Plant Disease Epidemiology in the Twentieth Century

A Picture by Means of Selected Controversies

The essence of science is to ask questions and to discuss possible answers. In the course of time, old questions may become irrelevant and old answers may prove to be incorrect. Science is alive as long as the discussion goes on. Controversy stirs discussion. In this paper on plant disease epidemiology in the twentieth century, I intend to discuss some selected controversies from three periods: before, during, and after J. E. Vanderplank.

The pre-Vanderplankian Period

The discussion during the nineteenth century was dominated by the feud between the autogenetists (autogenesis = *generatio spontanea*) and the pathogenetists, to borrow H. H. Whetzel's terminology (119). Summarizing briefly, the autogenetists claimed that fungus was the outcome of disease, whereas the pathogenetists said that fungus was the cause of disease. With a wink to Louis Pasteur (75) and Anton De Bary (20), phytopathologists (89) decided in favor of the pathogenetists and accepted Koch's "postulates" (55,56) as a methodological tool to demonstrate the pathogenicity of an organism, viruses included (8).

Many textbooks from the early twentieth century avoided the controversy by using a terminology that is vague and evasive in our present eyes (22,54,106,114), and I do not know when the postulates really came into use. Today, we try to distinguish clearly between cause and condition, hammering this difference into the heads of our students. We teach the convention that the pathogen is the *cause* of disease, but that the pathogen needs favorable *conditions* to do its work. Philosophically, the distinction between cause and condition is clear. In a given case, the distinction may be less obvious, especially when the short-term desire for profit wins over the long-term wisdom of sustainability. We tend to ignore the idea that the greedy human may set the conditions for the pathogen to do

harm, for example, by shortening the crop rotation period so much that problems are solicited due to soilborne fungi and nematodes. In this case, the pathogen is the proximate (4) cause and man is the ultimate cause of disease, as pictorialized by the disease tetrahedron (127; Fig. 1). And in this case, the major fight of the nineteenth century is not yet concluded, today, in the twenty-first century.

Following, are three controversies from the early part of the twentieth century, the pre-Vanderplankian period. The first, on Eriksson's mycoplasma theory, remained inconsequential. The second, on Marshall Ward's bridging host theory, has led through improved methodology to unexpected vistas. The last, on epidemiological growth curves, is still ongoing.

Controversy one: mycoplasma. The Swede Jakob Eriksson (1848–1931; Fig. 2), the international leader of phytopathology in his time (31), created the concept of the *formae speciales* in cereal rusts (2,32) and studied the overwintering of stripe rust (*Puccinia striiformis*) on wheat. To explain the apparent disappearance of rust during winter, he hypothesized that the rust mycelium residing in the leaves of field-grown winter wheat would lose its individuality by merging with the cytoplasm in the

host-cell to form a chimeric unit, which he called a "mycoplasma." Mycoplasma also explained the supposed seed transmission of stripe rust (24,26). At the time, the concept of mycoplasma was already known in medicine. Eriksson (25) wrote that he actually had seen mycoplasmas under the microscope.

The Brit Harry Marshall Ward (Fig. 3), famous for having solved (113) the problem of the coffee leaf rust (*Hemileia vastatrix*) in Ceylon (=Sri Lanka), accused Eriksson of poor microscopy (116,118). Eriksson (27) had to admit that the mycoplasmas he had seen were in fact haustoria. However, a petulant Eriksson did not give up, changed his argument (28,29), and fiercely defended his mycoplasma theory. He underpinned his views with fresh evidence from another pathosystem (30), rust on hollyhock (*Puccinia malvacearum*), evidence that was circumstantial at best. As far as I know, Eriksson chose to ignore the deadly comment by Biffen (7) on the mycoplasma theory as an explanation for seed transmission of stripe rust, a comment based on the just-detected mode of inheriting resistance against stripe rust. Klebahn (54) in 1912 dryly stated that the theory did not meet with much support ("kein Beifall"), and Brierley (9) in 1930 even

Fig. 1. The disease tetrahedron, dominated by man the farmer, manager, administrator. The horizontal plane or base represents the classical disease triangle. (127, printed with permission)

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used the word “ludicrous.” Fifty years later, simple experimental methods, already available to Eriksson in the 1890s, showed once more that the mycoplasma theory was superfluous (121). Numerous attempts at demonstrating seed transmission of stripe rust failed as miserably as those with stem rust (47).

Even great men may err. Eriksson was wrong, but we do not deny the existence of mycoplasma-like organisms as plant pathogens, demonstrated convincingly in later years. Here, I pay tribute to Jakob Eriksson who, mistaken in his mycoplasma theory,

Fig. 2. Professor Eriksson, Sweden. Portrait from the Eriksson Gold Medal for Excellence in Phytopathology. Picture kindly provided by the 1993 Laureate, Professor Ariena van Bruggen.

Fig. 3. Professor Henry Marshall Ward (39).

was nonetheless a great phytopathologist, a real internationalist, and a typical forerunner of epidemiology.

Controversy two: bridging hosts. Harry Marshall Ward (1854–1906) studied the rust of brome grasses, *Puccinia dispersa* (Erikss.), and formulated the “bridging host” theory (117). In his words, a “bridging species” allows “the passage of the fungus from species of one circle of alliance to those of another, in spite of the fact that it is usually closely adapted to species of one section of the genus only.” In other words, he assumed that a rust species found on a first grass species could pass over a second grass species to become infectious to a third one, not related to the first. The second host species he dubbed “bridging host.” Ward provided arguments from his own experiments and quoted guest workers studying rust and mildew of bromes (*Bromus* spp.).

Ward (115) developed a kind of axenic single-plant culture. He used spore batches and he did not mention single-spore or single-pustule cultures. (Ward’s confusion is quite understandable because differential interactions between pathogen and host phenotypes at levels from the *forma specialis* to the pathotype can be very complicated in natural situations [2].) Brooks (10) pointed out that in Ward’s time, “the importance of maintaining the purity of the physiologic races under investigation was not realized...” and he concluded that “The existence of ‘bridging hosts’ in connection with other fungi has not been substantiated.” The experimental results could not be replicated by others and could be ascribed as well to impurity of the inoculum

used. The bridging host theory was refuted by E. C. Stakman et al. (92).

The link between Marshall Ward and Stakman is E. M. Freeman from the University of Minnesota, who spent the spring and early summer of 1892 in Cambridge. (Ward apparently misquoted Freeman’s initials in some of his papers.) Freeman published on rusts of bromes (38), and he wrote a memorial to Ward (39). While head of the Division of Plant Pathology on the St. Paul campus, Freeman hired E. C. Stakman as an instructor in plant pathology. Later, he became Stakman’s Ph.D. supervisor (17). Stakman obviously liked methodological skirmishes, but curiously the first description of single-spore and single-pustule isolation is not attributed to Stakman but to E. B. Mains (62,77). Stakman’s work, in my opinion, led to the near-elimination of stem rust (*Puccinia graminis*) of wheat from the United States and many other parts of the world (17).

Controversy three: epidemiological growth curves. The never-ending discussion about growth curves is a very polite one, and the controversy is under the surface rather than explicit. The exponential growth of a population was first described by the Dutch microbiologist Anthonie van Leeuwenhoek in 1716 (23). A good second was the French physicist Réaumur, in about 1734. In 1798, the English reverend Malthus (63) developed his gloomy view on mankind based on its supposed exponential growth as opposed to the putative arithmetic increase of food resources. In 1838, the Belgian mathematician Verhulst (105) developed the logistic equation to describe the growth of human populations, an equation reinvented

Fig. 4. Example of logistic growth of *Puccinia striiformis* in a wheat field. Horizontal: time in days. Dots are observation points in time with severity x ($0 \leq x \leq 1.0$). a. S-shaped logistic curve. b. Exponential curve fitting the early data. c. 50% severity point. (127, printed with permission)

by the Americans Pearl and Read in 1920 (76). In 1960, Vanderplank (99) lighted the candle using the logistic equation as a tool in comparative epidemiological studies (100,121; Fig. 4).

In addition to the classical logistic equation, with one parameter only, a family of equations was created describing asymmetric S-shaped curves (12,42). Disease progress curves attained great descriptive precision, with coefficients of correlation between observed and calculated values exceeding 99%. Unfortunately, the explanatory value of these equations decreases with increasing descriptive precision. Hence, the usefulness of such precision is questionable. To quote a completely arbitrary example: "The Gompertz model gave the best statistical fit for the progression of disease incidence over time" (96), which I consider an empty statement. In methodological terms, the extra parameters needed to obtain the precision lack "empirical references." Descriptive over-perfection does not necessarily provide acceptable predictions.

In my opinion, the steady progress in calculation capacity has not contributed much to practical objectives based on disease progress curves such as spray warnings and loss prediction. In fact, prediction and spray warning using the simplest exponential equation may be perfectly adequate (123). The controversy, buried under peer admiration and editorial perfectionism, is worth a frank and open discussion.

Vanderplank and the Birth of Plant Disease Epidemiology

In my view, plant disease epidemiology gained its status as a separate discipline in 1963, due to two near-simultaneous events. One was the first international meeting of some 40 plant disease epidemiologists in

Pau, France. (The meeting was strictly informal, and no proceedings appeared. Pau started a sequence of similar informal meetings on plant disease epidemiology, including those at Wageningen [Netherlands, 1970], Raleigh [United States, about 1979], State College [United States, about 1982], Jerusalem [Israel, 1986], Giessen [Germany, 1990], Papendal [Netherlands, 1994], and Ouro Preto [Brazil, 2001].) The meeting was organized by Rick D. Schein from the United States and Jim Hirst from the United Kingdom as a corollary to another congress (43). The second event was the appearance of Vanderplank's famous book, *Plant Diseases: Epidemiology and Control* (100). Vanderplank (Fig. 5) was present at Pau, smiled and kept quiet, as he did not want to anticipate what was already in press. (Vanderplank told me that his British-born ancestors were of Flemish-Belgian descent and wrote their name as Vanderplank, in one word, as is still customary in Belgium. In official life he used the Dutch manner of writing, van der Plank, which was more congenial to the Afrikaners, the South Africans of Dutch descent.)

A body of knowledge obtains the status of discipline only when it is consistent in description and comparison and when it is applicable in extrapolation and prediction. Consistency cannot be measured, but it can be experienced. Under similar conditions, similar causes should lead to similar effects. Epidemiology suffers from the heterogeneity of its subject: so many crops, pathogens, weather types, and cultivation methods. Nonetheless, epidemiology is gaining consistency by the application of a limited vocabulary, the systematic use of generally accepted statistics to design experiments and test their results, and the assiduous application of mathematics. The feeling of commonality in a plethora of epidemiological problems and the need for a common language became apparent at the meeting in Pau, 1963. Vanderplank's 1963 book provided just that: a common language, consistency, and applicability.

Of course, Vanderplank used an already existing body of knowledge built by many outstanding scientists (13,126). But these scientists did not consider themselves epidemiologists, even when they were consciously engaged in epidemiological research. Their work, outstanding in their time, lacks the coherence, predictability, and general applicability of a discipline *per se*. These persons, among whom were the already-mentioned Marshall Ward and Eriksson, I designate as forerunners. I respect and admire them, but I do not call them epidemiologists.

Vanderplankian Controversies

Vanderplank's innovations sent shockwaves of surprise and commotion through the scientific world. He created and liked controversy, but he avoided discussion and

he pressed his opponents to prove him wrong by experimentation. I will mention three controversies created by Vanderplank.

Controversy one: horizontal resistance. In a brilliant combination of epidemiology and genetics, Vanderplank (100) postulated his antithesis of vertical and horizontal resistance (Fig. 6). I witnessed vehement and endless discussions between plant breeders and plant pathologists on the pros and cons of horizontal resistance (68). (Nelson's challenge of Vanderplank's definition of horizontal resistance was based on an omission in the 1963 book by Vanderplank, where he implied but did not state explicitly that the two genetic systems underlying vertical and horizontal resistance, respectively, were supposed to co-exist in one genotype.) In the beginning, Vanderplank met with fierce opposition from the breeders and with enthusiastic support by a small band of pathologists. At meetings, the converted preached to the believers (*vide* 59), generating a wealth of new ideas aimed at damping down the "boom-and-bust cycles" of epidemics. In the end, after about 25 years, the storm subsided and Vanderplank's ideas on "vertical" and "non-vertical" resistance became generally accepted (*vide* 48).

The interesting point is that Vanderplank was fundamentally wrong with his horizontal resistance. The existence of horizontal resistance cannot be proven, as follows from its definition (Sidebar 1). (The argument is in the observed reaction of known isolates; a next, not-yet-known isolate may refute the idea of horizontality by causing a very different reaction.) Detailed measurements did not confirm the posit of horizontal resistance (67,121). What remained were concepts such as "intermediate resistance" (122) and a special version thereof, "partial resistance" (72,73). In the vein of scientific rebellion, Parlevliet and Zadoks (74) proposed a model with differential interaction between host and parasite genotypes at the microlevel.

At the instigation of Raoul Robinson, the Food and Agriculture Organization (FAO) of the United Nations, Department of Plant Protection, initiated research on horizontal resistance in developing countries (83), largely financed by the Dutch government. Results, laid down in a set of Ph.D. theses, demonstrated that partial resistance provided an operational principle. The concept of partial resistance was further operationalized by another set of Ph.D. thesis researches (*vide* 48). The outcome will improve sustainability of resistance, without booms and busts and thus with less recourse to pesticides.

Controversy two: the mathematical point as the epidemiological universe. Mathematics in plant disease epidemiology were introduced primarily by Vanderplank (99). The light shone brightly when Vanderplank (100: equation 8.3, page 100)

Fig. 5. J. E. Vanderplank. (128, printed with permission)

advocated his famous equation

$$dx/dt = R_c \cdot (x_{t-p} - x_{t-i-p}) \cdot (1 - x_t)$$

where x = fraction of diseased plant material, R_c = corrected basic infection rate (Sidebar 1), p = latent period, i = infectious period, and t = time. This equation is intellectually satisfactory, and it has a strong explicative value; it is a great teaching aid. Nonetheless, the equation is fundamentally wrong. It represents the growth of an epidemic in time, but the spatial dimension of the epidemic is the mathematical point, the nonspace. However, an epidemic has a spatial dimension, which affects the shape of the epidemic growth curve in time (49,51).

Of course, Vanderplank was aware of the spatial aspects of epidemics. He discussed them extensively, introducing the term "horizon of infection" (98). In his books (100,102,103), he tended to discuss time and space separately. In a paper specifically devoted to "spread of plant pathogens in space and time" (101), he did not find an acceptable solution for the space-time problem, i.e., combining disease progress in time and in two-dimensional space by a single equation. Numerical mathematics showed a way out, in this case dynamic simulation using a digital computer (51,53). Analytical mathematics (21,49,50, 125) brought the final solution. One outcome was a theory on focus formation at the micro-, meso-, and continental levels (Fig. 7). Continued integration of epidemic processes in time and space is intellectually demanding, and the supporting experimentation is costly, but the rewards will be great (124).

Controversy three: numerical mathematics. The generation of phytopathologists before Vanderplank largely ignored the potential of analytical mathematics (*vide* quotation in 124). Vanderplank was a good mathematician and elaborated many epidemiological questions by the application of analytical mathematics. He did not yet appreciate the potential of numerical mathematics, as applied in dynamic simulation. He criticized the results of the famous simulators EPIDEM and EPIMAY (109,110) severely, although not unjustly (103: ch. 4.7).

Obviously, Vanderplank did not like dynamic simulation, as he told me personally. In his aversion to simulation, he was a typical representative of his generation. "Computers and plant pathology? Can't be done!" exclaimed a respectable colleague (who had never seen a computer) in 1976, when I proposed the project EIPRE (123) on supervised control of diseases and aphids in wheat. For a time, simulation became a subject of violent discussion between the pros and cons. At the close of the twentieth century, we focused on the complementarity of analytical and numerical approaches in epidemiology.

Toward the year 2000, the enthusiasm for simulation clearly diminished among plant pathologists, which is amazing in view of the problem-solving power of the technique (6,80). Simulation allows intricate combinatorics such as the integration of crop ecology, pathogen ecology, and human action in an overall picture of sometimes amazing precision as regards injury and damage (5). In the process of changing present high external input agriculture into more sustainable agriculture, dynamic simulation in combination with other computer techniques is highly supportive because it presents farmers with various alternatives (84).

Post-Vanderplankian Controversies

My eclectic survey of controversies in epidemiology is not exhaustive (see 108 for a more balanced overview). Nor did I do justice to epidemiologists other than Vanderplank. The publication of several textbooks (Sidebar 2) on epidemiology after the Vanderplankian era shows that the discipline is alive and kicking. Fortunately, I see several controversies emerging that need resolving. Moving from the proximate to the ultimate I mention three.

Controversy one: running or dispersive waves. van den Bosch et al. (97) and others elaborated the theorem of the running wave of expanding foci. The theorem implies that a focal epidemic, once it gains force, expands at a constant rate, whether the epidemic is a one-season one-meter

affair, a focus, or a pandemic spreading over a continent in a number of years (125). Ferrandino (36) developed the theorem of the dispersive wave, implying that an epidemic spreads at an ever increasing rate. The two views seem to be mutually exclusive. Nevertheless, both views are supported by empirical data. Either view finds mathematical support (*vide* 21), and various modifications were offered (1).

A final decision in this controversy is not yet possible, but a first step has been made. A mathematical technique allows us to distinguish sharply between the two theorems using small-scale experiments (37). If such a distinction can be made, the next step would be to determine the "domain of validity" of either theorem. This action might require a lot of epidemiological thinking and experimentation.

For applications such as outlining a strategy for biological control of pests and diseases, fundamental research on focal epidemics is dearly needed. Similarly, the practice of quarantine and containment may find support in the theory. Here, we transit from the technical-scientific discussion to a socioeconomic discourse with political implications.

Controversy two: crop loss assessment. Influential forerunners of epidemiology were recruited to solve phytopathological problems with serious socioeconomic consequences. Marshall Ward was hired to solve the coffee rust problem in Ceylon. Scientifically he made great strides, but he could not prevent the abandonment of coffee cultivation and bank-

Fig. 6. Resistance of cultivar Maritta to *Phytophthora infestans*. Along the horizontal axis are races with virulence genes in climbing order of complexity. On the vertical axis is resistance as a figure between 0 and 1.0. Maritta has 100% resistance to all races not containing virulence gene 1. Columns thus represent vertical resistance and susceptibility. If the races contain virulence gene 1, and the corresponding resistance is "broken," a moderate level (resistance = 0.4) of horizontal resistance becomes visible. The two types of resistance have different genetic backgrounds and may concur in one cultivar (redrawn after 102).

ruptcy of growers and banks (60). Before World War I, crop loss was the international theme that led to the foundation of the International Agricultural Institute in Rome, 1905, the predecessor of FAO (126). During the interbellum, several

authors showed an interest in the economic, social, and political dimensions of plant pathology. After 1945, recruitment of plant pathologists followed the intricacies of the academic world rather than economic necessities. The result was a loss of

interest in solving problems in the field (44).

In 1967, FAO organized a conference on crop loss in Rome (35). Quantification of worldwide crop losses was presented by Cramer (19) and applauded. New methodology for crop loss assessment was developed (15). The enthusiasm it generated (16) was a straw fire that, briefly rekindled by a few workshops (94), gradually died down. The topic did not have the glamour of biochemistry, let alone biotechnology.

My objections to the results of Cramer and of his successor Oerke et al. (69) are of three kinds (129). First, their results were largely based on chemical control experiments, comparing well-sprayed plots to nonsprayed controls, both positioned in a well-sprayed environment where natural, biological control was practically eliminated. Hence, the environment was ill-chosen. Second, no alternatives other than chemical treatment were considered, such as resistance breeding or biological control. Thus, the comparisons were false. Third, the losses attributed to the individual pests were combined to give one total loss figure. Such a figure will be exaggerated because not all pests concur on the same place at the same time and because pests that do concur may interact (127: ch. 8). Thus, the calculations were incorrect.

New methodology for large-scale surveys (85) was proposed using individual fields (or plots) as the research units and their agricultural and phytopathological variates as the attributes. Applying non-parametric statistics, a powerful tool was created with descriptive and explanatory value at the regional level, i.e., over hundreds to thousands of kilometers. With this methodology, the International Rice Research Institute (IRRI) at Los Baños, Philippines, analyzed rice cropping patterns, pest profiles, and crop loss (86,87). Scientists sampled hundreds of fields scattered over Southeast Asia. In the supporting experiments, they took care to avoid pesticide-induced changes in crop development and pesticide-induced damage to biological control. Two points became obvious: first, that severe outbreaks of pests and diseases are rare, and second, that pests interact, usually in such a way that the actual damage in farmers' fields is less than expected by adding up the damages attributed to the individual pests in those fields.

Controversy three: socioeconomic impact of epidemiology. Spanning the twentieth century once more, I come to my last controversy—or is it a self-criticism? At the beginning of the century, individuals such as Henry Marshall Ward and Jakob Eriksson (not to mention several others) were scientists with a keen eye for the socioeconomic consequences of plant disease epidemics. The young E. C. Stakman chose to study plant pathology because it was a science and it served people; he remained true to his choice and he became

Sidebar 1. Explanation of some epidemiological concepts

Differential interaction – Whereas in one variety resistance is high against one and low against another isolate of the pathogen, another variety shows the inverse pattern; usually based on gene-for-gene interaction.

Focus – The site of local concentrations of diseased plants or disease lesions, here about a primary source of infection.

Infection rate, exponential – Proportionality constant with dimension t^{-1} in equation for exponential growth.

Infection rate, logistic – Proportionality constant with dimension t^{-1} in equation for logistic growth.

Infection rate, corrected basic – Proportionality constant with dimension t^{-1} in equation for logistic growth corrected for latent period and infectious period.

Resistance, horizontal – Resistance of a variety supposedly equal to all isolates of a pathogen; no gene-for-gene interaction; resistance may be at any level from nearly zero to complete.

Resistance, intermediate – Resistance, either horizontal or vertical, which is incomplete, with a level somewhere between zero and complete resistance.

Resistance, partial – An intermediate resistance due to a reduction in the number of lesions, which are of normal (susceptible) type and size.

Resistance, vertical – Single-gene resistance against pathogen with corresponding virulence gene, according to a gene-for-gene interaction.

Fig. 7. Three orders of focal epidemics, schematically. At the center, the 0-order epidemic (microlevel) or focus, enlarged in the inset, developing within one season. Successive lines of equal severity (isopaths) are drawn. Size: one to a few meters in diameter. Black squares within the inner heavy line show the results of the first-order epidemic, developing within one season (meso-level), with two thin isopaths. Size: 1 to 1,000 km. Open squares between the two heavy lines represent the early results of the second-order epidemic (continental level), developing over successive years. (Shown is beginning of year 2 only.) Size: up to a whole continent. Original figure.

a “statesman of science” (17). Many textbooks before (3,93) but few after (91,95) World War II discussed socioeconomic aspects of epidemics in any depth.

Every nation has the right to choose the agriculture and the crop protection it wants, positioning itself somewhere along the line connecting organic agriculture at one end and high external input agriculture at the other. The latter engenders great social costs because of the high amounts of fertilizers and pesticides used (70,78,111), and the first might not be able to feed everybody. In the year 2000, the worldwide tendency toward sustainability requires redesigning crop protection, as painfully evidenced by the recent wheat scab catastrophe in the northern great plains of North America (120). Epidemiologists can go ahead, working hand-in-hand with crop scientists and with social scientists. What combination of crop control practices will approach the social optimum, considering farm income as well as consumer prices, environmental and human health, short-term as well as intergenerational effects, local and distant effects (129)? Transition from one way of growing crops to another may be painful, risky, and costly (33,34). Institutional constraints to change are one reason (112). Another reason may be that farmers get trapped by an economic mechanism called “path dependence” (18). It implies that a farmer will experience great difficulties and/or costs in changing from one disease control policy, e.g., intensive use of pesticides, to another one, e.g., Integrated Pest Management. On-farm experimentation, preferably by farmers (14), will be needed so that epidemiologists can return to where they began, the field. New field experience is needed, and models will help to understand and predict, to extrapolate and to steer the experimentation (124).

We may learn from the sociologists. Sociologists found that different management styles may all lead to a decent farm income, with higher or with lower inputs (90,104). Sociologists can also help to transit from one state of agriculture to another, more sustainable one. In parts of Southeast Asia and of Africa, Integrated Pest Management is passing the borderline between the natural and the social sciences, and is becoming applied social science (52,107).

Twentieth century epidemiology began in the field; it became strong in mathematical analysis but weak in agronomic wisdom, and less than weak in socioeconomic synthesis. The potential is there, but I have not yet seen, with the exception of Nagarajan et al. (66), the right frame of mind, the right attitude to bridge a long period, a wide distance, an astonishing agrobiodiversity, a socioeconomic perspective, and eventually, political implications. This is the criticism I offer; this is the challenge I present.

Sidebar 2. Textbooks on plant disease epidemiology (nonexhaustive overview)

Year	Authors/editors	Country	B/C/P
1901	Ward (114)	UK	C
1946	Gäumann (41)	Switzerland	C
1960	Horsfall & Dimond (46)	USA et al.	B
1963	Vanderplank (100)	South Africa	B
1973	Prasad et al. (79)	India et al.	P
1974	Kranz (57)	Germany et al.	B
1976	Robinson (82)	UK	B
1978	Scott & Bainbridge (88)	UK	B
1978	Horsfall & Cowling (45)	USA et al.	B
1979	Zadoks & Schein (127)	Netherlands, USA	B
1980	Palti & Kranz (71)	Germany et al.	P
1983	Nagarajan (64)	India	B
1986	Zeng Shimai (130)	China	B
1986	Leonard & Fry (61)	USA et al.	B
1988	Kranz & Rotem (58)	Germany et al.	B
1989	Jeger (50)	UK et al.	B
1990	Campbell & Madden (12)	USA	B
1991	Rapilly (81)	France	B
1994	Campbell & Benson (11)	USA et al.	B
1995	Nagarajan & Muralidharan (65)	India	B
1996	Bergamin & Amorim (6)	Brazil	B
1998	Gareth Jones (40)	UK et al.	B

B = book, C = chapter, P = proceedings.

Epilogue

I tried to give a bird’s-eye view of plant disease epidemiology as it developed into a discipline of its own during the twentieth century, developing its own language and finding support in mathematics. I indicated how forerunners of epidemiology discussed epidemiological questions, beginning with Eriksson and Marshall Ward around 1900, how Vanderplank really established the discipline as a consistent body of knowledge with predictive capacity, and how matters developed in the post-Vanderplankian period. I indicated what epidemiology left undone toward the end of the twentieth century. Limitations of time and of experience compelled me to make choices, personal choices. I could not discuss soilborne diseases, virus diseases, or diseases of perennial crops. Nonetheless, I attempted to give a balanced overview covering a century by focusing on selected controversies, open or hidden. I feel privileged by having participated during the second half of the twentieth century, and I am grateful for the invitation to report on that past century. The new century will provide wonderful new challenges to deepen and widen the discipline of plant disease epidemiology.

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