation, i.e. vector relationships, we postulate that as new isolates are characterized, their positions in the current nomenclature will become ambiguous; thus separation of strains will become nebulous and may require restructuring.

Already, current strain designations seem to be breaking down. The type isolate of PAV, described from New York State, is not transmitted by *R. maidis* (93), whereas variants of the same strain in parts of Europe and the Mediterranean appear to be transmitted by that aphid species (70), although perhaps by different genotypes. The type isolate of RPV, also described from New York, is transmitted specifically by *R. padi* (93), whereas a variant strain from California was found recently to be transmitted nonspecifically by two additional aphid species, *S. avenae* and *S. graminum* (26). Not only is the classification of BYDV breaking down because of specific variant interactions with specific vector species, it is also apparently being altered when variants of different strains occur together. One variant of a vector-specific strain can be transmitted by an additional aphid species if an appropriate variant of a companion strain is also present in the host plant, a phenomenon caused by genomic masking or perhaps phenotypic mixing. Thus, *R. padi* transmits most variants of RPV specifically and PAV nonspecifically, but it can transmit variants of RMV, MAV, and SGV in the presence of RPV variants, and occasionally with PAV variants. Several such examples have been reported (94, 96). Indeed, many isolates may have evolved quite recently and have not yet diverged much. This makes assigning some of the variants into discrete strain groups extremely difficult and currently inappropriate.

BYDV Detection in the Field

BYDV is restricted to phloem tissues, and overall it occurs in very low concentrations in plants, even though the concentration in phloem may be extremely high. Virus symptoms are often difficult to detect in the field. For example, a survey of grasses in Scotland established the prevalence of symptomless BYDV infections of PAV, RPV, and MAV variants in ryegrass (50). The use of enzyme-linked immunosorbent assays (ELISA) is an extremely efficient means of detecting the virus and has been important in

confirming the presence and abundance not only of BYDV, but of its various designated strains in the field. ELISA, using polyclonal antisera, is currently the preferred technique for detecting BYDV and its various strains (68), even though somewhat costly and labor intensive (it is inexpensive once the procedure is established). Quite recently, production of monoclonal antibodies and cDNA probes of selected BYDV strains are providing more sensitive methods of detection (28, 77, 78), although these newer techniques are even more labor intensive and costly. Accurate, timely, and cost-effective identification and characterization of the viruses and virus variants involved in the barley yellow dwarf pathosystem are fundamental to the understanding and study of epidemics on both local and regional scales.

COMPLEXITIES OF THE PATHOSYSTEM

Understanding how the biotic elements of the barley yellow dwarf pathosystem interact is difficult because of the intricate and multiple nature of the associations. This is particularly so when the environment is treated as a series of factors that govern how and at what rate the biotic elements interact.

BYDV Alters Plant Biology

BYDV greatly influences the growth and metabolism of its hosts. Depending on the BYDV strain and its virulence, infection may contribute to winter kill in cold, temperate regions; induce plant stunting; inhibit root growth; reduce or completely prevent flower production; or increase the host susceptibility to opportunistic pathogens, drought, and other stresses (13).

Production of autumn-sown cereals in temperate climates is severely affected by winter stresses that interact with BYDV. Most winter cereals are more resistant to BYDV than are those that are sown in the spring, but BYDV, when present, contributes substantially to winter kill (22). Under controlled environmental studies, Paliwal & Andrews (84) found that BYDV infection reduces the tolerance of oats and barley to low temperatures most severely and wheat less so, but has no effect on survival of rye, although rye can sustain high virus concentrations. It may be significant that infected ryegrass produces more total tillers and a higher ratio of vegetative to fertile tillers than healthy plants (17). BYDV causes a rise in the critical threshold temperature at which 50% of the plants are killed, of 4–8°C in barley and 2–4°C in wheat. This is extremely important considering that a change of only 0.5°C can significantly affect long-term field survival of these crop plants (84). BYDV infection reduces wheat cold hardiness by about 3.5°C; it also reduces ice tolerance during early low-temperature growth but increases it after 4 months at low temperatures (5).

Delserone et al (27) found that feeding by noninfective aphids on winter

barley cv. Pennrad neither decreases top and root growth nor increases crown injury nearly as much as does feeding by infective aphids. This is also true for wheat and oats, leading A. Comeau (personal communication) to postulate that in eastern Canada, noninfective aphids cause relatively little damage to cereals. Because BYDV reduces root growth more than it does shoot growth, its debilitating effects may not be obvious (18). During drought-ridden summers and without irrigation, plants infected with BYDV may not acquire sufficient water and nutrients to sustain growth and yield because of the impaired root structure. Thus BYDV infection can have disastrous consequences on cereal production in drought years.

Overall plant "fitness" can also be affected by the interaction of BYDV and other pathogens. Sward (105) and Sward & Kollmorgen (106) found that BYDV and the take-all fungus, *Gaeumannomyces graminus* var. *tritici,* for instance, each reduce grain yield and increase the number of "deadheads" in wheat in Australia; the combined effect of BYDV and take-all fungus appears greater, however, than a simple additive effect of each pathogen. Similarly, the losses due to leaf blotch, caused by *Septoria avenae,* on BYDV-infected oats, are twice those of oats infected with *Septoria* alone; Comeau & Pelletier (23) concluded from their studies that BYDV predisposes oat plants to damage by *S. avenae.* Furthermore, the ability of root pathogens to induce premature ripening is, according to Price & Stubbs (91), enhanced in BYDV-infected wheat plants, suggesting that BYDV predisposes wheat to root diseases (A. Comeau, personal communication).

Other evidence suggests that plant fitness is not always decreased by BYDV interacting with other pathogens. Although in some cases BYDV initially inhibits the expression of *Erysiphe graminis* (powdery mildew), Potter & Jones (90) concluded that, on comparing BYDV-infected with virus-free plants, the effect of powdery mildew was not ultimately different.

Host Plants Interact with the Environment to Alter Vector Biology

Plant species and cultivar, and the location of specific feeding sites, influence vector fecundity (33, 65, 66). In field and greenhouse tests, the number of *R. padi* alatae has been correlated positively with plant size and density (2). Plant growth stage also has a significant effect on aphid fecundity. Kieckhefer & Gellner (60) tested *S. graminum, S. avenae, R. padi,* and *R. maidis* under growth-chamber conditions for fecundity on several hosts at differing growth stages. They reported that *R. padi* and *S. graminum* had higher rates of reproduction on headed spring wheat than at earlier stages of growth, and that *R. padi* and *R. maidis* colonized older maize plants rather than the seedlings, possibly because of the initial protective effect of DIMBOA (2,4-dihydroxy-7-methoxy-1,4-benzoxazine-3-one) (62). Moreover, *S. graminum,*

R. padi, and *R. maidis* preferred young sorghum to older plants; the growth stage of barley affected fecundity of *R. maidis* but not of *R. padi*, *S. avenae*, or *S. graminum;* and no differences in fecundity of *S. graminum*, *R. padi*, or *S. avenae* were found between aphids placed on vernalized and nonvernalized winter wheat. *S. avenae* prefers young heading stages of wheat to ripening plants (113, 114).

In Australia, climatic factors affect the distribution of *R. maidis*, *Sitobion* spp., *M. dirhodum*, *R. padi*, and *Rhopalosiphum rufiabdominalis* directly by circumscribing the physical habitat, as well as indirectly by influencing the composition of the local flora. Johnstone et al (56) contended that different grass species vary in their susceptibility to BYDV variants and in the likelihood of being colonized by different vector species. It was also observed that variants of BYDV that are vector-specific tend to be common in regions where one vector species predominates, whereas vector-nonspecific variants appear to be most common, together with mixed infections, in areas where more than one vector species is prevalent.

BYDV Alters Vector Biology

Luteoviruses affect aphid biology, including feeding efficiency, morphology, reproduction, and the production of alates (36). Miller & Coon (76) found that viruliferous aphids had an increased developmental rate, longevity, and reproductive period, and produced more progeny and consumed 13.8% less oxygen than did nonviruliferous aphids. Araya & Foster (6) demonstrated that longevity decreased when *R. padi* fed on BYDV-infected rather than uninfected wheat. Nonetheless, total reproductive capacity appeared to increase when *R. padi* fed on virus-infected wheat, but not on virus-infected oats (6). As studied by Montllor & Gildow (79) using an electronic monitoring system, *S. graminum* fed better when oats were infected with an RPV variant of BYDV; however, *R. padi* seemed to feed equally well on infected and uninfected oats. According to Fereres et al (32), *S. avenae* had a shorter developmental time, a greater fecundity, and a greater intrinsic rate of natural increase when feeding on BYDV-infected wheat than when feeding on uninfected plants of the same cultivar. These studies suggest that there is some mutualistic interaction between BYDV and its vectors.

Gildow (34) showed that a consistently higher percentage of winged progeny is produced on oats infected with BYDV than on uninfected plants, regardless of aphid species, morphology of parent aphid, or the BYDV isolate used. The final adult morph of an aphid could be regulated by placing the first instar nymphs on BYDV-infected plants for a short time. When *S. avenae* and *R. padi* were reared on BYDV-infected plants, a far higher percentage of the eclosing adults were alatae (winged aphids) than with similar rearings on uninfected plants; this proved true for field-collected aphids as well as for

those from laboratory colonies (34). Later, Montllor & Gildow (79) discovered that although the proportion of *R. padi* that developed into alatae on BYDV-infected oats is greater than on healthy oats, *S. graminum* shows no such response. BYDV infection was found to increase total amino acid content of leaves at three growth stages of spring wheat; the effect was greatest in the earlier stages, but alanine and glutamine were always more abundant in infected than in healthy leaves (3). Senescing oat leaves also induce alate production; thus Gildow (34) postulated that changed nitrogen metabolism, resulting in increased amino acid concentrations in diseased or senescing plants, could trigger alate production. This shift in winged-morph production suggests that aphids from BYDV-infected plants are more prone to disperse (35), enhancing the potential increase in the rate of disease progress.

Coon (25) demonstrated that an increase in amino acid concentration accelerates alate production in *Rhopalosiphum insertum* on oats supplied with nitrogen fertilizer and also significantly increases progeny production. Markkula & Laurema (71) showed that reproduction of *R. padi* increases with the greater concentrations of free amino acids associated with BYDV-infection in oats, but reproduction is unaffected for *S. avenae* and *M. dirhodum*. Therefore, it was argued that changes in free amino acids alone cannot explain all of the changes in aphid reproduction.

Cereal cultivars, bred for their high yielding capacity, require high levels of fertilizer inputs. Because added nitrogen often increases aphid fecundity and alate production, it might well prove to be a key component in the explosive increase in populations of cereal aphids and BYDV, worldwide (8).

The Evolution of BYDV Strains

Because most BYDV variants infect many graminaceous species, relationships involving the BYDV viruses and individual host species do not appear to be significant factors in the evolution of this pathosystem. The abundances and activities of the predominant vector species in particular seem to be of far greater importance. Indeed, the selection of BYDV isolates and their evolution toward dominance locally or regionally may well be dependent upon the types, abundances, and activity patterns of vector species that occur in that area or that regularly migrate there from elsewhere, especially when considered in relation to the timing and scale of local cropping patterns. Thus the vectors appear to determine the rates and direction of the evolution within the BYDV complex on micro- and perhaps macro-regional scales.

RESOLVING THE FACTORS THAT DRIVE EPIDEMICS

Certain key aspects of ecology must be resolved if epidemics of barley yellow dwarf are to be managed. Three issues of crucial importance are the primary

inoculum sources, how BYDV variants and their vectors survive unfavorable periods, and how the vectors move and disseminate virus.

Primary Inoculum Sources

BYDV is neither mechanically nor seed-transmitted. Although infective aphids can retain the ability to transmit the virus after moulting and throughout their lifespans, there is no "vertical" transmission to the progeny. The current view is that aphids carry the virus into a newly sown field from some other existing host plant of the same or a different species that harbors the virus. Distinct variants, in fact, could be carried by vectors to a field from different sources. Therefore, an epidemic must begin by spread from one or more virus reservoirs after a crop is sown. The primary inoculum sources may be local, regional, or distant (52). It is important to realize that a plant, wild or otherwise, may harbor the virus without contributing to further spread. For spread to occur, a vector must move the virus from the reservoir to other hosts.

From surveys conducted in parts of the United States and Europe, overwintering reservoirs of BYDV in wild grasses near cereal fields seem not to contribute substantially to barley yellow dwarf epidemics in adjacent crops. This is inferred because the predominant strain variant constituting the epidemic in the cereal crop often differs from that constituting the majority of infections in the wild, perennial grasses nearby. For example, grasses are a perennial source of BYDV in England, but the isolates from cereals often differ in their geographical distribution from those of grasses (86). Up to 50% of grasses surveyed in Indiana contained PAV, MAV, RPV, or some combination of these strain variants; whereas, nearby cereals contained a preponderance of only PAV variants, suggesting that nearby wild grasses may not be the most important source of the virus for spread to cereals in the Midwest (31).

In Canada, overwintering *R. padi*, emerging from *Prunus padus* trees in the spring, were not viruliferous until they had fed on infected plants (103). Thus, immigrating *R. padi* derived from eggs cannot initiate epidemics unless they first spend some time feeding on BYDV-infected plants.

PAV variants commonly infect wild graminaceous plants in Spain (58). Nevertheless, contrary to the situation in midwestern North America, autumn and winter infections of BYDV in cereals in Spain were found to be predominantly RPV variants (80). PAV variants were most prevalent during a survey of several countries of North Africa and West Asia during the 1985/1986 season, although, based on vector-specific assays, RMV and RPV variants also occurred in that region (70). These facts strongly caution against assuming that local reservoirs invariably serve as primary inoculum sources for epidemics in nearby cereal crops.

Vector biology, operating in the context of the environment, is the overriding factor in determining the effectiveness of primary inoculum sources. Plumb (86) suggested that aphid biology, weather, and host availability determine which and when BYDV isolates are spread from grasses to cereals.

The temporal juxtaposition of crop phenology and patterns of aphid activity also influence which of the virus reservoirs serve as initial inoculum sources. For instance, *S. avenae* tends to be associated with the first BYDV infections of spring-sown cereals in Canada because *R. padi* populations decline before these crops are planted (103). McGrath et al (73) and McGrath & Bale (72) also implicated *S. avenae* as the primary vector of BYDV in winter barley in northern England. Thus, under those circumstances, BYDV is carried from reservoirs to crops by *S. avenae* and not by *R. padi*.

The Temporal Gap

In most areas where grass or cereal hosts of BYDV are grown, some climatic or other limitation prohibits continuous presence of those crops and, hence, the continuous spread of the virus. The limitations can be very cold winters in temperate regions or desolate, dry seasons in semitropical ones, both of which can restrict the survival of viable, virus-infected host plants and, consequently, their virus variants and aphid vectors. Any climatic regime that tends to break the crop sequence with a rather wide temporal gap can impose rigorous barriers to the continuity of BYDV epidemics. There must be survival of virus variants and vectors in order to span these imposed temporal barriers.

In climates with harsh winters, winter and early spring cereals are usually sown in the autumn. This allows autumn migrants to introduce virus to young crops before conditions deteriorate and growth is interrupted. The plants resume growth in the spring and serve as excellent reservoirs to initiate spring epidemics. A high incidence of BYDV in overwintering wheat and rye, for instance, suggested that winter cereal reservoirs of BYDV are common in Canada (103). In 1982 and 1983, winter wheat and barley were heavily infected, predominantly with a PAV variant of BYDV, but RPV and RMV variants also occurred (85).

Mild winters in temperate zones enable some vectors, as well as BYDV, to overseason in cereal crops. For example, *R. padi* overwinters anholocyclically in great abundance on graminaceous plants in southern England, an area where winters are usually mild, but *Rhopalosiphum insertum* and *R. maidis* did not appear to do so on these hosts in the area (48). According to Milinko & Nagy (75), a mild winter often allows vectors to persist in cereal crops of central Europe. In spring, this often results in severe barley yellow dwarf epidemics.

In the subtropical, dry climates of the Mediterranean region, there are

quite different means of perennation. In these situations, summer drought appears to be the most important barrier to the carryover of BYDV and its vectors between successive cereal crops (89). Wild grasses play an important role in the overseasoning ecology of BYDV in many parts of the world where there have been epidemiological studies. In rainfed areas of West Asia and North Africa, wild grasses and graminaceous crops that survive the hot, dry summers are few and are probably not significant primary inoculum sources for producing barley yellow dwarf epidemics in autumn-sown cereals. Perennial wild grasses in the more moist, cooler highlands may serve as primary inoculum sources during the summer drought in the lowlands, but this supposition should be substantiated through rigorous experimentation.

Major bridging crops such as maize also appear to play an important role in the carryover of BYDV through summer drought in the Mediterranean zone of southern Europe (89). In irrigated areas of Italy, maize, one of the crops most frequently colonized in summer by *R. padi*, appears to play a decisive role as a virus inoculum source; about 9% of the aphids that colonize that crop were infective with BYDV (19). Similarly, Refatti et al (92) found that 0.5 to 7.0% of the apterous *R. padi* randomly collected from maize in five localities in northern Italy transmitted BYDV. In the laboratory, *R. padi* readily transmitted a PAV variant of BYDV from maize to maize and from maize to oats (82). Coceano & Peressini (19) thus postulated that movement of *R. padi* from maize to barley or wheat could have a great influence on barley yellow dwarf epidemics in those cereal crops.

Knowledge of the role of maize and sorghum in the ecology of BYDV in North Africa and West Asia is lacking. However, Makkouk et al (70) suggested that the presence of maize, found to be infected by a PAV variant in Syria and Tunisia, and sorghum, found to be infected with BYDV in Tunisia, may be summer hosts. Thus, there is mounting evidence to implicate locally grown irrigated maize and sorghum as primary inoculum sources for autumn-sown crops of winter cereals.

The importance of irrigated maize as a bridging host is not confined to semitropical regions. In a survey of winter wheat in Washington State, 20% of samples were infected with BYDV, and irrigated areas where maize is grown that supported aphid vectors during the summer, along with those fields that were planted early in the autumn, had the highest incidences of BYDV (115). In eastern Washington, winter grain crops become infected with BYDV soon after seedlings emerge in the autumn. Brown et al (12) identified irrigated maize as a reservoir of both BYDV and its aphid vectors during the period between summer harvest and autumn sowing of winter cereals. BYDV was recovered from 58–65% of the maize fields surveyed, and from all cultivars, hybrids, and lines of maize tested. The isolates of BYDV from maize in eastern Washington appeared to be PAV variants (12),

whereas RMV variants predominated in maize during the barley yellow dwarf epidemic of 1981 in eastern Canada (83). In eastern Washington, infective *S. avenae* occurred in maize fields in June and July, while *R. padi* infested maize heavily in July, August, and September, and more than 60% of the individuals were infective (12).

Halbert et al (46) cautioned that the ability to predict epidemics of barley yellow dwarf would depend upon the ability to measure vector flight intensity and primary inoculum pressure, reasserting Kennedy's (59) dictum that vector activity in a crop is far more important than sheer numbers. Percentage transmission by *R. padi* collected from small grains was similar to that measured for this aphid from maize. The rate of transmission by *R. padi* from suction traps was higher than that by aphids collected in either crop. They concluded that a measure of the inoculum reservoir in maize might be a good predictor of primary inoculum in cereals in irrigated areas of the Pacific Northwest.

Recent evidence suggests that, while *R. padi* may play a prominent role in transmitting BYDV from maize to winter wheat in the Pacific Northwest, *R. maidis* may not. Blackman et al (11) found that samples of *R. maidis* from maize in Idaho were all 2n = 8 karyotypes whereas those from barley and *Echinochloa crus-galli* (barnyard grass) were all 2n = 10 karyotypes; and those from wheat were mainly 2n = 10, but the wheat contained some samples with 2n = 9 or 2n = 8. Because these karyotypes of *R. maidis* seem to discriminate between crops, Blackman et al (11) suggested it is unlikely that the maize karyotype would transmit BYDV to cereals or that the cereal karyotypes would transmit the virus to maize. Because of their findings, Blackman et al (11) postulated that barnyard grass may be a more important primary source than maize isolates of BYDV transmitted by *R. maidis* to cereals in North America's Pacific Northwest.

The situation with karyotypes of *R. maidis* in the Pacific Northwest may not occur in all geographical regions. Approximately 30% of the *R. maidis* tested under laboratory conditions in Syria could transmit BYDV, a far greater number than the 2.4% infection rate reported in similar North American tests (70), even though the four distinct "biotypes" of *R. maidis* differed in their abilities to transmit a single isolate of BYDV (99). Therefore, *R. maidis* may play a prominent role in BYDV epidemics in North Africa and West Asia where cereal crops are followed and preceded by maize (70), whereas in North America this species may be insignificant. This hypothesis must be tested by first determining the karyotypes of *R. maidis* on crops in the Mediterranean Region and then by following the movement of selected populations to determine if they disperse and so carry virus between maize and cereals.

Vector Movement

The movement of vectors is responsible for barley yellow dwarf epidemics, influences spatial and temporal patterns of infection in fields, and determines which fields become infected. How aphids respond to environmental perturbations and to physiologically induced cues, to a large extent regulates how, when, and how far they move. This is a crucially important and yet often neglected aspect of barley yellow dwarf epidemiology.

An alate, BYDV-infective vector landing on a susceptible crop plant can, if it feeds for a sufficient period, infect the plant and so initiate a new virus focus. If the alate reproduces, its apterous offspring can walk short distances to neighboring plants, transmitting the virus and thus enlarging the focus. At any time during this sequence, infective alates can fly to other plants in the field, potentially initiating new foci, or they fly out of the field to initiate new foci elsewhere (Figure 2). An alate emigrating from the field can move to a neighboring field or to a more distant field, depending on the physiological status of the aphid in relation to flight activity and on the meteorological conditions at the time of emigration. These four types of virus spread (enlarging existing foci and developing new foci within the same field, in nearby fields, or in distant fields) must be clearly distinguished because each leads to a very different pattern of spread and requires a very different management strategy. The term "secondary spread," could be used for any of the four modes mentioned above, but it is not used here in this context because the term is ambiguous and potentially misleading.

The four modes of virus spread generally correspond with types of vector movement: walking, short or moderately long host-seeking flights, and long-distance migratory flights. While any vector movement may lead to virus spread, this is not necessarily so because a vector may not be infective or may not land, acquire, or transmit. Thus, a vector might migrate far, but spread BYDV only locally.

Aphids that are present in a field can acquire BYDV in four ways: if they are born and develop on an infected plant, if they walk onto and colonize an infected plant and establish on it, if an infective aphid transmits the virus to the plant on which they occur, or if they fly to and establish (or at least feed for a considerable length of time) on an infected plant. Thus, identical aphid colonies can be in close proximity, one producing offspring that, by virtue of the plant on which they are born, become viruliferous, the other producing offspring that are likely to remain nonviruliferous. Only when the virus incidence in the field becomes high and when there is substantial movement of aphids is it likely that an aphid, not born on an infected plant, will become viruliferous. Moreover, a viruliferous aphid is not necessarily infective; to become infective, the virus isolate in the vector must be compatible with the

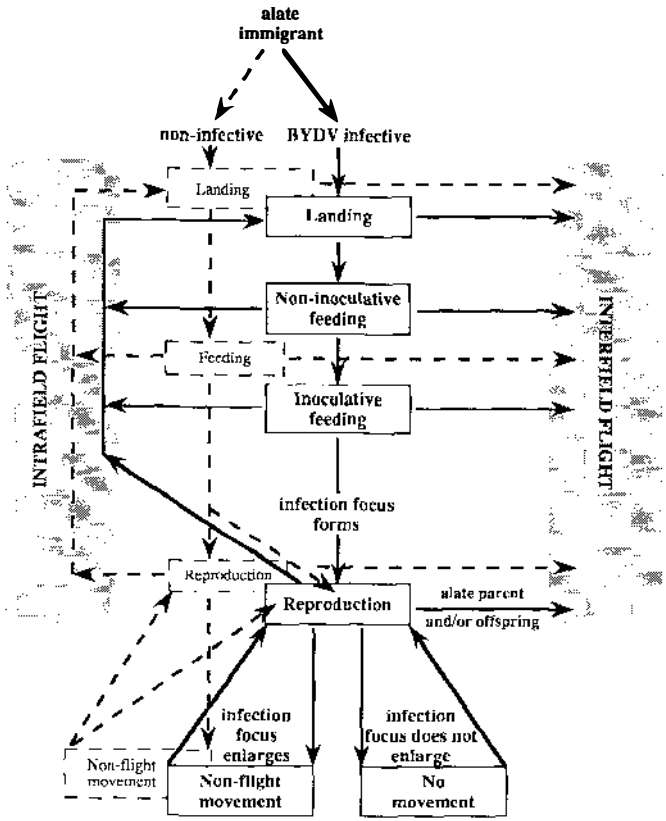

Figure 2 Flow diagram of BYDV spread, including the inter- and intrafield components of epidemics. This diagram highlights the differences in the intrafield spread by separating the initiation of new foci from the enlargement of existing foci.

genetic makeup of that aphid species. Therefore, an aphid enters the virus cycle only after overcoming many ecological, behavioral, physiological, and genetic barriers. This implies that there are many avenues for intervention in the virus cycle.

ENLARGING EXISTING BARLEY YELLOW DWARF FOCI Enlargement of barley yellow dwarf foci can be due to infective apterae or alatae walking between plants. It can also be caused by alatae flying from an infected plant to a nearby uninfected one, although from our observations, this option appears rather remote. In commercial cereal crops, plant densities are very great and flight from one plant to a near neighbor is no further than two centimeters, and

often closer. Presumably, the energy required for an alate to take off and fly such distances is far greater than the energy needed to walk, particularly when the canopy is interconnected. Moreover, our observations suggest that when an aphid takes flight, it traverses several centimeters before it responds to alighting stimuli. Thus, the minimal flight distance would take the alate aphid at least tens of centimeters away from the source plant, unless it chooses to circle back.

Several research findings affirm the role of aphids walking within cereal crops as a means of spreading BYDV to neighboring plants. According to Conti et al (24), periods of mild weather stimulate aphid movement in cereal crops in Italy during the winter, leading to enlargement of initial barley yellow dwarf foci. A similar phenomenon has been observed in winter wheat and barley fields in central Washington State. Halbert & Pike (47) determined that between 3.4 and 14.5% of the alate aphids collected from winter cereals during the autumn migration transmit BYDV. In November, after peak aphid flights had occurred for the year, an increase in the proportion of infected plants suggested active intrafield spread. Pitfall-trap collections demonstrated active walking by aphids, and trap plants became infected, confirming that virus spread was occurring. Post-migration surveys of apterae and nymphs established that the numbers of infective apterae correlate well with concurrent increases in spread of BYDV (47). Spread during the winter in the form of enlarged BYDV foci can occur only in areas where aphids overseason parthenogenetically.

Even though existing BYDV foci can expand in winter during mild periods, it is mainly a spring and summer phenomenon. Comeau & Dubuc (21) noted that enlargement of existing foci is a major factor leading to a high incidence of BYDV and, therefore, to epidemics in summer cereal crops in Quebec, Canada. In Australia, after initiation of cereal stem elongation, increases in virus incidence are thought to be due almost exclusively to local movement by apterae among plants (56).

Why do nymphs, apterae, and even alatae leave a host plant? They appear to leave because of stimuli from their host plants, "signaling" that the plant is stressed. The physiological status of an alate depends largely on prior environmental conditioning during its lifetime and on the conditioning of its parent. Plant-induced stress is a major factor influencing aphids. Thus, it seems that the physiological status of the host plant can trigger movement, especially as it influences the aphid's own physiological condition. Much spread of BYDV occurs when apterae leave overcrowded plants for nearby hosts. Orlob (81) noted that *S. graminum* multiplied more rapidly when it was attended by ants than when unattended, thus leading to earlier overcrowding and quicker exodus. Ant-mediated enlargement of barley yellow dwarf foci is, according to Orlob (81), typically confined to field edges.

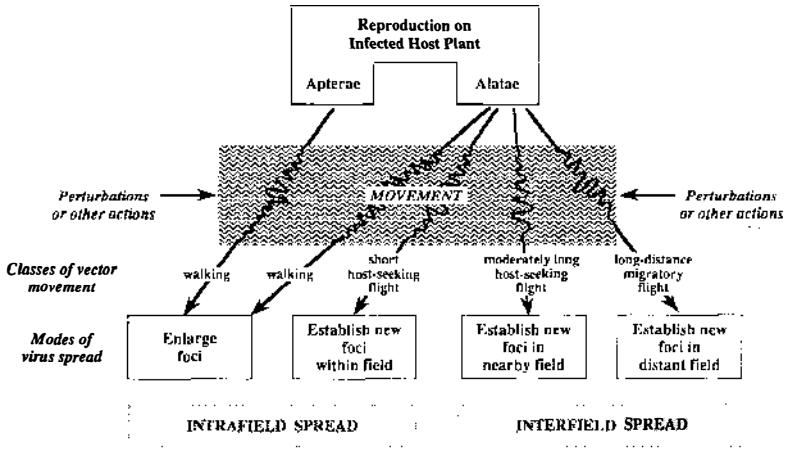

Figure 3 Conceptual diagram of aphid movement resulting from various perturbations to the system. These movements can result in spreading BYDV to other fields, or establishing new or enlarging existing barley yellow dwarf foci within the field.

Environmental disturbances can dislodge aphids or cause them to colonize new plants (Figure 3). These perturbations can be very subtle, such as wind swaying grass stems on which aphids occur, or they can be more overt, such as predators causing the aphids to emit an alarm pheromone, dislodge, and disperse. These perturbations are probably multifarious in a cereal field, but how they might influence aphid movement is only apparent from studies by Roitberg et al (98). They determined the influence of dense populations of a predator in a legume field colonized by an alarm pheromone-producing aphid, *Acyrthosiphon pisum*. The resulting enlargement of an infestation focus was greater at high than at low densities of the predator even though the numbers of aphids may have been decreased by predation.

DEVELOPING NEW FOCI WITHIN THE SAME FIELD, IN NEARBY FIELDS, OR IN DISTANT FIELDS Whether a new virus focus in a cereal field is initiated from an infective alate that developed in that field, or by an alate that acquired the virus in the field, or whether the infective aphid immigrated from a neighboring field or from afar has, to date, been almost impossible to establish with any degree of confidence. The inability to resolve this issue has impeded an understanding of barley yellow dwarf epidemiology, and has contributed to the limited success in controlling the disease.

Flight energetics studies, sometimes referred to as "lipid depletion" or "lipid utilization," have begun to establish procedures for determining the length of time an aphid has flown (20, 67). This type of research, coupled

with studies on vector flight dynamics (52), may eventually make it possible to discriminate among the different types of vector flight. This research is, however, only just beginning, and it is currently impossible to determine accurately the duration of vector flight.

There are divergent views regarding classes of vector flight and corresponding types of spread of BYDV in cereal crops. These may be explained by the fact that studies have been conducted in very different regions. Several scientists believe that BYDV spreads mainly on a local scale. According to Tatchell et al (108), in England, nonsexual alates introduce BYDV from comparatively local sources. Moreover, Johnstone et al (56) suggested that most initial BYDV foci in cereal fields in Australia are caused by aphids flying short distances from reservoirs of infection in nearby grasses. This view was supported by Plumb (89) who found little evidence of long-distance transport of aphids in Europe.

By contrast, several scientists have attributed local epidemics to long-distance migration. Paliwal (83) argued that local reservoirs such as grasses, winter wheat, and maize were unimportant as virus sources for eastern Canada's cereal crops during the 1981 barley yellow dwarf epidemic; virus inoculum introduced by aphids from elsewhere was considered to be the main source of infection. Similarly, Canada's 1983 epidemic of barley yellow dwarf was attributed to a large aphid migration into autumn-sown crops in October 1982 (85). Moriones et al (80) in Spain determined that the more prevalent PAV variant in cereals is associated with high late-spring populations of *S. avenae* and *M. dirhodum* that, they postulated, migrate and introduce virus from distant areas. Conti et al (24) also concluded that migrating aphids bring BYDV into autumn-sown Italian cereals. They further determined that the infectivity of these incoming migrants is relatively low, and initial foci are generally scattered and sparse. Elsewhere, some evidence suggests that *S. avenae* moves considerable distances (69), as into Scandinavia from mainland Europe in some years, causing outbreaks of an MAV variant in areas where variants transmitted by *R. padi* usually predominate (89).

Paliwal & Comeau (85) found little evidence of BYDV movement from autumn-sown winter cereals to spring-sown grains during 1983 in eastern Canada. This might be explained by the observations of Slykhuis et al (103) that populations of *R. padi* decline before Canadian cereals are sown in the spring; thus populations of *S. avenae*, which may have been scarce during 1983, appear to be associated with the first BYDV infections in spring-sown cereals.

In England, a greater number of alatae of *R. padi* are caught in suction traps at a height of 12.2 m than at 1.5 m during the autumn, whereas the reverse occurs in the spring and summer. This suggested to Tatchell et al (108) that

alatae are moving out of the fields during the autumn and into the fields during the spring and summer. That aphids emigrate during crop maturation was substantiated by Milinko & Nagy (75), who determined that when Hungarian cereal crops begin to ripen in June, aphids migrate to fields of immature maize and to volunteer cereal plants, which are the most important summer-hosts of BYDV vectors in that country (75). There exists, therefore, a supposition that emigration may be rather local in scope, but this is far from proven. Although Taylor (109) provided an eloquent, fact-filled chapter detailing aphid migration and virus spread, the fact remains that, while aphids can migrate hundreds and sometimes thousands of kilometers—a point discussed by Hendrie et al (49), Johnson (54, 55), Thresh (110), and Irwin & Thresh (52)—only circumstantial evidence links these aphid migrations to long-distance transport of BYDV.

Another long-standing controversy surrounding barley yellow dwarf epidemiology concerns the possible attractiveness of BYDV-infected foliage to vectors. Some authors assert that yellowing caused by BYDV leads to increased landing by vectors, which, in turn, could promote epidemics in cereals. It is difficult in field experiments to separate the effects of foliage color from the stunting associated with infection and the consequent change in apparency and ground cover. In England, BYDV-infected plants support significantly higher numbers of *S. avenae* and *M. dirhodum* than do uninfected plants. Ajayi & Dewar (4) attributed this to the attractiveness of the yellow infected plants. In flight-chamber experiments using alates of both species, Ajayi & Dewar (4) confirmed that more specimens are attracted to BYDV-infected leaves than to healthy ones. Interestingly, Kieckhefer et al (61) earlier found that *S. graminum, S. avenae,* and *R. padi* preferred to settle on healthy green leaves rather than BYDV-infected ones. This agrees with our observations in soybean fields that more individuals of several aphid species tend to land on healthy, dark-green soybeans than on an isoline that is deficient in chlorophyll and pallid (51). The larger number of aphids reported by Ajayi & Dewar (4) could be explained by population increases and the greater numbers of alates on infected plants, but this does not explain the results of their flight chamber experiments. Thus, the relative attractiveness of diseased and healthy cereal plants remains an unresolved question of considerable importance.

CAN BARLEY YELLOW DWARF EPIDEMICS BE MANAGED?

Three broad tactical spheres have been investigated in attempts to control barley yellow dwarf: breeding for host plant resistance mainly to the virus but also to the vector, applying chemical pesticides to reduce vector populations,

and manipulating the crop environment to minimize or retard epidemics. Each approach has achieved some success that may have direct application for managing epidemics of barley yellow dwarf on local, regional, or global scales.

Breeding for Host Plant Resistance

Breeding for resistance to the virus has long been considered for control of barley yellow dwarf. Nevertheless, Larkin et al (64) admitted that few sources of BYDV resistance have been discovered, at least within the Triticeae. The known sources include the Yd_3 and Yd_2 genes, and resistance from the wheatgrass *Thinopyrum intermedium* (*Thinopyrum* is a senior synonym of *Agropyron*). In the latter case, the resistance to BYDV infection of several *Thinopyrum* species is usually due to the failure of the virus to replicate, but it can also be caused by the inability of the vector to locate phloem cells (100). The Yd_2 gene, transferred from Ethiopian land races, confers a degree of resistance to BYDV in oats, manifest by mild symptoms and limited replication, but this gene seems to be linked with undesirable agronomic characteristics such as lodging (22). McGuire & Qualset (74) have successfully transferred the Yd_2 gene from barley cultivars to a Chinese spring wheat *(Triticum aestivum)*. Apparently, the undesirable agronomic characteristics associated with the Yd_2 gene can be overcome in some instances (22).

Some BYDV tolerance has been described in wheat, but, according to Larkin et al (64), no major resistance has been discovered in this cereal. Thus, genes conferring resistance or tolerance to BYDV appear rather limited, with some apparently effective only against certain variants or specific BYDV strains (64). Furthermore, after repeated attempts to transfer these genes to agronomically acceptable cultivars, there is relatively little to show for these efforts other than certain oat and barley cultivars and the potential for improved wheat cultivars.

New approaches may soon change this situation. Low virus multiplication or true resistance to BYDV, found in perennial grasses, appears to be transferrable to wheat using molecular techniques (44, 116), through the identification of a specific gene and through cytological and molecular hybridization studies. These studies have demonstrated that wheat var. Zhong 4 carries BYDV resistance on a set of seven pairs of non-wheat chromosomes derived from a combination of the E and X genomes found in *Thinopyrum intermedium*.

One important feature is evident concerning genetic resistance and tolerance to BYDV in cereals. While the Ethiopian Yd gene seems effective in many areas, at least against certain variants, resistance is not necessarily effective from one region to another (15). A line of durum wheat found to have a relatively high level of BYDV resistance or tolerance in Canada, did

not show the same degree of resistance when grown at ICARDA in Syria (A. Comeau, personal communication). Therefore, locality-specific aspects influence the effectiveness of the resistance because different BYDV variants and perhaps also different vectors are involved.

We are unaware of attempts to incorporate the vector-resistance characteristics of certain grasses into agronomically adapted cultivars of cereals, even though vector-tolerant and vector-resistant genotypes have been identified. For example, resistance has been detected to *Diuraphis noxia,* an aphid that colonizes cereals, although, in this case, a species that does not seem to be an important vector of BYDV (29). Tsumuki et al (111) suggested that surface wax on leaves, which is an inherited trait, is an important component of the resistance of barley to colonizing aphids, particularly *R. padi*. This was concluded from results indicating that resistance levels correlate positively with surface wax rather than with other traits such as leaf color. Because BYDV is spread predominantly by vector species that colonize cereals, and because there is a reasonable expectation that some, if not most, of the spread in certain fields is associated with vectors walking or making short, within-field flights, incorporation of vector-resistance genes into agronomically acceptable cultivars is a worthwhile goal. Breeding for tolerance to vectors, i.e. breeding for the ability of a host to sustain an infestation of vectors without the associated yield reductions, however, is unlikely to be appropriate.

Thus, within the tactical sphere of host plant resistance, two approaches appear to have high potential: attempting to incorporate BYDV-resistance genes into agronomically acceptable cereal cultivars, which may involve molecular engineering techniques, and attempting to incorporate vector-resistance genes into agronomically acceptable cereal cultivars, through methods of traditional breeding or molecular engineering.

Applying Chemical Pesticides

Insecticides are frequently used to reduce vector populations in cereal fields. Whether this action routinely and effectively reduces or retards epidemics of barley yellow dwarf in such fields is unclear. Because virus spread is due to vector movement, the importance of aphidicides in reducing or delaying epidemics depends largely on the type of vector movement occurring at and soon after the time of the application. If the vectors are mainly walking, i.e. enlarging existing virus foci, then the effectiveness of the application will depend upon the extent to which walking is decreased. Some insecticides, because they perturb colonized aphids, invoke rapid movement, usually walking, and thus may increase the rate of focus expansion. If the aphidicide acts rapidly and kills the aphids before they have time to move, foci enlargement should be both delayed and reduced. Aphidicides are often applied in

such a way that they do not effectively reduce populations of aphids because some of the species feed and reproduce in habitats that are difficult for the pesticide to reach or penetrate. For instance, some species are subterranean for parts of their life cycles, while others are protected within tightly coiled leaf whorls, and yet others occupy the undersides of lower leaves.

The timing of insecticide applications is as important as the type of pesticide used. In the temperate zones of Australia, climatic factors affect the distribution of *R. maidis, Sitobion* spp., and *M. dirhodum*. A single aphidicide applied to autumn-sown crops during the winter appeared to be beneficial where foci of infection and infestation occurred. The extent of virus infection in the spring seemed, therefore, to be related to the effectiveness and timing of aphid control the previous autumn (56). McGarth et al (73) confirmed the importance of timing of aphidicide applications in reducing barley yellow dwarf epidemics in England. They claimed that an application too early in the autumn allows reinfestation by aphids before the onset of winter, whereas intrafield infections had proliferated before late sprays were applied. The importance of aphidicides in controlling aphid reproduction and movement, and the critical importance of timing is evident.

Another important consideration is how aphidicides might alter the relationships between vectors and natural enemies, particularly regarding population dynamics and vector behavior. Yet another consideration is whether vectors become resistant to a specific chemical or to an entire class of chemicals. These interactions have immense repercussions to the system as a whole and to the control of BYDV, particularly when considered in the light of potential long-term management strategies that take cultural practices into account.

Certainly, more knowledge is needed concerning the effectiveness of pesticides in limiting vector movement, on biological control interactions, on pesticide resistance, and how each influences the subsequent spread of BYDV. A much greater understanding is needed to avoid the "blanket" insecticide spray used routinely by many farmers (50) and to avoid irreversible mistakes that could result from the untimely or improper use of these potent chemicals. This emphasizes the need for a better knowledge of vector movement, including which type, under which situations, and at what times aphidicides can effectively reduce or retard epidemics of barley yellow dwarf. Information is required on the interaction of natural enemies, not only with the vectors, but also with the vectors' abilities to spread the virus, and it should be appreciated that prolonged or routine use of chemical pesticides will engender vectors immune to their lethal effects. In short, chemical pesticides are powerful weapons in our arsenal for controlling epidemics of barley yellow dwarf, but they must be used carefully and wisely and as a last-ditch resort rather than a front-line defense.

Manipulating Crop Environments

Modifying cropping practices has long played an important role in managing virus-disease epidemics. Cropping practices can include alterations in sowing dates, crop rotations, crop spacing and density, sanitation procedures, and even regulatory measures that enforce regional sanitation practices or synchronization of crop phenologies and temporal gaps between growing seasons. Such tactics generally target vectors, attempting to manipulate their overseasoning habitat, movement, phenology, reproduction, and establishment to delay or reduce barley yellow dwarf epidemics. They can, however, also aim to reduce alternative virus reservoirs, especially through sanitary practices that eliminate weeds and volunteer cereals that persist between growing seasons.

Sowing date is perhaps the best example of how cropping practices can be altered to minimize barley yellow dwarf epidemics. Plumb (88) stressed that sowing date can be important for avoiding BYDV infection in England. By sowing autumn cereals after the major aphid flights, the amount of barley yellow dwarf can be reduced considerably. However, by delaying sowing in the autumn, potential yield is reduced. Similarly, later sowing of spring cereals will reduce potential incidence of barley yellow dwarf, but this practice also reduces potential yields because the crop has less time to reach maturity (88). Tatchell et al (108) observed that sexual alate forms of the vector species predominate during the autumn in England. They also noted that only asexual alates were trapped in summer and that they were more than eight times as infective as those, largely sexual, alates trapped during the autumn (74% compared with 9%). The sexual forms migrate to their primary hosts and do not contribute to BYDV spread in the autumn. Thus, autumn-sown cereals emerging before mid-September, prior to the transition of migrant aphids to a mostly sexual population, are exposed to colonization by nonsexual alates and to the associated greater risk of virus spread. Johnstone et al (56) argued that a judicious choice of sowing date for wheat in relation to the major autumn and spring peaks of aphid flight activity can reduce barley yellow dwarf epidemics in Australia. Jorda et al (57) in Spain monitored flights of *R. padi* into rice and concluded that delays in planting rice reduce epidemics of barley yellow dwarf in that crop.

Weeds in an area can influence how barley yellow dwarf epidemics are initiated and progress through time and space, clearly demonstrating the intricate nature of the interactions between vector species, virus complexes and their hosts within a changing environment (45). In New Zealand, barley yellow dwarf occurred most abundantly in cereal fields at the margin alongside grasses and around ryegrass clumps regenerating from the grass ley (104). In Tasmania, *R. padi* is the dominant vector of BYDV and colonizes different weeds to different extents. The species is responsible for inducing

different incidences of barley yellow dwarf in the crops of those areas (45). Moreover, different strains of BYDV were found to be prevalent in different weed hosts: a PAV variant on *Festuca* (fescue) and *Lolium* (ryegrass), and an RPV variant on *Dactylis* (cocksfoot) and *Phalaris* (canarygrass) (45).

Lapierre & Moreau (63) indicated from experiences in France that the risk of BYDV spread is enhanced by intensive rotations only when prophylactic, sanitation measures are lacking and when the total crop area is being increased. This suggests that intensive rotations, if accompanied by sanitation practices and under normal crop production allotments, could partially disrupt the epidemiological cycle.

Plumb (88) contended that, in England, the previous crop can influence the incidence of barley yellow dwarf in a field. If the previous crop was a potential host of BYDV, it is likely that the following cereal will have a greater virus incidence than if a nonhost had been grown. This is further evidence that intrafield movement of BYDV is prevalent and important for spring-sown cereals in England. It also suggests not only that crop rotation is important, but that which crops are grown can greatly influence the course of epidemics.

The incidence of disease is reportedly greater in oat fields at wide row spacing than at close spacings, when the fields are sown later rather than earlier, and where fields are near unsprayed apple and *Prunus padus* (102). These observations demonstrate the complex nature of the interactions that influence epidemics. Alate aphids of several species often alight preferentially on plants at wide spacing (51), leading to greater rates of spread. It is also suspected that, at wide spacings or decreased plant density, infective apterae and nymphs walking between plants reach fewer plants, so decreasing the amount of spread from existing foci. However, a low plant density also means that a greater proportion of the total stand will be infected by a similar number of infective immigrants entering the field, thus increasing the ratio of infected to uninfected plants. The observation that virus incidence is greater in later sown crops is probably a function of the timing of vector flights relative to the timing of crop phenology, as discussed previously. Apple orchards and associated ground vegetation, hedges, or windbreaks probably provide a sanctuary for natural enemies of vectors as well as a potential source of both vectors and virus. Under the circumstances reported by Slykhuis et al (102), relatively natural areas adjacent to cereals were evidently more important as reservoirs of virus or vectors than of natural enemies, but these aspects of the dynamics of virus-disease epidemics are little understood. This is borne out by Holmes (50) who argued that the current infectivity indexing scheme of Plumb (88) and colleagues at the Rothamsted Experimental Station, England, does not consider aphid population dynamics as influenced by aphid predators and diseases. Nearby trees of *Prunus padus,* a primary host of *R. padi,* could

provide an early spring source of vectors, which would enhance spread of BYDV. Moreover, such an area could also be associated with virus reservoirs and secondary hosts of the vectors, and these could further fuel epidemics. Thus, with complex systems as described by Slykhuis et al (102), rigorous experimentation is needed to identify the main epidemiological factors involved.

Management Strategy and Forecasting Epidemics

Epidemiological information is essential to develop truly effective control strategies for barley yellow dwarf (52). One essential ingredient is a knowledge of the type of vector movement during epidemics; without this, management tactics cannot be targeted on the weakest links of the epidemiological cycle. This information must be understood in the context of the influences it has on epidemics under different management systems. The key to good control is to integrate the various tactics into a cohesive strategy that ultimately reduces the impact of BYDV on crop yield, not only over the short term, but over successive seasons, while at the same time safeguarding the environment and wildlife.

An example of how many epidemiological factors are integrated to forecast barley yellow dwarf epidemics can be seen in France. Vector reservoirs, suction-trap catches, and field observations are considered, as are the location of alternative host plants in relation to the fields to be protected, the wind direction during aphid flights, and the percentage of total aphids that are viruliferous (9). Even this does not enable the system to be manipulated to retard or delay such epidemics. So far, little other than proper timing of autumn-applied aphidicides, resistance or tolerance factors bred into agronomically acceptable cultivars, some understanding of when to sow the crops in relation to vector flight activity, or a degree of rotation has been attempted. There is still much to be learned about the epidemiology of barley yellow dwarf and, similarly, to put into practice what is already known.

CONCLUSIONS

Barley yellow dwarf is indeed a complex pathosystem, every ecological facet replete in convoluted intricacy. The epidemiology of such a system is exceptionally difficult to elucidate and predict because it is influenced by so many interlocking activities, only some of which are known and measurable. It is one thing to know that a certain proportion of immigrating alate aphids are viruliferous and quite another to know whether this incoming potential inoculum will lead to epidemics, as an inordinate number of intertwined biological and physical interactions intercede.

Writing this article has made it apparent that, even with the wealth of

information already known about this important and fascinating pathosystem, very little of its ecology is fully understood. This begs the question, why? We believe the answer lies in two significant aspects of the biology of the pathosystem: the virus complex itself and how vectors move and disseminate virus among plants. The complexity of the viruses causing barley yellow dwarf around the world has long been apparent, but only now are the viruses being fully characterized. A coherent classification is still lacking, and the strain scheme now being used globally is fraught with problems. This restricts an understanding of the pathosystem's ecology because the various viruses behave differently, and it is currently impossible to gather sufficient coherent information to develop definitive principles to account for epidemics of barley yellow dwarf until what is being dealt with is understood.

Without an understanding of what makes vectors move, how far they travel, and what causes them to settle, feed, and reproduce, it is difficult to explain how barley yellow dwarf epidemics progress, for it is the specific behavioral traits of vectors that drive and sustain epidemics. Simply designating aphids as vectors of BYDV is quite misleading also, for the various species that transmit do so differently. Moreover, within each species a very diverse range of populations and biotypes exists, each of which has its own specific vector capabilities, responds slightly differently to external stimuli, and is potentially keyed to settle on different plant genotypes.

All of this makes it questionable whether it will ever be possible fully to understand barley yellow dwarf epidemics. Nevertheless, predicting epidemics will eventually become routine once we have a better understanding of the major biological components that collectively constitute the pathosystem, how these components interact within given environments, and, with a knowledge of primary inoculum sources, how temporal gaps are overcome, and how vectors disperse, coupled with competent modeling efforts. Furthermore, the knowledge base needed to predict epidemics is much the same as that needed to construct sound management strategies. Barley yellow dwarf epidemiology is, indeed, a study in ecological complexity. It presents a challenge that must be met if the disease system is to be managed. Assuming adequate resources for experimentation, we believe that within a decade much of the knowledge needed to understand and control this pathosystem will be available and operating proficiently in at least some developed countries.

ACKNOWLEDGMENTS

This article represents concepts that were formed through interactions with colleagues who have reviewed the manuscript and made substantial additions. We are greatly indebted to Catherine E. Eastman, Adrianna D. Hewings, and Gail E. Kampmeier for their assistance and guidance.

Literature Cited

1. A'Brook, J. 1981. Vectors of barley yellow dwarf virus. In *EURAPHID 1980: Aphid Forecasting and Pathogens and a Handbook for Aphid Identification*, ed. L. R. Taylor, p. 21. Harpenden, England: Rothamsted Exp. Stn.
2. Ahman, I., Weibull, J., Pettersson, J. 1985. The role of plant size and plant density for host finding in *Rhopalosiphum padi* (L.) (Hemiptera: Aphididae). *Swed. J. Agric. Res.* 15:19–24
3. Ajayi, O. 1986. The effect of barley yellow dwarf virus on the amino acid composition of spring wheat. *Ann. Appl. Biol.* 108:145–49
4. Ajayi, O., Dewar, A. M. 1983. The effect of barley yellow dwarf virus on field populations of the cereal aphids, *Sitobion avenae* and *Metopolophium dirhodum*. *Ann. Appl. Biol.* 103:1–11
5. Andrews, C. J., Paliwal, Y. C. 1983. The influence of preinfection cold hardening and disease development periods on the interaction between barley yellow dwarf virus and cold stress tolerance in wheat. *Can. J. Bot.* 61:1935–40
6. Araya, J. E., Foster, J. E. 1987. Laboratory study on the effects of barley yellow dwarf virus on the life cycle of *Rhopalosiphum padi* (L.). *J. Plant Dis. Protect.* 94:578–83
7. Baltenberger, D. E., Ohm, H. W., Foster, J. E. 1987. Reactions of oat, barley and wheat to infection with barley yellow dwarf virus isolates. *Crop Sci.* 27:195–98
8. Baranyovits, F. 1973. The increasing problems of aphids in agriculture and horticulture. *Outlook Agric.* 7:102–8
9. Bayon, F., Ayrault, J. P. 1989. Barley yellow dwarf virus: Epidemiology and risk forecasting. See Ref. 14, pp. 336–39
10. Blackman, R. L., Eastop, V. F. 1985. *Aphids on the World's Crops: An Identification Guide*. New York: Wiley & Sons. 466 pp.
11. Blackman, R. L., Halbert, S. E., Carroll, T. W. 1990. Association between karyotype and host plant in corn leaf aphid, *Rhopalosiphum maidis* (Fitch) (Hemiptera: Aphididae) in the northwestern United States. *Environ. Entomol.* In press
12. Brown, J. K., Wyatt, S. D., Hazelwood, D. 1984. Irrigated corn as a source of barley yellow dwarf virus and vectors in eastern Washington. *Phytopathology* 74:46–49
13. Burnett, P. A. 1984. *Barley Yellow Dwarf, a Proceedings of the Workshop*. Preface, pp. 6–13. CIMMYT, Mexico. 209 pp.
14. Burnett, P. A., ed. 1989. *Barley Yellow Dwarf Virus, the Yellow Plague of Cereals*. CIMMYT, Mexico. 511 pp.
15. Burnett, P. A., Mezzalama, M. 1989. The barley yellow dwarf screening program at CIMMYT. See Ref. 14, pp. 434–40
16. Carrigan, L. L., Ohm, H. W., Foster, J. E. 1983. Barley yellow dwarf virus translocation in wheat and oats. *Crop Sci.* 23:611–12
17. Catherall, P. L. 1966. Effects of barley yellow dwarf virus on the growth and yield of single plants and simulated swards of perennial rye-grass. *Ann. Appl. Biol.* 57:155–62
18. Catherall, P. L., Parry, A. L. 1987. Effects of barley yellow dwarf virus on some varieties of Italian, hybrid and perennial rye grasses and their implication for grass breeders. *Plant Pathol.* 36: 148–53
19. Coceano, P. G., Peressini, S. 1989. Colonization of maize by aphid vectors of barley yellow dwarf virus. *Ann. Appl. Biol.* 114:443–47.
20. Cockbain, A. J. 1961. Fuel utilization and duration of tethered flight in *Aphis fabae* Scop. *J. Exp. Biol.* 38:163–74
21. Comeau, A., Dubuc, J. P. 1977. Observations on the 1976 barley yellow dwarf epidemic in eastern Canada. *Can. Plant Dis. Surv.* 57:42–44
22. Comeau, A., Jedlinski, H. 1989. Successful breeding for barley yellow dwarf resistance or tolerance: A systematic approach related to the agronomic characteristics. See Ref. 14, pp. 441–51
23. Comeau, A., Pelletier, G. J. 1976. Predisposition to septoria leaf blotch in oats affected by barley yellow dwarf virus. *Can. J. Plant Sci.* 56:13–19
24. Conti, M., D'Arcy, C. J., Jedlinski, H. 1989. The "yellow plague" of cereals, barley yellow dwarf virus. See Ref. 14, pp. 1–6
25. Coon, B. F. 1959. Aphid populations on oats grown in various nutrient solutions. *J. Econ. Entomol.* 52:624–26
26. Creamer, R., Falk, B. W. 1989. Characterization of a nonspecifically aphid-transmitted CA-RPV isolate of barley yellow dwarf virus. *Phytopathology* 79:942–46
27. Delserone, L. M., Cole, H. Jr., Frank, J. A. 1987. The effects of infections by *Phrenophora teres* and barley yellow

dwarf virus on the freezing hardiness of winter barley. *Phytopathology* 77:1435–37
28. de Pace, C., Delre, V., Porceddu, E., Casa, R. 1989. The use of cloned hybridization probes to detect barley yellow dwarf virus infection in tetraploid wheat germplasm. See Ref. 14, pp. 421–28
29. du Toit, F. 1989. Resistance to *Diuraphis noxia* in wheat: Screening techniques and identification of resistance. See Ref. 14, pp. 452–55
30. Eweida, M., Oxelfelt, P., Tomenius, K. 1988. Concentration of virus and ultrastructural changes in oats at various stages of barley yellow dwarf virus infection. *Ann. Appl. Biol.* 112:313–21
31. Fargette, D., Lister, R. M., Hood, E. L. 1982. Grasses as a reservoir of barley yellow dwarf virus in Indiana. *Plant Dis.* 66:1041–45
32. Fereres, A., Lister, R. M., Araya, J. E., Foster, J. E. 1989. Development and reproduction of the English grain aphid (Homoptera: Aphididae) on wheat cultivars infected with barley yellow dwarf virus. *Environ. Entomol.* 18:388–93
33. Foster, J. E., Stamenkovic, S. S., Araya, J. E. 1988. Life cycle and reproduction of *Rhopalosiphum padi* (L.) (Homoptera: Aphididae) on wheat in the laboratory. *J. Entomol. Sci.* 23:216–22
34. Gildow, F. E. 1980. Increased production of alatae by aphids reared on oats infected with barley yellow dwarf virus. *Ann. Entomol. Soc. Am.* 73:343–47
35. Gildow, F. E. 1983. Influence of barley yellow dwarf virus-infected oats and barley on morphology of aphid vectors. *Phytopathology* 73:1196–99
36. Gildow, F. E. 1989. Barley yellow dwarf-aphid vector interactions associated with virus transmission and vector specificity. See Ref. 14, pp. 111–22
37. Gildow, F. E., Ballinger, M. E., Rochow, W. F. 1983. Identification of double-stranded RNAs associated with barley yellow dwarf virus infection in oats. *Phytopathology* 73:1570–72
38. Gildow, F. E., Frank, J. A. 1988. Barley yellow dwarf virus in Pennsylvania: Effect of the PAV isolate on yield components of Noble spring oats. *Plant Dis.* 72:254–56
39. Gildow, F. E., Frank, J., Bingaman, D., Powell, C. 1987. Barley yellow dwarf viruses in small grains of Pennsylvania: Isolate identification, distribution, and vector efficiency. *Plant Dis.* 71:922–26
40. Gill, C. C. 1968. Rate of movement of barley yellow dwarf virus out of inoculated cereal leaves. *Phytopathology* 58:870–71
41. Gill, C. C. 1969. Cyclical transmissibility of barley yellow dwarf virus from oats with increasing age of infection. *Phytopathology* 59:23–28
42. Gill, C. C. 1969. Annual variation in strains of barley yellow dwarf virus in Manitoba, and the occurrence of greenbug-specific isolates. *Can. J. Bot.* 47:1277–83
43. Gill, C. C., Chong, J. 1979. Cytopathological evidence for the division of barley yellow dwarf virus isolates into two subgroups. *Virology* 95:59–69
44. Guang-he, A., You-ting, Q., Zhou-min, C., Li-yang, W. 1989. A cereal germplasm, "Zong 4," for resistance to barley yellow dwarf virus in China. See Ref. 14, pp. 394–95
45. Guy, P. L. 1988. Pasture ecology of barley yellow dwarf viruses at Sandford, Tasmania. *Plant Pathol.* 37:546–50
46. Halbert, S. E., Bishop, G. W., Blackmer, J., Connelly, J., Johnston, R., Sandovol, L., Pike, K. S. 1989. Barley yellow dwarf infectivity of *Rhopalosiphum padi* in maize as an estimate of primary inoculum pressure in irrigated winter wheat. See Ref 14, pp. 273–74
47. Halbert, S. E., Pike, K. S. 1985. Spread of barley yellow dwarf virus and relative importance of local aphid vectors in central Washington. *Ann. Appl. Biol.* 107: 387–95
48. Hand, S. C. 1989. The overwintering of cereal aphids on Gramineae in southern England, 1977–1980. *Ann. Appl. Biol.* 115:17–29
49. Hendrie, L. K., Irwin, M. E., Liquido, N. J., Ruesink, W. G., Mueller, E. A., et al. 1986. Conceptual approach to modeling aphid migration. In *Movement and Dispersal of Agriculturally Important Biotic Agents*, ed. D. R. MacKenzie, C. S. Barfield, G. G. Kennedy, R. D. Berger, pp. 541–82. Danville, IL: Claitor's. 611 pp.
50. Holmes, S. J. 1989. Improving the forecasting of barley yellow dwarf virus high risk conditions in autumn-sown cereals. *Proc. 4th Int. Plant Virus Epidemiology Workshop: Resistance to Viruses and Vectors. Temperate and Tropical Plants, Montpellier, France*, 3–8 Sept. pp. 238–41
51. Irwin, M. E., Kampmeier, G. E. 1989. Vector behavior, environmental stimuli and the dynamics of plant virus epidemics. In *Spatial Components of Plant Disease Epidemics*, ed. M. J.

Jeger, pp. 14–39. Englewood Cliffs, NJ: Prentice-Hall
52. Irwin, M. E., Thresh, J. M. 1988. Long-range aerial dispersal of cereal aphids as virus vectors in North America. *Philos. Trans. R. Soc. London Ser. B* 321:421–46
53. Jensen, S. G. 1973. Systemic movement of barley yellow dwarf virus in small grains. *Phytopathology* 63:854–56
54. Johnson, C. G. 1967. International dispersal of insects and insect-borne viruses. *Neth. J. Plant Pathol.* 73 (Suppl. 1):21–43
55. Johnson, C. G. 1969. *Migration and Dispersal of Insects by Flight*. London: Methuen. 763 pp.
56. Johnstone, G. R., Sward, R. J., Farrell, J. A., Greber, R. S., Guy, P. L., et al. 1989. Epidemiology and control of barley yellow dwarf viruses in Australia and New Zealand. See Ref. 14, pp. 228–39
57. Jorda, C., Medina, V., Garcia Jimenez, J., Alfaro, A. 1987. Incidence of barley yellow dwarf virus on rice in Spain. *Phytopatho. Mediterr.* 26:11–14
58. Jorda, C., Osca, J. M., Alfaro, A. 1989. Barley yellow dwarf in Spain. See Ref. 14, pp. 45–48
59. Kennedy, J. S. 1950. Aphid migration and the spread of plant viruses. *Nature* 165 (4208):1024–25
60. Kieckhefer, R. W., Gellner, J. L. 1988. Influence of plant growth stage on cereal aphid reproduction. *Crop Sci.* 28:688–90
61. Kieckhefer, R. W., Stoner, W. N., Thysell, J. R. 1976. Preferences of flight-active barley yellow dwarf virus vectors for healthy or diseased leaves. *Plant Dis. Reptr.* 60:939–41
62. Kogan, M. 1975. Plant resistance in pest management. In *Introduction to Insect Pest Management*, ed. R. L. Metcalf, W. H. Luckmann, pp. 103–46. New York: Wiley & Sons. 587 pp.
63. Lapierre, H., Moreau, J. P. 1986. Les rotations céréalières intensives. *Six années d'études concertées INRA-ONIC-ITCF, 1973–1983*. Paris: INRA
64. Larkin, P. J., Brettell, R. I. S., Banks, P., Appels, R., Waterhouse, P. M., et al. 1989. Identification, characterization, and utilization of sources of resistance to barley yellow dwarf virus. See Ref. 14, pp. 415–20
65. 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-oat aphid, *Rhopalosiphum padi*. *Ann. Appl. Biol.* 97:135–41
66. Leather, S. R., Dixon, A. F. G. 1982. Secondary host preferences and reproductive activity of the bird cherry-oat aphid, *Rhopalosiphum padi*. *Ann. Appl. Biol.* 101:219–28
67. Liquido, N. J., Irwin, M. E. 1986. Longevity, fecundity, change in degree of gravidity and lipid content with adult age, and lipid utilisation during tethered flight of alates of the corn leaf aphid, *Rhopalosiphum maidis*. *Ann. Appl. Biol.* 108:449–59
68. Lister, R. M., Rochow, W. F. 1979. Detection of barley yellow dwarf virus by enzyme-linked immunosorbent assay. *Phytopathology* 69:649–54
69. Loxdale, H. D., Tarr, I. J., Weber, C. P., Brookes, C. P., Digby, P. G. N., Castanera, P. 1985. Electrophoretic study of enzymes from cereal aphid populations. III. Spatial and temporal genetic variation of populations of *Sitobion avenae* (F.) (Homoptera: Aphididae). *Bull. Entomol. Res.* 75:121–41
70. Makkouk, K. M., Azzam, O. I., Skaf, J. S., El-Yamani, M., Cherif, C., Zouba, A. 1989. Situation review of barley yellow dwarf virus in West Asia and North Africa. See Ref. 14, pp. 61–65
71. Markkula, M., Laurema, S. 1964. Changes in the concentration of free amino acids in plants induced by virus diseases and the reproduction of aphids. *Ann. Agric. Fenn.* 3:265–71
72. McGrath, P. F., Bale, J. S. 1989. Cereal aphids and the infectivity index for barley yellow dwarf virus (BYDV) in northern England. *Ann. Appl. Biol.* 114:429–42
73. McGrath, P. F., Bale, J. S., Tones, S. T. 1987. Effects of sowing date and spray date on the incidence of cereal aphids and BYDV in winter barley. *Proc. Crop Protection in Northern Britain*, pp. 105–10. Dundee, Scotland: Scott. Crop Res. Inst.
74. McGuire, P. E., Qualset, C. O. 1989. Transfer of the Yd_2 gene from barley to wheat. See Ref. 14, pp. 476–81
75. Milinko, I., Nagy, P. 1989. Barley yellow dwarf in Eastern Europe. See Ref. 14, pp. 57–60
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 granarium*. *J. Econ. Entomol.* 57:970–74
77. Miller, W. A., Waterhouse, P. A., Gerlach, W. L. 1988. Sequence and organization of barley yellow dwarf virus genomic RNA. *Nucleic Acids Res.* 16:6097–111
78. Miller, W. A., Waterhouse, P. A., Ger-

lach, W. L. 1988. Sequence and identification of the barley yellow dwarf virus coat protein gene. *Virology* 165: 306–9

79. Montllor, C. B., Gildow, F. E. 1986. Feeding responses of two grain aphids to barley yellow dwarf virus-infected oats. *Entomol. Exp. Appl.* 42:63–69
80. Moriones, E., Ortego, F., Ruiz Tapiador, M., Gutierrez, C., Castanera, P., Garcia-Arenal, F. 1989. Epidemics of RPV- and PAV-like barley yellow dwarf viruses in cereals in Spain. See Ref. 50, p. 250
81. Orlob, G. B. 1963. The role of ants in the epidemiology of barley yellow dwarf virus. *Entomol. Exp. Appl.* 6:95–106
82. Osler, R., Loi, N., Lorenzoni, C., Snidaro, M., Refatti, E. 1985. Barley yellow dwarf virus infections in maize (*Zea mays* L.) inbreds and hybrids in northern Italy. *Maydica* 30:285–99
83. Paliwal, Y. C. 1982. Role of perennial grasses, winter wheat, and aphid vectors in the disease cycle and epidemiology of barley yellow dwarf virus. *Can. J. Plant Pathol.* 4:367–74
84. Paliwal, Y. C., Andrews, C. J. 1989. Barley yellow dwarf virus-host plant interactions affecting winter stress tolerance in cereals. See Ref. 14, pp. 313–20
85. Paliwal, Y. C., Comeau, A. 1984. Epidemiology of barley yellow dwarf virus in Ontario and Quebec in 1982 and 1983. *Can. Plant Dis. Surv.* 64:21–23
86. Plumb, R. T. 1977. Grass as a reservoir of cereal viruses. *Ann. Phytopathol.* 9:361–64
87. Plumb, R. T. 1983. Barley yellow dwarf virus—a global problem. In *Plant Virus Epidemiology*, ed. R. T. Plumb, J. M. Thresh, pp. 185–98. London: Blackwell Sci. 377 pp.
88. Plumb, R. T. 1984. Chemical and cultural control of barley yellow dwarf. See Ref. 14, pp. 52–57
89. Plumb, R. T. 1989. The epidemiology of barley yellow dwarf in Europe. See Ref. 14, pp. 215–27
90. Potter, L. R., Jones, I. T. 1981. Interaction between barley yellow dwarf virus and powdery mildew in four barley genotypes. *Plant Pathol.* 30:133–39
91. Price, R. D., Stubbs, L. L. 1963. An investigation of the barley yellow dwarf virus as a primary or associated cause of premature ripening or "deadheads" in wheat. *Aust. J. Agric. Res.* 14:154–64
92. Refatti, E., Loi, N., Lorenzoni, C., Snidaro, M., Carraro, L. 1989. Maize, a natural and experimental host of barley yellow dwarf virus in northern Italy. See Ref. 14, pp. 269–72
93. Rochow, W. F. 1970. Barley yellow dwarf virus: Phenotypic mixing and vector specificity. *Science* 167:875–78
94. Rochow, W. F. 1982. Dependent transmission by aphids of barley yellow dwarf luteoviruses from mixed infections. *Phytopathology* 72:302–5
95. Rochow, W. F. 1984. Letter to Peter Burnett. See Ref. 14, pp. 204–5
96. Rochow, W. F., Hsu, J. S., Foster, R. L., Hsu, H. T. 1987. Parallel identification of five luteoviruses that cause barley yellow dwarf. *Plant Dis.* 71:272–75
97. Rochow, W. F., Jedlinski, H., Coon, B. F., Murphy, H. C. 1965. Variation in barley yellow dwarf of oats in nature. *Plant Dis. Reptr.* 49:692–95
98. Roitberg, B. D., Myers, J. H., Frazer, B. D. 1979. The influence of predators on the movement of apterous pea aphids between plants. *J. Anim. Ecol.* 48:112–22
99. Saksena, K. N., Singh, S. R., Sill, W. H. Jr. 1964. Transmission of barley yellow dwarf virus by four biotypes of the corn leaf aphid. *Rhopalosiphum maidis*. *J. Econ. Entomol.* 57:569–71
100. Shukle, R. H., Lampe, D. J., Lister, R. M., Foster, J. E. 1987. Aphid feeding behavior: Relationship to barley yellow dwarf virus resistance in *Agropyron* species. *Phytopathology* 77:725–29
101. Skaria, M., Lister, R. M., Foster, J. E. 1984. Lack of barley yellow dwarf virus dosage effects on virus content in cereals. *Plant Dis.* 68:759–61
102. Slykhuis, J. T., Zillinsky, F. J., Hannah, A. E., Richards, W. R. 1959. Barley yellow dwarf virus on cereals in Ontario. *Plant Dis. Reptr.* 43:849–54
103. Slykhuis, J. T., Zillinsky, F. J., Young, M., Richards, W. R. 1967. Notes on the epidemiology of barley yellow dwarf virus in eastern Ontario in 1959. *Plant Dis. Reptr.* 262:317–22
104. Smith, H. C. 1963. Control of barley yellow dwarf virus in cereals. *NZ J. Agric. Res.* 6:229–44
105. Sward, R. J. 1989. The effects of the interaction of barley yellow dwarf virus and take-all fungus on the growth and yield of wheat. Ref. 14, pp. 305–12
106. Sward, R. J., Kollmorgen, J. F. 1986. The separate and combined effects of barley yellow dwarf virus and take-all fungus (*Gaeumannomyces graminis* var. *tritici*) on the growth and yield of wheat. *Aust. J. Agric. Res.* 37:11–22
107. Swenson, K. G. 1963. Effects of insect and virus host plants on transmission of viruses by insects. *Ann. NY Acad. Sci.* 105:730–40
108. 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. *Ann. Appl. Biol.* 112:1–11

109. Taylor, L. R. 1986. The distribution of virus disease and the migrant vector aphid. In *Plant Virus Epidemics: Monitoring, Modelling, and Predicting Outbreaks*, ed. G. D. McLean, R. G. Garrett, W. G. Ruesink, pp. 35–57. Sydney: Academic 550 pp.
110. Thresh, J. M. 1983. The long-range dispersal of plant viruses by arthropod vectors. *Philos. Trans. R. Soc. London Ser. B* 302:497–528
111. Tsumuki, H., Kanehisa, K., Kawada, K. 1989. Leaf surface as a possible resistance factor of barley to cereal aphids. *Appl. Entomol. Zool.* 24:295–301
112. Voegtlin, D. J., Steiner, W. W. M., Irwin, M. E. 1987. Searching for the source of the annual spring migrants of *Rhopalosiphum maidis* (Homoptera: Aphididae) in North America. In *Population Structure, Genetics, and Taxonomy of Aphids and Thysanoptera. Proc. Int. Symp., Smolenice, Czechoslovakia.* Sept. 9–14, 1985, ed. J. Holman, J. Pelikan, A. F. G. Dixon, L. Weismann, pp. 120–33
113. Walters, K. F. A., Dixon, A. F. G. 1982. The effect of host quality and crowding on the settling and take-off of cereal aphids. *Ann. Appl. Biol.* 101: 211–18
114. Watt, A. D. 1979. The effect of cereal growth stages on the reproductive activity of *Sitobion avenae* and *Metopolophium dirhodum*. *Ann. Appl. Biol.* 91:147–57
115. Wyatt, S. D., Seybert, L. J., Mink, G. 1988. Status of the barley yellow dwarf problem in winter wheat in eastern Washington. *Plant Dis.* 72:110–13
116. Xin, Z. Y., Brettell, R. I. S., Cheng, Z. M., Waterhouse, P. M., Appels, R., et al. 1988. Characterization of a potential source of barley yellow dwarf virus resistance for wheat. *Genome* 30:250–57