

Quantification and Modeling of Crop Losses: A Review of Purposes

Serge Savary,¹ Paul S. Teng,²
Laetitia Willocquet,¹ and Forrest W. Nutter, Jr.³

¹INRA, UMR Santé Végétale, BP81, Villenave d'Ornon 33883, France;
email: ssavary@inra.bordeaux.fr, lwillocq@inra.bordeaux.fr

²Natural Science and Science Education, National Institute of Education, Nanyang Technological University, 637616 Singapore; email: psteng@nie.edu.sg

³Department of Plant Pathology, Iowa State University, Ames, Iowa 50011;
email: fwn@iastate.edu

Annu. Rev. Phytopathol.
2006. 44:89–112

First published online as a
Review in Advance on
February 15, 2006

The *Annual Review of
Phytopathology* is online at
phyto.annualreviews.org

doi: 10.1146/
annurev.phyto.44.070505.143342

Copyright © 2006 by
Annual Reviews. All rights
reserved

0066-4286/06/0908-
0089\$20.00

Key Words

agroecosystem, decision, disease management, multiple pest systems, sustainability, systems analysis

Abstract

This review considers the cascade of events that link injuries caused by plant pathogens on crop stands to possible (quantitative and qualitative) crop losses (damage), and to the resulting economic losses. To date, much research has focused on injury control to prevent this cascade of events from occurring. However, this cascade involves a complex succession of components and processes whereby knowledge on crop loss generates entry points for management. Proposed here is a framework linking different types of knowledge on crop loss to a range of decision categories, from tactical to strategic short- or long-term. Important advances in this field are now under way, including a probabilistic treatment of the injury-damage relationship, or analyses of the sources of uncertainty attached to some components of the decision process. Management of injury profiles, rather than individual injuries, and shifts in dimensionality of crop losses are anticipated to contribute to the design of sustainable agricultural systems, and address global issues concerning food security and food safety.

Yield loss: decrease in quantitative output of a crop resulting from the injury caused by a pathogen (or more generally, a pest), or from injuries caused by several pathogens (or pests)

INTRODUCTION: INITIAL QUESTIONS

Plant diseases have played a key role in the selective breeding of modern crop varieties since the domestication of wild species some thousands of years ago. The social and economic effects of diseases and the consequent crop losses have been well-documented, ranging from systemic, often accepted, changes in growth and development to complete devastation of entire crops. Among the most notable historically were the potato late blight epidemics in Western Europe (5) and rice blast in China and India (90). While dramatic reduction in biomass output (yield loss) of key staple food crops are often cited to illustrate their importance, plant pathogens are recurrent reducing factors in the quality of agricultural outputs. Yield loss and quality loss are included in the concept of crop loss (15a). The very discipline of plant pathology grew out of the need to study plant pathogens in order to properly manage them. James qualified (44) crop losses as the *raison d'être* for plant pathology, and rightly asked why it is, in spite of their importance (and all the effort devoted to estimation; see 88), that reliable estimates are still so scant in the literature.

The state of knowledge on crop losses caused by plant diseases, and the methodology for crop loss assessment, have been reviewed previously (28, 72, 44, 46, 123, 129). In the decades following the initiative by the United Nations Food and Agriculture Organization (FAO) (14), a strong focus was on the development of methodology in field studies and mathematical modeling. Attempts were made to collect crop loss data over geographical units ranging from states and provinces to entire countries. Problems associated with up-scaling, such as sampling and spatial variability in diseased populations, received much attention, but the advent of increased computing power made available new tools such as geographic information systems, often with georeferencing equipment. In the 1980s and 1990s, research concentrated on the physio-

logical basis for yield loss, on the macro- and microeconomics of losses caused by individual diseases, then on multiple pests, and finally on the incorporation of crop loss information for disease management. Concepts in disease and crop loss assessment were influenced by advances in our understanding of biological systems at the molecular and ecosystem levels. Of particular significance was the research into techniques to use crop loss information for descriptive, problem-diagnostic purposes rather than prescriptive purposes.

Zadoks (146) described crop loss research as having undergone three historical periods: exploratory, emergency, and implementary. The exploratory period was characterized by interest stimulated by severe epidemics such as those of the potato late blight (1840s), an early period before common or standardized concepts and methodology for research. The emergency period covered the two world wars and saw a plethora of work in Europe and North America on methods to estimate crop losses on food staples such as potato and wheat (64, 69). The FAO (14, 15) pioneered efforts to standardize concepts, methods, and estimates of field losses globally, particularly methods of disease assessment and yield loss experimentation. Particularly important was the application of agroecological concepts such as levels of yield (theoretical, attainable, actual) in response to constraints (diseases, insects, physical environment, physiology, genetics), and the use of relatively simple mathematical models in quantitative studies (149). In retrospect, the powerful desktop computers available from the mid-1980s enabled many of these concepts to be explored further through complex crop physiological models, which in turn necessitated still stronger interdisciplinary cooperation in studying topics as complex as crop yield and how it is affected by single or multiple biotech and physical factors during the period of yield formation. The focus expanded from a primary treatment of the pathogen (disease symptom), with the host plant as a secondary property, to treatment

of the host plant's growth and development as the primary set of variables. Disease effects (together with other yield-limiting or -reducing factors) were then accounted for through a number of possible outcomes on plant morphology or physiology, depending on the pathogen type (4, 63, 99). Conceptually, the translation of potential pest effects on plants (e.g., reduction of leaf area, reduction in photosynthetic rate) into a number of equations to link disease to plants and plant stands was an important contribution to explaining yield loss and developing a generic framework for yield loss estimation that linked generalized concepts to locality-specific situations. A review of the concepts and methods is presented in the next section.

Crop losses, in particular yield losses, have been studied in the hope that the knowledge will provide quantitative estimates of the effect of disease on its host crop. Concurrently, pathologists involved in making decisions on disease management require information on the benefits accruing from a particular management action taken. Research leaders and agencies funding plant pathology vis-à-vis other disciplines have used crop loss information as a basis for resource allocation and research prioritization (35). A significant new trend in agriculture is the increasing participation by the private sector in agricultural research, starting in the 1990s with genetically modified crops expressing virus resistance (45). Corporate entry into agricultural research in anticipation of market development for their products made plain the need for information to justify investment. This need has been especially pertinent as it relates to transgenic traits that confer improved protection against insect pests and diseases because information on crop loss is a basis on which to calculate potential net returns over various timeframes and, consequently, develop business strategies (126). A corollary is that the crop loss models used in conjunction with georeferenced data to estimate benefits are often the same as those used to assess potential risks of damage from unintended

disease introductions, a very timely topic in the new field of biosecurity. However, as noted in a later section, many projections on losses or benefits from ameliorating losses lack corroborating data on actual field losses in the geographic areas of interest. It still belies the question raised by Lyman (69) as to why collection of crop loss data is so underinvested when so many important decisions depend on reliable data.

Most of this review mainly addresses quantitative losses (yield losses), but considers the other component of crop loss (14–15a, 76, 144, 146, 149), qualitative losses, as well.

CROP LOSS QUANTIFICATION AND MODELING: OVERVIEW OF CONCEPTS AND METHODS

This section offers a diagrammatic overview (**Figure 1**) of the flow of concepts (boxes) and approaches (rounded boxes) that have been used. The conceptual map of **Figure 1** only partly follows a chronological logic; more important in its construction is the sequence from concepts to approaches, possibly leading to new concepts and approaches.

Considerable attention has been devoted to the adoption of a common terminology (e.g., see 7, 86, 87). Throughout this article, we use the definitions listed in the glossary for injury, injury profile, yield loss, crop loss (damage), and loss (76, 144, 145, 149), and for production situation and attainable yield (6, 98, 109, 113).

Injury quantification implies measurements of both the disease and of the host (healthy and diseased), i.e., a preliminary portfolio (**Figure 1a**) (64, 149), which will include a range of methods, especially assessment keys (42). The response of plant canopies to diseases may vary, and often may be difficult to measure quantitatively. Use of remote sensing tools may be an efficient way to develop more precise injury-damage relationships, for example, in the cases of the peanut leaf spot (84) and of alfalfa foliar diseases (83).

Injury: any observable deviation from the normal (healthy) crop; injury may lead to crop loss (damage)

Injury profile: a given combination of injury levels caused by a range of pests during a crop cycle

Crop loss (damage): any decrease in quantity (yield loss) and/or quality of a crop output; damage may lead to loss. Note that crop loss encompasses yield loss

Loss: any decrease in economic returns from damage, and the cost of agricultural activities designed to reduce damage

Production situation: the physical, biological, technical, social, and economic context in which agricultural production takes place. In order to address crop losses, as here, it is often convenient to exclude pests from the biological components of production situations

Attainable yield: a reflection of a given production situation; the yield performance of a crop that has not been exposed to yield-reducing factors, especially pests

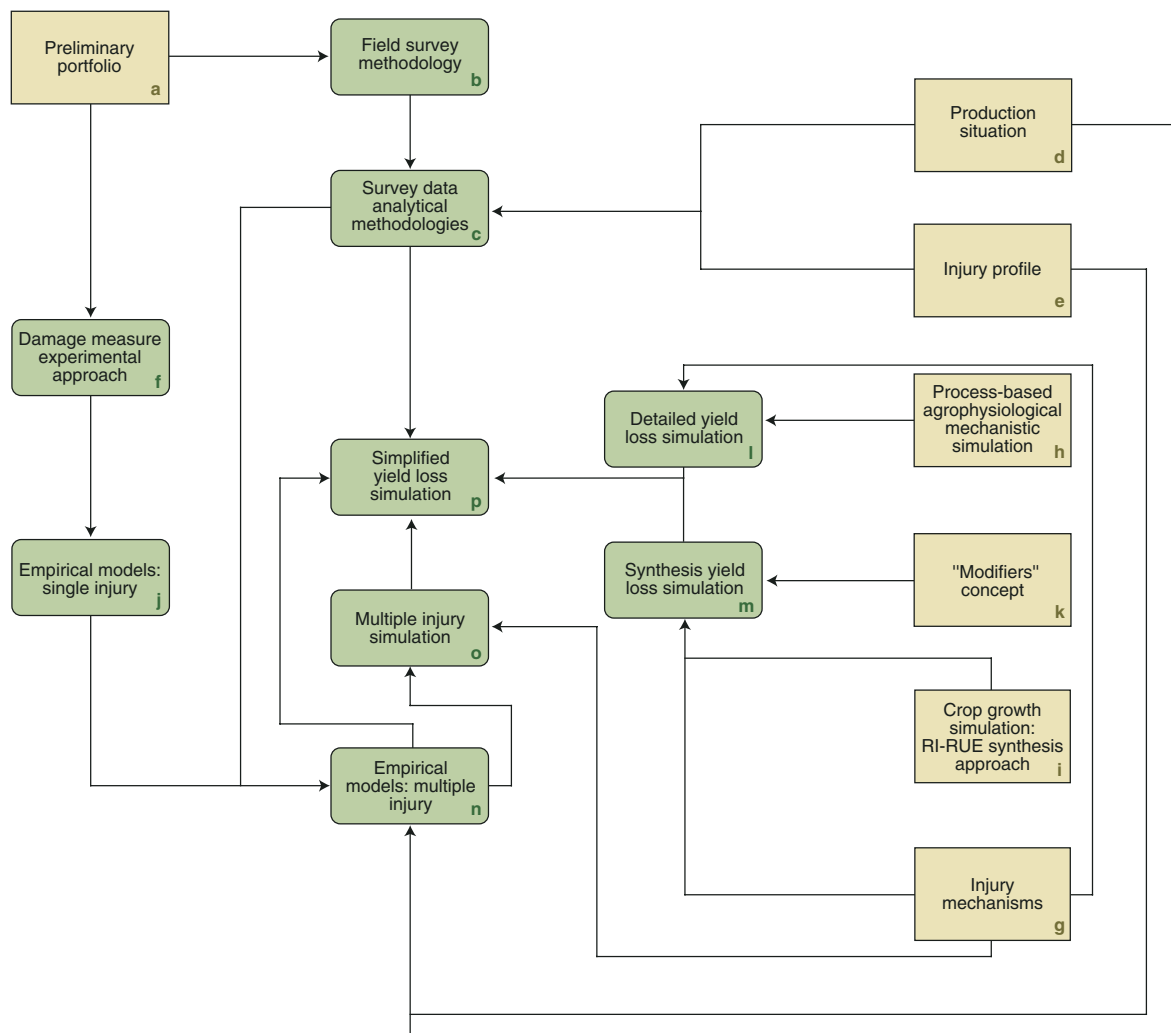


Figure 1

Diagrammatic overview of concepts and approaches used for crop loss quantification and modeling. Yellow boxes: concepts; rounded green boxes: approaches.

Proper injury quantification (both sufficiently precise and accurate; 85) is a key component to the conduct of field surveys (Figure 1b), where levels of injuries in a range of crop stands are measured at a number of development stages, along with several attributes and characteristics of the crop system (e.g., 46, 78, 109, 136, 137)—designing a survey procedure is constrained by formal

scientific requirements as stringent as the definition of the system to be modeled (98). The guiding principles for conducting disease surveys were outlined long ago (13, 16). The methodology was further discussed by several authors (e.g., 122, 149) and applied in a number of landmark studies [e.g., spring barley in the U.K. (43, 59), winter wheat in the U.K. (60) and Australia (120), and pea

in Idaho (137)]. Field surveys generate information at the level of the population of crop stands rather than of the individual plot, field, or orchard. Such surveys raise questions regarding variation in injury and its relationships to crop management, crop environment, crop performance, and the occurrence of multiple injuries. Analytical methodologies (**Figure 1c**) for survey data enable the resulting information to be synthesized and hypotheses proposed. The methodology depends on both the data collected and the hypotheses tested, and has been reviewed by Madden & Nutter (72). The diversity, or change, in production situations (**Figure 1d**) may be a key element for describing variations in injuries. Production situations are also key elements to describe (**Figure 1f, j**) or explain (**Figure 1b, l, p**) damage quantitatively. Field surveys often indicate that pest injuries seldom occur alone (92, 78); hence it is more relevant to consider injury profiles (112).

Measurement of damage requires experiments (**Figure 1f**), which in turn cannot be implemented without measurements (**Figure 1a**) and an experimental methodology (72, 123, 129). One prime objective of such experiments has been to identify injury-damage relationships (116), i.e., damage functions (145). The literature on this topic has been thoroughly reviewed (e.g., 10, 44, 46). Outputs of this research consist of single-point (injury measured at one date or stage of the crop), multiple-point (injury measured at several dates or stages), area under progress curve, or response-surface models (**Figure 1j**). Several multivariate (e.g., 70) and nonlinear approaches (e.g., 70, 73) have been developed to analyze these relationships.

Variation in production situation (**Figure 1d**, or some of its components) and consideration of injury profiles (**Figure 1e**) have been addressed in a comparatively smaller number of studies. Following the pioneering work on potato pests in the United States (47, 50, 53), studies have been

conducted on multiple injuries (**Figure 1n**) of bean in Brazil (20, 31), peanut in Africa (18, 113–115), and rice in Asia (111). Several studies have shown that injury profiles commonly correspond to production situations; in other words, syndromes of multiple injuries echo syndromes of production (110b).

A range of concepts has emerged that made mechanistic, agrophysiology-based simulation modeling possible. The diversity of harmful organisms can be condensed in a small number of guilds, each guild functionally corresponding to one type of injury mechanism (**Figure 1g**) (4, 77, 99). Thus, process-based agrophysiological models (**Figure 1b**) can be used to simulate yield losses (100, 107). Modifiers (**Figure 1k**) (67) can also be used to represent reduced performances at specified points of the system. Building upon Monteith's (82) simplified approach of crop growth, injuries have also been pooled in two main groups (**Figure 1i**): intercepted radiation (RI) or radiation use efficiency (RUE) reducers (48, 135). On the one hand, mechanistic physiological modeling (**Figure 1b**), combined with injury mechanism (**Figure 1g**) or modifier (**Figure 1k**) concepts, were used to develop detailed yield-loss simulation models (**Figure 1l**) (e.g., 32, 103, 127). On the other hand, the RI-RUE approach has helped in developing simpler models (2). Both (**Figure 1l, m**) approaches have value, leading to measurement of processes, as well as the simplifications that model evaluation requires (121). Simplified, mechanistic yield-loss simulation models (**Figure 1p**), though few in number (e.g., 51, 103), are invaluable for understanding processes at work in complex systems. When combined with experience gained on multiple pathosystems either through characterization (**Figure 1b**) or through experimental-empirical (**Figure 1f, n**) models, these approaches have enabled simulation models to be developed that incorporate multiple injuries (**Figure 1o**). There are still few models of this kind (49, 96, 140).

Tactical decision: decision pertaining to methods to solve a given pest problem or details of how a chosen method should be applied

Strategic decision: decision aimed at optimizing a multicomponent approach to the management of a pest or pests, including the selection of the appropriate methods and decision rules for their most effective application

EXPECTED OUTCOMES OF CROP LOSS QUANTIFICATION AND MODELING

Framework for Linking Crop Loss Knowledge and Decisions

Crop loss knowledge has very diverse forms, reflecting its context, use, and ultimate aim. One given form of knowledge may be better suited than another to a specific purpose; this section highlights relationships among forms of knowledge and usages. We distinguish six types of knowledge on disease (generally, pest) harmfulness (**Table 1**).

Much of the research on disease harmfulness has been related to the empirical environment:disease-intensity (i.e., environment-injury) relationships with the implicit assumption of a direct, fixed link between injury and damage. In spite of this implicit, and often simplistic, assumption, these environment-injury relationships have been, and still are, much used. Environment-injury

relationships are therefore included here as a first type of knowledge on crop loss. A second type of knowledge consists of disease-crop loss (injury-damage) relationships. These two first types refer to one level of integration (the crop stand) only. A third type, mechanistic simulation models, is based on knowledge at a level of integration finer than the crop stand, i.e., injury mechanisms incurred by a given crop-disease (pest) interaction. Mechanistic simulation models that consider a range of different harmful organisms represent a fourth type. A fifth type refers to crop loss knowledge encompassing a range of geographical, climatic, or production contexts, and provides the means for up-scaling knowledge. A sixth category is considered, whereby knowledge is generated through production situation-based modeling.

Measuring or predicting the harmfulness of diseases, i.e., crop loss knowledge, leads to decisions for disease management. As early as 1964 (12), tactical decisions were distinguished from strategic decisions (61), and we use these terms as defined in the glossary. Following and elaborating on Conway (19) and Zadoks (145), four types of decisions are considered: (i) T, tactical short-term, during-season decisions; (ii) S, strategic short-term, between-season decisions (e.g., preplanting for annual crops); (iii) D, strategic long-term decisions (e.g., design of a breeding program, or development of IPM strategies within domains); and (iv), P: very long-term decisions pertaining to research prioritization.

Type 1 knowledge (**Table 1**) is a common approach to managing disease in a given field, season, and context. It typically leads to T-decisions (especially including pesticide use), which are based on assumptions on harmfulness of a given level of injury. When these assumptions are invalid or absent, it becomes a liability, inviting overprotection and sometimes underprotection (as in the case of barley powdery mildew in the 1960s; 44). Invalidity of such relationships is one of the main causes for mis- or overuse of pesticides, globally (56, 57, 62, 130, 131).

Table 1 Expected usefulness of crop loss knowledge for decisions

Types of crop loss knowledge ^a	T ^b	S ^b	D ^b	P ^b
1	++	—	—	—
2	+++	++	+	—
3	+	+	++	+
4	+	+	++	++
5	—	++	+++	+
6	—	+	+++	+++

^aType 1: Empirical environment-disease (environment-injury) relationships; type 2: empirical disease-crop loss (injury-damage) relationships; type 3: mechanistic simulation models—single disease; type 4: mechanistic simulation models—multiple diseases; type 5: risk zoning—disease prevalence; type 6: production situation-based models.

^bDecision categories: T: tactical—during season decisions; S: strategic—between season decisions (at the field level); D: strategic—domains for management; P: strategic—research priorities.

Note:— equals no expected usefulness; +, ++, +++ equal increasing degrees of assessed usefulness.

Quantification, analysis, and modeling crop loss at the field level, i.e., type 2 knowledge, as a core tool for tactical decisions, remains a major theme for phytopathological research. Type 2 knowledge is primarily useful for T- and S-decisions. Early modeling work, either analytical (58, 70, 73, 74, 122) or mechanistic (128), led to methodological and conceptual developments to mobilize field disease measurements, link them with crop growth, and model crop losses. An important by-product of these efforts was to define and standardize concepts, data formats (86, 87), and parameter formats (100, 9) for analysis and modeling of crop loss, and model performance assessment (121). Tactical decisions of an action or nonaction (e.g., treat/not treat) and the uncertainty of biological processes have recently been addressed using logistic regression (143) and probabilistic (41, 142, 78) approaches, respectively. We perceive the probabilistic approach as one key advance in the field (71), and this is addressed further.

Type 3 knowledge is exemplified by a number of mechanistic simulation models, where processes occurring at the target level of integration (typically, yield formation in a crop stand) are modeled through integration of processes that pertain to the nearest, lower level of integration (crop development and growth, and injury mechanisms). Because of this integration process, type 3 knowledge is primarily useful for D-decisions (e.g., designing ideotypes for host plant resistance or reduced crop vulnerability). This area has been pioneered by several research groups (32, 51, 127, 98), and several reviews of the approach are available (67, 100, 107, 148). Central to the approach is the dynamic linkage of (experimental) information at a level of integration to quantitatively project its consequences at the next level, and the matching of simulated and observed quantitative performances of the system (yield), thus providing a measure of the understanding gained on how the system behaved.

An individual crop stand is usually not exposed to a single organism but rather multiple

harmful organisms. Type 4 knowledge considers the effects of several harmful organisms (pathogens, pests, weeds) using experimental-empirical (20, 47, 50, 53, 75, 113–115, 133) or mechanistic-simulation approaches (49, 52, 96, 138, 139), or both. Because of its integrating nature and its scope over a range of harmful organisms, type 4 knowledge is particularly relevant to D-decisions as well as P-decisions. Although fewer in number, type 4 studies nevertheless demonstrate the relevance of knowledge of this kind at the field level in terms of improving both the understanding of complex systems and prospects for their management.

James (44) argued compellingly for the need of up-scaling information, i.e., type 5 approaches, from the field to higher scales. He emphasized field surveys as the basic means for acquiring information and expanding knowledge to higher scales (which need not necessarily be geographical only, 147a). This subject has been addressed in a number of reviews to which the reader is referred (122, 129, 137, 149). Type 5 knowledge is particularly relevant to S- and D-decisions. Up-scaling may be achieved using a number of approaches, using statistical (analysis and synthesis of field data) or modeling approaches (projection from available data into simulated possible scenarios; 130). Several modeling approaches have been developed to conceptualize (34, 132, 151) and assess (141) disease expansion at a number of scales. Three points are stressed here. First, epidemiological modeling provides the means to measure the risk of spread of a disease but not a measure of crop loss it may cause within a range of expansion. Conversely, field surveys and the associated analytical approaches provide a framework in which to characterize disease intensities and their relationships with attributes (including crop management) of the environments where injuries occur; but they usually do not generate crop loss data per se (109, 137). Third, up-scaling may involve scales other than geographical ones, because production situations need not be site-specific (147b). Upscaling may for instance enable injury profiles (which

may be caused by several harmful organisms) to be associated with given production situations (112). Linking the range of possible expansion of disease (whether in a given geographic area or in a specified production situation) to the possible consequences this may have on crop performances amounts to linking risk probability to risk magnitude (21, 108). This is the scope of type 6 knowledge, where information is combined to address simultaneously the linkages among disease severity and injury profiles, and the linkages between crop vulnerability within production situations to specified injury profiles. Type 6 knowledge enables D- and P-decisions to be addressed. Knowledge of this kind is still rudimentary; it has been addressed in very few cases in plant pathology, although it represents an avenue for important and new advances.

For decades, good phytopathology has dealt with controlling injury to prevent likely damage and possible loss. In many ways, however, having to control a disease when it is established in a crop is already a failure. Given that plant pathogens often are integral components of complex systems, better science should aim at management, rather than at removal, of (micro)organisms in (agro)ecosystems whose performances must be sustained. As knowledge evolves from levels 1 to 6 in **Table 1**, horizons for decisions are displaced toward longer terms. Short-term decisions are for near-emergencies; long-term decisions may enable sustainable ways to ensure disease management.

Using Crop Loss Knowledge for Tactical Decisions: Threshold for Control

The threshold theory (145) is the basis for the use of crop loss knowledge for tactical decisions. It evolved from field-entomological concepts and defines a damage threshold as the level of injury where the benefit of control (gain in yield or quality produce) just exceeds its cost. The theory further defines

warning and action thresholds as injury levels where control will be planned and conducted, respectively, in a timely manner to prevent the damage threshold from being reached. The damage threshold depends on the shape of damage functions, but losses also depend on other components, such as the value of the harvested product, and the cost of disease management (145).

One excellent and successful example of the application of this theory is the EPIPRE project (EPIdemiology, PREdiction, and PREvention) developed as early as the late 1970s in the Netherlands. EPIPRE makes use of crop loss knowledge of type 2, aimed at multiple T-decisions (**Table 1**). It is a field-specific warning system combining tactical, chemical control for six winter wheat diseases and aphid pests; EPIPRE calculations account for varietal characteristics, crop management, and (simple) farm economics (147); it converts farmers' own estimates for disease and pest injuries into injury levels at the end of a prediction period, relates these to damage thresholds, performs a cost/benefit analysis, and recommends a decision: Do not spray, spray, or wait (101, 147). EPIPRE started its first year with 300 participants. By the end of the project, it had reached about 30% of Dutch winter wheat growers, confidence in it had increased from 30% in 1981 to about 70% in 1985 (147), and versions of the system had been developed for use in Belgium, Switzerland, Sweden, France, and the United Kingdom.

The usefulness of the threshold theory may be weakened when too much uncertainty affects factors determining thresholds, such as market prices (145) or population dynamics (104). Uncertainty can successfully be incorporated in tactical decisions (104–106), however. Interestingly, damage thresholds derived from stochastic (i.e., uncertainty-bound) modeling of aphids and brown rust damage in winter wheat were lower than those derived from deterministic modeling. The sources of uncertainty were quantified, allowing knowledge gaps to be filled. A framework was

further developed incorporating the range of objectives of decision-makers (i.e., farmers), with attitudes ranging from risk-accepting to risk-adverse, and providing operational definitions of these objectives in terms of profitability and risk, which can be stochastically calculated.

The Bayesian decision theory has recently been applied to botanical epidemiology (41, 143) to develop predictors for epidemics. Combining the decision theory and the threshold theory may bring a powerful way to bridge information on crop losses and tactical decisions for disease management. Such a combination was applied to develop a decision tool for insecticide treatments against aphids based on *Barley yellow dwarf virus* (BYDV) damage threshold in winter barley (23).

Unlike EPIPRE, however, these developments address one pest at a time. Approaches that consider multiple pathosystems as the primary level of analysis may be keys to enhance the actual use of disease management tools. This is a new, scientifically challenging area for progress and application.

Using Crop Loss Knowledge for Short-Term Strategic Decisions at the Field Scale

Short-term strategic decisions include the choice of crop (which can be established as a mono-species or as an interspecific association) and cultivar, the choice of tillage, and the choice of genotype (single or several genotypes; 150). They also include other choices, such as the timing of crop establishment, the type of seed treatment used, the sowing density, and the spatial pattern of plants. The usefulness of crop loss knowledge for this type of decision is highlighted by two examples where type 2 information (**Table 1**) has been generated. Crop loss knowledge can provide a basis for the choice of a crop and cultivar, which is critical when no efficient tactical tool for management is available. This is well illustrated in the case of soilborne diseases, where quanti-

tative information of propagule density–yield loss relationships, linked with the quantification of propagules in the soil, can be used to decide before planting if the field is suitable for a given crop, or to select a tolerant cultivar when available. Such information has been generated, for example, in the case of pea root rot (91), *Verticillium* wilt of cotton (33, 94), and potato early dying disease (26). After the introduction of wheat stripe rust to New Zealand, a disease management program was developed to identify a combination of pest control tools to reduce yield losses. This program was based on the acquisition of knowledge on the dynamics of disease, the relationships between disease intensity and yield loss, and the effect of different management tools on yield loss. One output of this program was the recommendation for the use of seed treatment (29).

It is important here to stress that the use of crop loss information for short-term strategies is sometimes disregarded because of socioeconomic constraints. For example, the shift to monoculture (cultivation of a single species in annual or seasonal succession), caused by the need to specialize crop production, is often associated with disease increase over the succession of crops leading to increased crop losses. This is especially the case in many soilborne diseases, e.g., caused by pathogens of the genera *Verticillium* or *Fusarium* (93).

Using Crop Loss Knowledge for Long-Term Strategic Decisions

Crop loss knowledge, especially types 3 to 6 (**Table 1**), is useful to develop long-term strategic decisions that enable decision makers to identify or anticipate where IPM is needed for a given (possibly emerging) disease, and when (or under which production context) the disease does not represent a threat to a crop.

Production situations have been used to develop this type of knowledge in the case of rice in South-East Asia, and this example

is developed in a following section. A geographical basis has, however, more commonly been used, illustrated here by a few examples. Disease risk zoning was performed for stripe rust in the United States in the late 1960s, where multisite field trials with major cultivars grown in the region provided estimates of yield losses caused by stripe rust. This information allowed assessment of the importance of the disease, the efficiency of its management, and research priorities (66). Another example is the modeling of wheat yield losses caused by foliar diseases in China in a range of weather scenarios, which provided policy makers with a tool to manage diseases (68). Exploratory analyses based on weather databases, coupled with a simulation model of soybean rust epidemics and a simulator of soybean growth, allowed analysis of potential yield losses a decade before its appearance in mainland U.S.A. (141).

As early as 1977, C.E. Main (76) stated, "Yield loss information should have heuristic value for host plant resistance and disease tolerance." Although yield loss estimates seldom represent relevant, practical criteria in breeding for resistance or tolerance (95), a good understanding of the mechanisms that determine yield losses can help in characterizing attributes for tolerance and resistance in cultivars, and assist breeding programs. A good example is provided by a study on potato late blight, where simulations of epidemics and of their impact on potato growth and yield enabled the ecophysiological traits for breeding that would increase the tolerance of potato against the disease to be determined (134).

Using Crop Loss Knowledge to Set Research Priorities

In a context where funding for research organizations is limited, priority setting is necessary to optimize research initiatives in plant protection for maximum efficacy, with a time frame of at least 10 to 15 years (22). Priority setting requires identifying diseases that should be targeted for research in the contexts

of present and future production scenarios. Simulation models that incorporate processes and interactions involved in the behavior of the environment-injury-crop system represent powerful tools with which to undertake such analyses. The reliability of results depends heavily on the degree of understanding of the system. Two examples of such analyses are given here to illustrate how yield loss information can help in research prioritization for pest management (information types 5 and 6; **Table 1**). A first example is the modeling of yield losses at large scale for multiple pests of a given crop. Outputs can provide a hierarchy of pests in terms of importance (reflected by the level of yield losses they individually cause), and thus identify those pests for which priority should be given for research (breeding for resistance/tolerance, IPM; 22). Estimates of rice yield losses caused by pests in South and South-East Asia as predictors of current and future production have recently provided a basis for research priorities for rice pest management in this region. This example is described in details in the following section. A second example is the modeling of yield losses due to diseases under future scenarios of climate. Climate change may imply changes in geographical distribution of diseases and also changes in the response of crops to injuries. The effects involved and research conducted on this issue have been reviewed by Coakley et al. (17). Recent developments are also discussed by Garrett in this issue (27). Experimental work and model development have so far been limited, and need to be strengthened to help in developing research agendas and efficient disease management strategies in the context of climate change in the coming decades.

Disease-Induced Toxin Accumulation as an Example of Qualitative Losses

The accumulation of toxins in harvests caused by plant pathogens is an example of primary, direct crop loss (149), where the quality of the

product is reduced. This type of crop loss is as ancient as agriculture, may have grave consequences on human and farm animal health, and has been difficult to quantify. There has been progress recently, especially related to *Fusarium* diseases of cereal grains (95a, 95b). Such crop losses are particularly prevalent in the tropics and subtropics, and are well exemplified by the classical problem of production of aflatoxins by *Aspergillus flavus* in many agricultural products, especially peanuts (97), and of fumonisin accumulation in maize infected by *Fusarium moniliforme* (30). In *Fusarium* head blight of wheat, there are three components to the damage: a reduction in yield (yield loss), a reduction in grain milling quality, and an increase in concentrations of toxins. The injury (disease intensity)-toxin relationship can be very complex, as it depends on the fungal species, its strain, host plant resistance, disease dynamics, and numerous environmental factors (3, 11, 79, 117). A recent review (11) articulates why damage components need to be separately quantified in relation to injury, and how relationships between damage components need quantitative analysis.

EXEMPLIFICATION OF CROP LOSS KNOWLEDGE: LOWLAND RICE IN TROPICAL ASIA

The diversity of tropical rice pests is as broad as the diversity of rice production environments (124). Today, pest management in rice relies primarily on strategic short-term (S) decisions, and is grounded in the use of resistant cultivars by farmers (65, 80). Research on brown plant hopper (*Nilaparvata lugens*) demonstrated on the world's most important agricultural commodity that pesticide use was not only ineffective, but actually the cause for crop losses (55, 125) in this case, because of the detrimental effects of pesticide on nontarget, beneficial organisms. Recent work also has shown that management of rice tungro disease—whose vectors had for decades been uselessly targeted by insecticide applications (80)—could safely rely on the deployment

of (incomplete) resistant germplasm in synchronous plantings (8). Tactical (T) decisions nowadays are limited to a small number of harmful agents, including *Rhizoctonia solani* and stem borers, and often involve crop husbandry (especially water management) rather than pesticides. Key issues nevertheless remain (81); these pertain to improving crop husbandry and so indirectly to disease management, how best to deploy the available host plant resistance genes—a nonrenewable resource, and how to design or incorporate in the rice genome new genes for resistance.

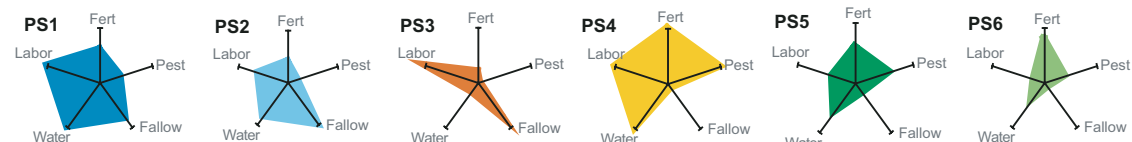
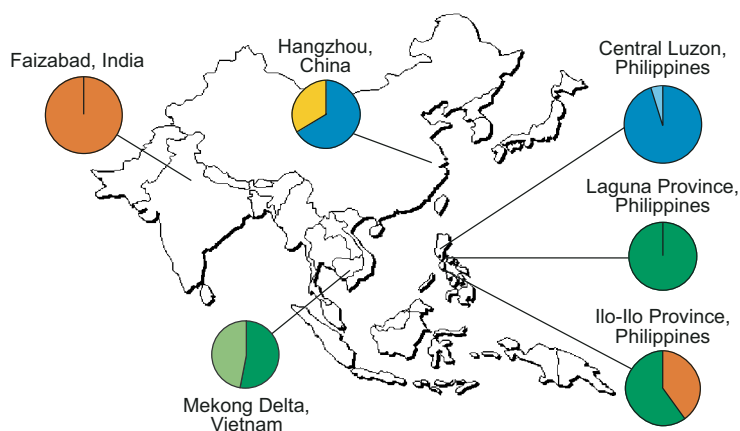
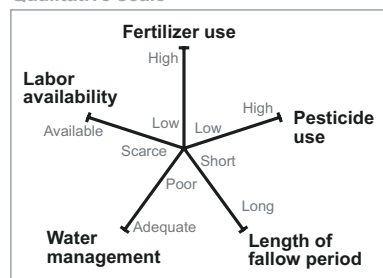
Characterization of crop management and injuries due to pathogens, insects, and weeds (injury profiles) is a key entry point to identify possible linkages among components of complex agroecosystems. One major result of a study conducted at several sites across tropical Asia over several years (112) was the demonstration that (i) there are a few broad patterns of crop management which are found across sites (**Figure 2a**); (ii) a limited number of injury profiles exist, which are also shared among sites (**Figure 2b**); (iii) and there is a strong association between patterns of crop management and injury profiles. These results offer grounds to define recommendation domains for IPM (D) and research priorities (P).

Injury profiles were dominated by stem rot and sheath blight (IP1); by bacterial leaf blight, plant hoppers, and leaf folders (IP2); and by sheath rot, brown spot, leaf blast, and neck blast (IP3). IP1 corresponds to higher (mineral) fertilizer input, long fallow periods, lower pesticide use, and good water management in (mostly) transplanted rice crops of a rice-rice rotation (PS1). IP2 corresponds to direct-seeded crops in a continuous rice-rice rotation with low (PS5), or high (PS6), fertilizer inputs, medium-low pesticide inputs, and poor water management (PS5). IP3 corresponds to labor intensive crops (hand weeding and transplanting) with low fertilizer and pesticide inputs in very diverse rotation systems, where water supply is uncertain (PS3).

Survey data do not provide direct measurement of damage, except where there are

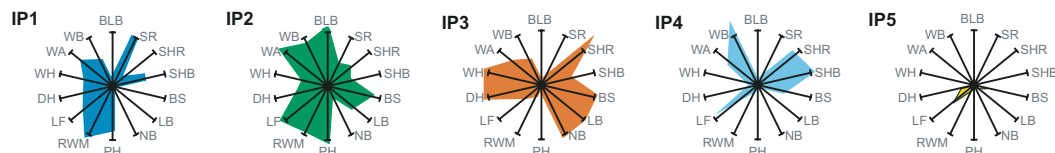
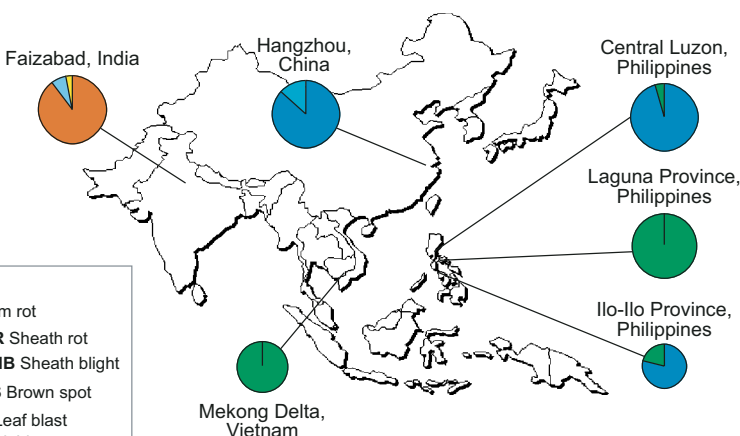
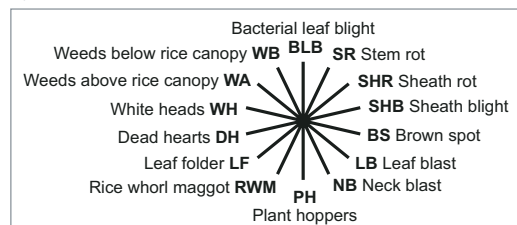
a Production situation (PS)

Qualitative scale



b Injury profile (IP)

Quantitative scale



total losses linearly linked to observed injury over an area. The injuries caused by 11 rice pests were manipulated in a series of linked field experiments, where the attainable yield (2 to 11 t.ha⁻¹) was varied by altering levels of production factors in different seasons and years (111). Statistical multivariate analyses demonstrated interactions between injuries and attainable yield, i.e., indicated varying levels of yield vulnerability depending on crop management patterns and injury profiles.

RICEPEST, a simulation model of rice yield losses, enables the simultaneous handling of production situations and injury profiles ('drivers'), as well as the modeling of management options and strategies (138–140). Overall, simulations indicate that 120 to 200 × 10⁶ tons of grain yield are lost yearly to pests over the 87 × 10⁶ ha of lowland rice in tropical Asia. This also is a measure of the potential gain that future pest management strategies could achieve, if deployed. Operational definitions for management efficacy (injury reduction) and management efficiency (yield gain) were developed. This modeling work led to classification of rice pests into two broad research priority-setting categories (140), with respect to simulated damage and management efficiency: pests for which effective management tools need to be developed (weeds, sheath blight, and brown spot), and pests for which the efficiency of current management methods must be maintained (leaf blast, neck blast, bacterial leaf blight, and brown plant-hoppers).

METHODOLOGICAL PERSPECTIVES

Linking Surveys, Experiments, and Modeling

Linking surveys and experiments to assess crop losses was advocated decades ago (44, 64). Surveys provide information on the occurrence of diseases, whereas yield loss experiments provide information on the impact of diseases in terms of (quantitative) damage. Their combination thus generates a strong basis for yield loss assessment. An operational way to link the two types of information is derived from risk analysis (108), where injury prevalence represents the probability of an event to occur (injury to take place), and damage functions correspond to the magnitude of the outcome of the event (yield loss caused by injury). Risk (108) is measured by the product of probability by magnitude, and represents the overall expected yield loss for a given pest in a given system (110). Surveys allow estimation of the prevalence of injuries in the area surveyed. Estimation of yield loss based on the level of injury (damage function) can be achieved by two main ways: (i) quantification of the relationship between injury level and yield loss from field experiments where injury levels are manipulated, assessed, and the corresponding yield loss measured (10, 44, 116, 145); (ii) quantification of the injury mechanisms and scaling-up at the field level by simulation modeling, using agrophysiological models with coupling points for injury mechanisms (100, 107). If the first, empirical

Figure 2

Production situations and injury profiles of lowland rice in tropical Asia. Geographical patterns of production situations (PS; *a*) and injury profiles (IP; *b*) of lowland rice fields in tropical Asia, determined from a characterization study carried out on 456 rice fields. The star-shaped graphs represent the characteristics of PSs and IPs. For example, PS1 is characterized by medium-high fertilizer inputs, good water management, long fallow period preceding rice crop, good labor availability, and low pesticide use; IP1 is dominated by a few injuries: weeds, sheath blight, stem rot, stem borers, and defoliators. Circled graphs represent the proportions of fields pertaining to a given PS or IP in a subregion. For example, PS5 is represented in Mekong Delta, Laguna Province, and Ilo-Ilo Province, and rice fields grown in Mekong Delta pertain to PS5 or PS6; IP1 is represented in Hangzhou, Central Luzon, and Ilo-Ilo Province, and rice fields in Ilo-Ilo Province are affected by IP1 and IP2.

approach is chosen, its potential for extrapolation will be constrained by its experimental basis, which will have to encompass the range of production situations it aims to address. On the other hand, mechanistic, dynamic simulation modeling is a tool of extrapolation per se, with the potential to address current as well as future environmental contexts that do not (yet) exist, and that cannot be addressed experimentally, through scenario analyses. This second approach, however, must be grounded on field-experimental work, commensurate with the diversity of production situations of injury profiles, and the model evaluation effort.

Recent work has shown the power of using meta-analyses to synthesize data originating from different studies in plant pathology (95b, 102). The use of such approaches for the combined analysis of survey data or yield loss experimental studies are bound to greatly enhance the accuracy and scope of results previously obtained.

The combination of survey, experimental, and modeling approaches is a powerful tool to analyze yield losses at large scales, as shown above where the example of rice yield losses was outlined. Few analyses have been conducted using this type of framework, perhaps due to the heavy experimental load required to achieve results. When large-scale, complex (agro)ecosystems are considered, linked approaches of this kind, leading to such information, are nevertheless needed to develop strategies for rational disease management.

So far, the bulk of these methods has been used to address yield losses only, and not quality losses, where it should lead to very significant progress in disease management.

Spatial Aspects

Although spatial aspects of epidemics have received wide attention in the past 20 years, much less work has addressed the impact of the spatial structure of injuries on the resulting yield losses (36).

One mechanism by which spatial pattern of injury influences yield losses is the physiological compensation that can occur between neighboring units (leaves, roots, tillers, plants, trees) that differ in injury level. For example, an increase of aggregation of injured plants would increase yield losses, because physiological compensation between plants would be reduced (44, 149). This mechanism has been addressed through the use of graphical frameworks (40) and reciprocal equations that relate crop yield to plant population density (37).

The estimation of yield loss can also be affected by injury aggregation when the associated damage function is nonlinear, because aggregation corresponds to changes in the frequency distribution of injury levels. Depending on the sign of the second derivative of the damage function, a yield loss value derived from the injury level averaged over samples will over- or underestimate the true value when injury aggregation increases, as compared with yield loss estimated from weighted average over the expected yield loss of each sample (25, 38, 39).

Only a limited number of studies that have investigated experimentally relationships between spatial pattern of injuries and yield loss have been conducted (39), thus hampering the forwarding and testing of hypotheses on these relationships. Injury aggregation is seldom accounted for in the development of damage functions (injury-yield loss relationships). There is a need to investigate further these relationships in experimental, analytical, and modeling terms to assess the extent of their implications on damage functions, and to optimize sampling schemes used to assess injury levels. A possible way to better understand these relationships would be to use spatialized agrophysiological simulation models, including the spatial patterns of injury and mechanisms for physiological compensation. Potential entry points for this type of approach were offered by Guttierrez et al. (33), who incorporated compensation mechanisms when modeling the effect of *Verticillium* wilt on cotton,

and by Ferrandino (24), who included the spatial structure of injury when analyzing potato crop losses caused by late blight.

FINAL REMARKS AND PERSPECTIVES

In **Table 1**, we linked crop loss knowledge with its expected use, expressed as types of decision for disease (pest) management. Linking decision types to attributes of usefulness (**Table 2**) is a further step for assessing crop loss knowledge. A number of frameworks could be used to meet that aim (see 1). A prime use of crop loss knowledge is a reduction of yield losses (*l*) and improved yield quality (*q*). Processing and use of the information may also translate into reduced management costs (*c*); reduced use of chemical pesticides (*cb*); of other agricultural inputs (*i*; including irrigation water, fertilizer, energy, labor); and maintenance of natural resources (*nr*; including biodiversity, topsoil, water). In many production situations, especially in subsistence agriculture contexts, stabilized (rather than maximized) yield (*sy*) is a key objective. In other production situations where the produce is directed to specific markets, stabilized quality (*sq*) is the aim. Overall, crop loss knowledge can and should contribute to stabilized systems performances (*ss*) and societal benefits (*sb*). **Table 2** outlines predictable relationships between decision types and criteria for impact from crop loss knowledge. Benefits pertaining to longer time-steps or to broader social implications are associated with decisions shifting from T (tactical, during season), to S (strategic, between season), to D (strategic, domains for management), and P (strategic, priorities).

Conventional dimensions for measuring (quantitative) damage are biomass [dimension: (M)], or biomass per production area [(M.L⁻²)]. Considering the untaken harvest (89) enables accounting for loss of labor time [T], of energy [(M.L².T⁻²); e.g., fertilizer, traction], financial (\$) as well as of environmental resources (topsoil, water) invested into

Table 2 Expected relationships among decision types (columns) and impact from crop loss knowledge on agricultural systems, environments, and societies

Criteria for impact of crop loss knowledge ^a	T ^b	S ^b	D ^b	P ^b
<i>l</i>	+++	+++	++	+
<i>q</i>	+++	+++	++	+
<i>c</i>	+	+++	+++	++
<i>cb</i>	+	+++	++	+++
<i>i</i>	+	++	+++	+++
<i>nr</i>	+	++	+++	+++
<i>sy</i>	++	++	++	+++
<i>sq</i>	++	++	++	+++
<i>ss</i>	+	++	+++	+++
<i>sb</i>	+	++	+++	+++

^aCriteria for impact of crop loss knowledge: *l*, reduced yield losses (Ya–Y), where Ya: attainable yield, Y: actual yield; *q*, improved yield quality; *c*, reduced management costs; *cb*, reduced use of chemical pesticides; *i*, reduced use of other inputs (including irrigation water, fertilizer, energy, labor); *nr*, reduced exploitation/depletion of natural resources; *sy*, stabilized yields; *sq*, stabilized quality; *ss*, stabilized systems performances; *sb*, benefit to society.

^bDecision categories: T, tactical–during season decisions; S, strategic–between season decisions (at the field level); D, strategic–domains for management; P, strategic–research priorities (see **Table 1**).

agricultural production. One main point this article emphasizes is the validity of the very concept of crop loss as encompassing yield and quality reductions (149). The cascade of relationships between injury, damage, and loss (145) offers both challenging scientific avenues and prospects for advancement. Shifting the dimension of crop loss away from its conventional dimensions allows links to be established between crop loss and (economical) loss [dimension: (\$)], which in turn can be related to gross (\$), or net (\$. \$⁻¹) return. Shifting the dimensionality of damage also leads to accounting for costs other than direct ones, and so to better decisions. Such links are possible; they certainly require expertise outside the conventional phytopathological area; and successes have been reported, which we briefly reviewed here, notably through modeling approaches. With a solid field and

experimental basis for continuing progress, the injury–damage–loss paradigm brings phytopathological research into the broader framework of ecological sciences and operational research, of systems science and systems management.

The statement that “our means of measuring the severity of symptoms [that plant pathogens can cause] is often subjective and qualitative rather than objective and quantitative” (119) sadly echoes in 2005 the call for quantitative data made by Lyman (69) nearly a century ago. Plant pathology, however, offers the scientific community expertise to quantitatively assess the impact of available, or yet-to-come, disease management tools for efficient and integrated crop production, as well

as to identify research priorities. The concept of damage (crop loss) encompasses reductions in quantity and in quality, which are both amenable to quantitative analysis, even if the former has been the subject of far more contributions than the latter. Whereas qualitative reductions in the performances of crops are now dominant concerns in developed countries, reductions in both yield quantity and quality are critical issues in the developing world, where 1300 million people live on less than \$1 a day, and 800 million do not have adequate food (118). Increased efficiency of performance in agroecosystems can contribute to the solution of the global issues of food security and of quality of agricultural products, including food safety (35).

SUMMARY POINTS

1. In this paper, we propose that the hierarchy of injury, damage (crop loss, qualitative and/or quantitative), and loss is a valid framework to quantify, analyze, model, and manage damage.
2. This hierarchy enables phytopathological research to be considered in the broader framework of ecological sciences and operational research.
3. This framework is valid in the case of quantitative as well as in qualitative losses. Application of this framework to qualitative damage opens the way to novel research, better problem identification, and thus improved management.
4. Progress achieved by the application of Bayesian decision theory to decision-making and modeling uncertainty of decision outcomes has focused on both tactical and (mostly) single injury targets. A remaining challenge is to expand this approach to injury profiles (multiple injuries) and to longer-term decisions (strategic-short term or strategic long-term).
5. The shift from yield loss caused by diseases (pests) to yield gain from management has been made possible by modeling the efficiency of different disease management tools. This advance allows analysis of current and exploration of future options for management of multiple pathosystems.
6. The translation of damage outside conventional dimensions (biomass, biomass per unit area) opens perspectives to consider the performance of agrosystems more fully, and to objectively assess novel management strategies.
7. Challenges remain in the field of damage quantification, analysis, and modeling, especially for qualitative losses. Many of these derive from changes in perspectives or shifts in dimensions, which link phytopathological research with other scientific fields and invite new methodologies.

LITERATURE CITED

1. Antle JM, Capalbo SM. 1995. Measurement and evaluation of the impacts of agricultural chemical use: a framework for analysis. In *Impact of Pesticides on Farmer Health and the Rice Environment*, ed. PL Pingali, P Roger, pp. 23–57. Norwell MA: IRRI-Kluwer
2. Ayres PG. 1981. *Effects of Disease on the Physiology of the Growing Plant*. Cambridge Univ. Press: Cambridge
3. Bai GH, Plattner R, Desjardins AE, Kolb F. 2001. Resistance to *Fusarium* head blight and deoxynivalenol accumulation in wheat. *Plant Breed.* 120:1–6
4. Boote KJ, Jones JW, Mishoe JW, Berger RD. 1983. Coupling pests to crop growth simulators to predict yield reductions. *Phytopathology* 73:1581–87
5. Bourke PMA. 1964. Emergence of potato late blight, 1843–45. *Nature* 203:805–8
6. Breman H, De Wit CT. 1983. Rangeland productivity and exploitation in the Sahel. *Science* 221:1341–47
7. Butt DJ, Royle DJ. 1980. The importance of terms and definitions for a conceptually unified epidemiology. In *Comparative Epidemiology. A Tool for Better Disease Management*, ed. J Palti, J Kranz, pp. 29–45. Wageningen: Pudoc
8. Cabunagan RC, Castilla N, Coloquio EL, Tiongco ER, Truong XH, et al. 2001. Synchrony of planting and proportion of susceptible varieties affect rice tungro disease epidemics in the Philippines. *Crop Prot.* 20:499–510
9. Campbell LC, Madden LV. 1990. *Introduction to Plant Disease Epidemiology*. New York: Wiley
10. Campbell LC, Madden LV. 1990. Assessment of plant diseases and losses. See Ref. 9, pp. 393–422
11. Champeil A, Doré T, Fourbet JF. 2004. *Fusarium* head blight: epidemiological origin of the effects of cultural practices on head blight attacks and the production of mycotoxins by *Fusarium* in wheat grains. *Plant Sci.* 166:1389–415
12. Chant DA. 1964. Strategies and tactics of insect control. *Can. Entomol.* 96:182–201
13. Chester KS. 1950. Plant disease losses: their appraisal and interpretation. *Plant Dis. Rep. Suppl.* 193:190–362
14. Chiarappa L. 1971. *Crop Loss Assessment Methods: FAO Manual on the Evaluation and Prevention of Losses by Pests, Diseases and Weeds*. Farnham Royal: FAO/CAB
15. Chiarappa L, ed. 1981. *Crop Loss Assessment Methods. Supplement N° 3*. FAO/CAB, Farnham, England. 123 pp.
- 15a. Chiarappa L. 1981. Establishing the crop loss profile. See Ref. 15, pp. 21–24.
16. Church BM. 1971. The place of sample survey in crop loss estimation. See Ref. 14, pp. 2.2/1–2.2/12
17. Coakley SM, Scherm H, Chakraborty S. 1999. Climate change and plant disease management. *Annu. Rev. Phytopathol.* 37:399–426
18. Cole DL. 1982. Interaction between *Cercospora arachidicola* and *Phoma arachidicola* and their effects on defoliation and kernel yield of groundnut. *Plant Pathol.* 31:355–62
19. Conway GR. 1984. *Pest and Pathogen Control: Strategic, Tactical, and Policy Models*. Chichester, UK: Wiley. 504 pp.
20. de Jesus WC Jr, Vale FXR, Coelho RR, Zambolim L, Costa LC, Bergamin Filho A. 2001. Effects of angular leaf spot and rust on yield loss of *Phaseolus vulgaris*. *Phytopathology* 91:1045–53
21. de Jong MD, Scheepens PC, Zadoks JC. 1990. Risk analysis for biological control: a Dutch case study in biocontrol of *Prunus serotinia* by the fungus *Chondrostereum purpureum*. *Plant Dis.* 74:189–94

22. Evenson RE, Herdt RW, Hossain M, eds. 1996. *Rice Research in Asia: Progress and Priorities*. Wallingford, UK: CAB Int./Los Baños, Philipp.: IRRI
23. Fabre F, Dedryver CA, Leterrier JL, Plantegenest M. 2003. Aphid abundance on cereals in autumn predicts yield losses caused by barley yellow dwarf virus. *Phytopathology* 93:1217-22
24. Ferrandino FJ. 1989. Spatial and temporal variation of a defoliating plant disease and reduction in yield. *Agric. For. Meteorol.* 47:273-89
25. Ferrandino FJ. 1989. A distribution-free method for estimating the effect of aggregated plant damage on crop yield. *Phytopathology* 79:1229-32
26. Franc L, Madden LV, Rowe RC, Riedel RM. 1987. Potato yield loss prediction and discrimination using preplant population densities of *Verticillium dahliae* and *Pratylenchus penetrans*. *Phytopathology* 77:579-84
27. Garrett KA. 2006. The impact of global climate change on plant diseases. *Annu. Rev. Phytopathol.* 44:000-00
28. Gaunt RE. 1995. The relationship between plant disease severity and yield. *Annu. Rev. Phytopathol.* 33:119-44
29. Gaunt RE, Cole MJ. 1987. A disease management response to the introduction of wheat stripe rust to New Zealand. *Plant Dis.* 71:102-5
30. Gelderblom WCA, Jaskiewicz K, Marasas WFO, Thiel PG, Vleggaar R, Kriek NPJ. 1988. Fumonisin—novel mycotoxins with cancer promoting activity produced by *Fusarium moniliforme*. *Appl. Environ. Microbiol.* 54:1806-11
31. Gomes Carneiro SMTP, Amorim L, Bergamin Filho A, Hau B, Bianchini A. 2000. Dinâmica de área foliar, desfolha e variáveis de área foliar sadia em feijoeiros com infecções isoladas e conjuntas de *Phaeoisariopsis griseola* e *Colletotrichum lindemuthianum*. *Summa Phytopathol.* 26:406-12
32. Gutierrez AP, Falcan LA, Loew W, Leipzig PA, Van de Bosch R. 1975. An analysis of cotton production in California: a model of Accala cotton and the effect of defoliators on its yield. *Environ. Entomol.* 4:125-36
33. Gutierrez AP, DeVay JE, Pullman GS, Friebertshauser GE. 1983. A model of Verticillium wilt in relation to cotton growth and development. *Phytopathology* 73:89-95
34. Heesterbeek JAP, Zadoks JC. 1987. Modelling pandemics of quarantine pests and diseases: problems and perspectives. *Crop Prot.* 6:211-21
35. Herdt RW. 2006. Priorities for plant disease epidemiology and developing world food security. In *Plant Disease Epidemiology: Facing Challenges of the 21st Century*, ed. M Cooke, S Savary. *Eur. J. Plant Pathol.* Spec. Issue. In press
36. Hughes G. 1988. Models of crop growth. *Nature* 332:16
37. Hughes G. 1988. Modelling the effect of spatially heterogeneous pest injury on crop yields. *Crop Res. (Hortic. Res.)* 28:137-44
38. Hughes G. 1988. Spatial heterogeneity in crop loss assessment models. *Phytopathology* 78:883-84
39. Hughes G. 1996. Incorporating spatial pattern of harmful organisms into crop loss models. *Crop Prot.* 15:407-21
40. Hughes G, McKinlay RG. 1988. Spatial heterogeneity in yield-pest relationships for crop loss assessment. *Ecol. Model.* 41:67-73
41. Hughes G, McRoberts N, Burnett FJ. 1999. Decision-making and diagnosis in disease management. *Plant Pathol.* 48:147-53
42. James WC. 1969. An illustrated series of assessment keys for plant diseases, their preparation and usage. *Can. Plant Dis. Surv.* 51:39-65

43. James WC. 1969. A survey of foliar diseases in spring barley in England and Wales in 1967. *Ann. Appl. Biol.* 63:253–63
44. James WC. 1974. Assessment of plant diseases and losses. *Annu. Rev. Phytopathol.* 12:27–48
45. James WC 2003. *Global Status of Commercialized Transgenic Crops: 2002. Feature: Bt Maize*. ISAAA Briefs no. 29. Ithaca, NY: ISAAA
46. James WC, Teng PS. 1979. The quantification of production constraints associated with plant diseases. In *Advances in Applied Biology*, ed. TH Coaker, 3:201–267. London: Academic
47. Johnson KB. 1986. *Systems analysis and modeling of yield loss in potato caused by interacting populations of early blight, Verticillium wilt, and the potato leafhopper*. PhD thesis. Univ. Minn., 201 pp.
48. Johnson KB. 1987. Defoliation, disease, and growth: a reply. *Phytopathology* 77:1495–97
49. Johnson KB. 1992. Evaluation of a mechanistic model that describes potato crop losses caused by multiple pests. *Phytopathology* 82:363–69
50. Johnson KB, Radcliffe EB, Teng PS. 1986. Effects of interacting populations of *Alternaria solani*, *Verticillium dahliae*, and the potato leafhopper (*Empoasca fabae*) on potato yield. *Phytopathology* 76:1046–52
51. Johnson KB, Teng PS. 1990. Coupling a disease progress model for early blight to a model of potato growth. *Phytopathology* 80:416–25
52. Johnson KB, Teng PS, Radcliffe EB. 1987. Coupling feeding effects of potato leafhoppers, *Empoasca fabae* (Homoptera: Cicadellidae) nymphs to a model of potato growth. *Environ. Entomol.* 16:250–58
53. Johnson KB, Teng PS, Radcliffe EB. 1987. Analysis of potato foliage losses caused by interacting infestations of early blight, verticillium wilt, and potato leafhopper, and the relationship to yield. *Z. Pflanzenkr. Pflanzenschutz* 94:22–33
54. Deleted in proof
55. Kenmore PE, Cariño FO, Perez CA, Dyck VA, Gutierrez AP. 1984. Population regulation of the rice brown plant hopper (*Nilaparvata lugens* Stal) within rice fields in the Philippines. *J. Plant Prot. Trop.* 1:19–38
56. Kenmore PE, Heong KL, Putter CAJ. 1984. Political, social and perceptual aspects of integrated pest management programs. *Proc. Semin. Integr. Pest Manag. Malaysia*, pp. 47–69. Kuala Lumpur: MAPPS
57. Kenmore PE, Litsinger JA, Bandong JP, Santiago AC, Salac MM. 1987. Philippine rice farmers and insecticides: thirty years of growing dependency and new options for change. In *Management of Pests and Pesticides: Farmers' Perceptions and Practices*, ed. J Tait, B Napompeth, pp. 98–108. Boulder: Westview
58. Kim CH, MacKenzie DR. 1987. Empirical models for predicting yield loss caused by a single disease. See Ref. 123, pp. 126–32
59. King JE. 1977. Surveys of foliar diseases of spring barley in England and Wales. *Plant Pathol.* 26:21–29
60. King JE. 1977. Surveys of diseases of winter wheat in England and Wales, 1970–1975. *Plant Pathol.* 26:8–20
61. Kogan M. 1998. Integrated pest management: historical perspectives and contemporary developments. *Annu. Rev. Entomol.* 43:243–70
62. Kortenhoff A. 1993. Developments in the use of pesticides. In *Modern Crop Protection : Development and Perspectives*, ed. JC Zadoks, pp. 3–10. Wageningen: Wageningen Pers
63. Kropff MJ, Teng PS, Rabbinge R. 1995. The challenge of linking pest and crop models. *Agric. Syst.* 49:413–34

64. Large EC. 1966. Measuring plant disease. *Annu. Rev. Phytopathol.* 4:9–28
65. Leung H, Fan JX, Zhu Y, Chen H, Revilla-Molina I, et al. 2003. Using genetic diversity to achieve sustainable rice disease management. *Plant Dis.* 87:1156–69
66. Line RF. 2002. Stripe rust of wheat and barley in North America: a retrospective historical review. *Annu. Rev. Phytopathol.* 40:75–118
67. Loomis RS, Adams SS. 1983. Integrative analysis of host-pathogen relations. *Annu. Rev. Phytopathol.* 21:341–62
68. Luo Y, Shen ZR, Zeng SM. 1993. Risk analysis of disease epidemics on wheat by simulation studies. *Agric. Syst.* 43:67–89
69. Lyman GR. 1918. The relation of phytopathologists to plant disease survey work. *Phytopathology* 8:219–28
70. Madden LV. 1983. Measuring and modeling crop loss at the field level. *Phytopathology* 73:1591–96
71. Madden LV. 2006. Botanical epidemiology: some key advances and its continuing role in disease management. See Ref. 35
72. Madden LV, Nutter FW. 1995. Modeling crop losses at the field scale. *Can. J. Plant Pathol.* 17:124–37
73. Madden LV, Pennypacker SP, Antle CE, Kingsolver, CH. 1981. A loss model for crops. *Phytopathology* 71:685–89
74. Madden LV, Pennypacker SP, Kingsolver CH. 1981. A comparison of crop loss models. *Phytopathol. Z.* 101:196–201
75. Madariaga RB, Scharen AL. 1986. Interaction of *Puccinia striiformis* and *Mycosphaerella graminicola* on wheat. *Plant Dis.* 70:651–54
76. Main CE. 1977. Crop destruction—the raison d'être of plant pathology. In *Plant Disease—An Advanced Treatise. Vol. 1. How Disease Is Managed*, ed. JG Horsfall, EB Cowling, pp. 55–78. New York: Academic
77. McNew GL, 1960. The nature, origin and evolution of parasitism. In *Plant Pathology—An Advanced Treatise*, Vol. 2, ed. JG Horsfall, AE Dimond, pp. 20–69. New York: Academic
78. McRoberts N, Hughes G, Savary S. 2003. Integrated approaches to understanding and control of diseases and pests in field crops. *Aust. Plant Pathol.* 32:167–80
79. Mesterházy Á, Bartók T, Lamper C. 2003. Influence of wheat cultivar, species of *Fusarium*, and isolate aggressiveness on the efficacy of fungicides for control of Fusarium head blight. *Plant Dis.* 87:1107–15
80. Mew TW. 1991. Disease management in rice. In *CRC Handbook of Pest Management in Agriculture*, ed. D Pimentel, 3:279–299. Boca Raton: CRC Press. 2nd ed.
81. Mew TW, Leung H, Savary S, Vera Cruz CM, Leach JE. 2004. Looking ahead in rice disease research and management. *Crit. Rev. Plant Sci.* 23:103–27
82. Monteith JL. 1972. *Principles of Environmental Physics*. London: Arnold
83. Nutter FW Jr, Guan J, Gotlieb AR, Rhodes LH, Grau CR, Sulc RM, 2002. Quantifying alfalfa yield losses caused by foliar diseases in Iowa, Ohio, Wisconsin, and Vermont. *Plant Dis.* 86:269–77
84. Nutter FW Jr, Littrell RH. 1996. Relationships between defoliation, canopy reflectance and pod yield in the peanut late-leaf spot pathosystem. *Crop Prot.* 15:135–42
85. Nutter FW Jr, Schultz PM. 1995. Improving the accuracy and precision of disease assessments: selection of methods and use of computer-aided training programs. *Can. J. Plant Pathol.* 17:174–84
86. Nutter FW, Teng PS, Royer MH. 1993. Terms and concepts for yield, crop loss, and disease thresholds. *Plant Dis.* 77:211–15

87. Nutter FW, Teng PS, Shokes FM. 1991. Disease assessment terms and concepts. *Plant Dis.* 75:1187–88
88. Oerke EC, Dehne HW, Schönbeck F, Weber A. 1994. *Crop Production and Crop Protection. Estimated Losses in Major Food and Cash Crops*. Amsterdam: Elsevier
89. Ordish G. 1952. *Untaken Harvest*. London: Constable
90. Ou SH. 1985. *Rice Diseases*. Farnham Royal, UK: CAB Int. 2nd ed.
91. Oyarzeun PJ. 1993. Bioassay to assess root rot in pea and effect of root rot on yield. *Neth. J. Plant Pathol.* 99:61–75
92. Padwick GW. 1956. Losses caused by plant diseases in the tropics. *Comm. Mycol. Inst. Phytopathol. Pap. N°1*. Kew. 60 pp.
93. Palti J. 1981. *Cultural Practices and Infectious Crop Diseases*. Berlin: Springer-Verlag
94. Paplomatas EJ, Bassett DM, Broome JC, DeVay JE. 1992. Incidence of *Verticillium* wilt and yield losses of cotton cultivars (*Gossypium hirsutum*) based on soil inoculum density of *Verticillium dahliae*. *Phytopathology* 82:1417–20
95. Parlevliet JE. 1981. Crop loss assessment as an aid in screening for resistance and tolerance. See Ref. 15, pp. 111–14
- 95a. Paul PA, El-Allaf SM, Lipps PE, Madden LV. 2005. Relationships between incidence and severity of Fusarium head blight on winter wheat in Ohio. *Phytopathology* 95:1049–60
- 95b. Paul PA, Lipps PE, Madden LV. 2005. Relationship of visual estimates of Fusarium head blight intensity and deoxynivalenol accumulation in harvested wheat grain: a meta-analysis. *Phytopathology* 95:1225–36
96. Pinnschmidt H, Batchelor WD, Teng PS. 1995. Simulation of multiple species pest damage in rice using CERES-rice. *Agric. Syst.* 48:193–222
97. Porter DM, Smith DH, Rodríguez-Kábana R, eds. 1984. *Compendium of Peanut Diseases*. St Paul: APS Press
98. Rabbinge R, de Wit CT. 1989. Systems, models and simulation. In *Simulation and Systems Management in Crop Protection*, ed. R Rabbinge, SA Ward, HH Van Laar, pp. 3–15. Wageningen: Pudoc
99. Rabbinge R, Rijsdijk FH. 1981. Disease and crop physiology: a modeler's point of view. In *Effects of Disease on the Physiology of the Growing Plant*, ed. PG Ayres, pp. 201–20. Cambridge: Cambridge Univ. Press
100. Rabbinge R, Ward SA, Van Laar HH, eds. 1989. *Simulation and Systems Management in Crop Protection*. Wageningen: Pudoc
101. Reinink K. 1986. Experimental verification and development of EIPRE, a supervised disease and pest management system for wheat. *Neth. J. Plant Pathol.* 92:3–14
102. Rosenberg MS, Garrett KA, Su Z, Bowden R.L. 2004. Meta-analysis in plant pathology: synthesizing research results. *Phytopathology* 94:1013–17
103. Rossing W. 1991. Simulation of damage in winter wheat caused by the grain aphid *Sitobion avenae*. 2. Construction and evaluation of a simulation model. *Neth. J. Plant Pathol.* 97:25–54
104. Rossing WAH, Daamen RA, Hendrix EMT. 1994. Framework to support decisions on chemical pest control under uncertainty, applied to aphids and brown rust in winter wheat. *Crop Prot.* 13:25–34
105. Rossing WAH, Daamen R, Jansen MJW. 1994. Uncertainty analysis applied to supervised control of aphids and brown rust in winter wheat. Part 1. Quantification of uncertainty in cost-benefit calculations. *Agric. Syst.* 44:419–48
106. Rossing WAH, Daamen RA, Jansen MJW. 1994. Uncertainty analysis applied to supervised control of aphids and brown rust in winter wheat. Part 2. Relative importance of different components of uncertainty. *Agric. Syst.* 44:449–60

107. Rouse DI. 1988. Use of crop-growth models to predict the effects of diseases. *Annu. Rev. Phytopathol.* 26:183–201
108. Rowe WD. 1980. Risk assessment approaches and methods. In *Society, Technology, and Risk Assessment*, ed. G Conran, pp. 3–29. New York: Academic
109. Savary S, Elazegui FA, Moody K, Litsinger JA, Teng PS. 1994. Characterization of rice cropping practices and multiple pest systems in the Philippines. *Agric. Syst.* 46:385–408
110. Savary S, Elazegui FA, Pinnschmidt H, Teng PS. 1997. Characterization of rice pest constraints in Asia: an empirical approach. In *Applications of Systems Approaches at the Farm and Regional Levels*, ed. PS Teng, MJ Kropff, HFM ten Berge, JB Dent, FP Lansigan, HH van Laar, 1:83–98. Dordrecht: Kluwer/IRRI: ICASA
- 110a. Savary S, Mille B, Rolland B, Lucas P. 2006. Patterns and management of crop multiple pathosystems. See Ref. 35: In press
111. Savary S, Willocquet L, Elazegui FA, Castilla NP, Teng PS. 2000. Rice pest constraints in tropical Asia: quantification of yield losses due to rice pests in a range of production situations. *Plant Dis.* 84:357–69
112. Savary S, Willocquet L, Elazegui FA, Teng PS, Du PV, et al. 2000. Rice pest constraints in tropical Asia: characterization of injury profiles in relation to production situations. *Plant Dis.* 84:341–56
113. Savary S, Zadoks JC. 1992. Analysis of crop loss in the multiple pathosystem groundnut-rust-leaf spot. I. Six experiments. *Crop Prot.* 11:99–109
114. Savary S, Zadoks JC. 1992. Analysis of crop loss in the multiple pathosystem groundnut-rust-leaf spot. II. Study of the interaction between diseases and crop intensification in factorial designs. *Crop Prot.* 11:110–20
115. Savary S, Zadoks JC. 1992. Analysis of crop loss in the multiple pathosystem groundnut-rust-late leaf spot. III. Correspondence analyses. *Crop Prot.* 11:229–39
116. Shane WW, Teng PS. 1987. Generating the data base for disease-loss modeling. See Ref. 123, pp. 82–89
117. Sinha RC, Savard ME. 1997. Concentration deoxynivalenol in single kernels and various tissues of wheat heads. *Can. J. Plant Pathol.* 19:8–12
118. Smil V. 2000. *Feeding the World: A Challenge for the Twenty-First Century*. Cambridge, MA: MIT Press
119. Strange RN, Scott PR. 2005. Plant disease: a threat to global food security. *Annu. Rev. Phytopathol.* 43:83–116
120. Styne BA. 1980. Synoptic methodologies for crop loss assessment. See Ref. 129, pp. 166–80
121. Teng PS. 1981. Validation of computer models of plant disease epidemics: a review of philosophy and methodology. *Z. Pflanzenkr. Pflanzenschutz* 88:49–63
122. Teng PS. 1983. Estimating and interpreting disease intensity and loss in commercial fields. *Phytopathology* 73:1587–90
123. Teng PS, ed. 1987. *Crop Loss Assessment and Pest Management*. St. Paul, MN: APS Press
124. Teng PS. 1990. Integrated pest management in rice. An analysis of the status quo with recommendations for action. *Univ. Hawaii Rep.* 63 pp.
125. Teng PS 1994. Integrated pest management in rice. *Exp. Agric.* 30:115–37
126. Teng PS. 1999. Current and future importance of biotechnology to crop protection. In *International Crop Protection: Achievements and Ambitions*, pp. 101–22. *Br. Crop Prot. Counc. Symp. Proc. no. 73*
127. Teng PS, Blackie MJ, Close RC. 1977. A simulation analysis of crop yield loss due to rust disease. *Agric. Syst.* 2:189–98

128. Teng PS, Gaunt RE. 1980. Modelling systems of disease and yield loss in cereals. *Agric. Syst.* 6:131–54
129. Teng PS, Krupa SV, ed. 1980. *Assessment of Losses Which Constrain Production and Crop Improvement in Agriculture and Forestry. Proc. EC Stackman Commem. Symp. Univ. Minn., St. Paul*
130. Teng PS, Savary S. 1992. Implementing the system approach in pest management. *Agric. Syst.* 40:237–64
131. Teng PS, Savary S, Revilla I. 1993. Systems of plant protection. In *Crop Protection and Sustainable Agriculture*, ed. DJ Chadwick, J Marsh, pp. 116–139. Chichester, UK: Wiley
132. van den Bosch F, Metz JAJ, Zadoks JC. 1999. Pandemics of focal plant disease, a model. *Phytopathology* 89:495–505
133. van der Wal AF, Shearer B, Zadoks JC. 1970. Interaction between *Puccinia recondita* and *Septoria nodorum* on wheat, and its effect on yield. *Neth. J. Plant Pathol.* 76:261–63
134. van Oijen M. 1992. Evaluation of breeding strategies for resistance and tolerance to late blight in potato by means of simulation. *Neth. J. Plant Pathol.* 98:3–11
135. Waggoner PE, Berger RD. 1987. Defoliation, disease, and growth. *Phytopathology* 77:393–98
136. Walls GC, Jeger MJ, Frederiksen RA. 1989. The relationship of yield loss to foliar diseases on sorghum grown by subsistence farmers in southern Honduras. *Trop. Pest Manag.* 35:57–61
137. Wiese MV. 1982. Crop management by comprehensive appraisal of yield determining variables. *Annu. Rev. Phytopathol.* 20:419–32
138. Willocquet L, Savary S, Fernandez L, Elazegui F, Teng PS. 2000. Development and evaluation of a multiple-pest, production situation specific model to simulate yield losses of rice in tropical Asia. *Ecol. Model.* 131:133–59
139. Willocquet L, Savary S, Fernandez L, Elazegui FA, Castilla N, et al. 2002. Structure and validation of RICEPEST, a production situation-driven, crop growth model simulating rice yield response to multiple pest injuries for tropical Asia. *Ecol. Model.* 153:247–68
140. Willocquet L, Elazegui FA, Castilla NP, Fernandez L, Fischer KS, et al. 2004. Research priorities for rice disease and pest management in tropical Asia: a simulation analysis of yield losses and management efficiencies. *Phytopathology* 94:672–82
141. Yang XB, Dowler WM, Royer MH. 1991. Assessing the risk and potential impact of an exotic plant disease. *Plant Dis.* 75:976–82
142. Yuen JE, Hughes G. 2002. Bayesian analysis of plant disease prediction. *Plant Pathol.* 51:407–12
143. Yuen JE, Twengström E, Sigvald R. 1996. Calibration and verification of risk algorithms using logistic regression. *Eur. J. Plant Pathol.* 102:847–54
144. Zadoks JC. 1967. Types of losses caused by plant diseases. *Proc. FAO Symp. Crop Losses*, pp. 149–58. Rome: FAO
145. Zadoks JC. 1985. On the conceptual basis of crop loss assessment: the threshold theory. *Annu. Rev. Phytopathol.* 23:455–73
146. Zadoks JC. 1987. Rationale and concepts of crop loss assessment for improving pest management and crop protection. See Ref. 123, pp. 1–5
147. Zadoks JC. 1989. EPIPRE, a computer-based decision support system for pest and disease control in wheat: its development and implementation in Europe. In *Plant Disease Epidemiology*, ed. KJ Leonard, WE Fry, 2:3–29. New York: McGraw-Hill
- 147a. Zadoks JC, Anderson PK, Savary S. 1995. An eco-regional perspective of crop protection problems. In *Eco-Regional Approaches for Sustainable Land Use and Food Production*, ed. J Bouma, pp. 437–52. Amsterdam: Kluwer

148. Zadoks JC, Rabbinge R. 1985. Modelling to a purpose. In *Advances in Plant Pathology*, Vol. 3. *Mathematical Modelling of Crop Diseases*, ed. CA Gilligan, pp. 231–44. London: Academic
149. Zadoks JC, Schein RD. 1979. *Epidemiology and Plant Disease Management*. New York: Oxford Univ. Press
150. Zadoks JC, Schein RD. 1980. Epidemiology and plant disease management, the known and the needed. In *Comparative Epidemiology*, ed. J Palti, J Kranz, pp. 1–17. Wageningen: Pudoc
151. Zeng, SM. 1991. PANCRIN, a prototype model of the pandemic cultivar-race interaction of yellow rust on wheat in China. *Plant Pathol.* 40:287–95



Contents

A Retrospective of an Unconventionally Trained Plant Pathologist: Plant Diseases to Molecular Plant Pathology <i>Seiji Ouchi</i>	1
The Current and Future Dynamics of Disease in Plant Communities <i>Jeremy J. Burdon, Peter H. Thrall, and Lars Ericson</i>	19
A Catalogue of the Effector Secretome of Plant Pathogenic Oomycetes <i>Sophien Kamoun</i>	41
Genome Packaging by Spherical Plant RNA Viruses <i>A.L.N. Rao</i>	61
Quantification and Modeling of Crop Losses: A Review of Purposes <i>Serge Savary, Paul S. Teng, Laetitia Willocquet, and Forrest W. Nutter, Jr.</i>	89
Nonsystemic Bunt Fungi— <i>Tilletia indica</i> and <i>T. horrida</i> : A Review of History, Systematics, and Biology <i>Lori M. Carris, Lisa A. Castlebury, and Blair J. Goates</i>	113
Significance of Inducible Defense-related Proteins in Infected Plants <i>L.C. van Loon, M. Rep, and C.M.J. Pieterse</i>	135
Coexistence of Related Pathogen Species on Arable Crops in Space and Time <i>Bruce D. L. Fitt, Yong-Hu Huang, Frank van den Bosch, and Jonathan S. West</i>	163
Virus-Vector Interactions Mediating Nonpersistent and Semipersistent Transmission of Plant Viruses <i>James C.K. Ng and Bryce W. Falk</i>	183
Breeding for Disease Resistance in the Major Cool-Season Turfgrasses <i>Stacy A. Bonos, Bruce B. Clarke, and William A. Meyer</i>	213
Molecular Ecology and Emergence of Tropical Plant Viruses <i>D. Fargette, G. Konaté, C. Fauquet, E. Muller, M. Peterschmitt, and J.M. Thresh</i> ...	235
Biology of Flower-Infecting Fungi <i>Henry K. Ngugi and Harald Scherm</i>	261

A Model Plant Pathogen from the Kingdom Animalia: <i>Heterodera glycines</i> , the Soybean Cyst Nematode <i>T.L. Niblack, K.N. Lambert, and G.L. Tylka</i>	283
Comparative Genomics Reveals What Makes an Enterobacterial Plant Pathogen <i>Ian K. Toth, Leighton Pritchard, and Paul R. Birch</i>	305
The Dawn of Fungal Pathogen Genomics <i>Jin-Rong Xu, You-Liang Peng, Martin B. Dickman, and Amir Sharon</i>	337
Fitness of Human Enteric Pathogens on Plants and Implications for Food Safety <i>Maria T. Brandl</i>	367
The Role of Ethylene in Host-Pathogen Interactions <i>Willem F. Broekaert, Stijn L. Delaure, Miguel F.C. De Bolle, and Bruno P.A. Cammue</i>	393
Phenazine Compounds in Fluorescent <i>Pseudomonas</i> Spp. Biosynthesis and Regulation <i>Dmitri V. Mavrodi, Wulf Blankenfheldt, and Linda S. Thomashow</i>	417
Long-Distance RNA-RNA Interactions in Plant Virus Gene Expression and Replication <i>W. Allen Miller and K. Andrew White</i>	447
Evolution of Plant Pathogenicity in <i>Streptomyces</i> <i>Rosemary Loria, Johan Kers, and Madhumita Joshi</i>	469
Climate Change Effects on Plant Disease: Genomes to Ecosystems <i>K.A. Garrett, S.P. Dendy, E.E. Frank, M.N. Rouse, and S.E. Travers</i>	489

INDEX

Subject Index	599
---------------------	-----

ERRATA

An online log of corrections to *Annual Review of Phytopathology* chapters (if any, 1977 to the present) may be found at <http://phyto.annualreviews.org/>