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Systems analysis of wheat stripe rust epidemics in China

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Abstract Stripe rust is the most destructive disease of wheat in China and shows long-distance spread with interregional epidemics. Systems analysis had been implemented by the epidemiology group at the China Agricultural University to study the epidemiology of this disease from field to regional levels. This paper reviews major achievements by this group from the 1970s to the present in this pathosystem in China. Field experiments were conducted to obtain necessary parameters for constructing various simulation models. Field-level models were generated to study the spatial and temporal disease developments, while region-level models were developed to study the disease long-distance spread, interregional epidemics and strategies for disease management. Interactions between host, pathogen and environment were also studied with modelling approaches to generate information on deployment of resistance for regional disease management.

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Features of wheat stripe rust epidemics in China

Stripe rust, caused by *Puccinia striiformis* f. sp. *tritici*, is the most devastating wheat disease in China. The epidemics of 1950, 1964, 1990 and 2002 in China caused yield losses up to 6.0, 3.0, 1.8, and 1.3 million tons, respectively (Li and Zeng 2002; Wan et al. 2004). Although occurrence of wheat stripe rust may have a long history in this country, intensive research on the disease began only in the 1940s with studies on physiological specialization of the pathogen (Fang 1944) and varietal resistance (Fang and Chen 1955). The earliest severe epidemics of the disease occurred from late 1950s to early 1960s when the new race CYR1 overcame the resistance of the variety Bima #1 which was grown in 6 million hectares covering in northern, north-western, north-central and central China. The following significant boom-bust cycles from the 1960s to the present due to uses and losses of resistance in vast areas demonstrated the 'man-guided' evolution (Johnson 1961) of this pathogen and resulted in pandemics of the disease in China (Li and Zeng 2002).

In addition to similar features of disease epidemics in Europe and North America (Nagarajan and Singh 1975; Line 2002; Roelfs 1985; Zadoks 1961) including pathogen long-distance dispersal between differ-

ent geographic regions to complete the pathogen life cycle and the special climates for pathogen overwintering and oversummering, in China, wheat stripe rust manifests an independent epidemic system in which the pathogen completes its life cycle in different geographical regions of the country with very little chance of pathogen population exchange with those of other countries (Zeng and Luo 2006). As an obligate parasite (Zadoks 1961; Burleigh et al. 1969; Line 2002;), the pathogen has to overwinter and oversummer in living green wheat plants to complete its life cycle (Li and Zeng 2002). The lower temperature limit for overwintering is −6°C to −7°C (monthly average of daily air temperatures), or as low as −10°C when wheat is under snow cover (Li and Zeng 2002). The upper limit for oversummering is 20°C to 22°C (Xun average highest temperature) (Li and Zeng 2002). (Chinese Xuns are equivalent to 10-day periods: upper Xun: days 1–10, mid Xun: days 11–20, and lower Xun: days 21–28/31). However, in most wheat-growing areas in China, the pathogen cannot finish its life cycle by both overwintering and oversummering locally due to the temperature limits. Since China shows a great variation between wheat growing areas in geomorphological and climatological features in cultural characteristic and varieties grown, there is a considerable diversity in epidemiological conditions between the wheat-growing areas. In China, rust oversummers primarily in the mountainous regions of southern and eastern Gansu Province and northwestern Sichuan Province due to three major reasons: (1) the average maximum temperature <22°C in the hottest Xun, (2) wheat crops of different maturity dates grown at different elevations, and (3) an overlap exists between the period of epidemics on late crops and the period with self-sown seedlings from early crops (Li and Zeng 2002). After oversummering, vast amounts of spores that are produced in this mountain area can be continuously blown eastward to cause infections on seedlings of newly-sown crops in more easterly regions in the fall. Since west winds prevail during the fall in China, vast winter wheat-growing areas in northern, north-central and eastern China may receive spores originating from these mountain areas. In addition to the important epidemic regions discussed below, Yili basin of Xinjiang province and Yunan–Guizhou plateau are another two regions where wheat stripe rust epidemics occasionally occur. However, there is no evidence to show that the epidemics in these two regions relate to each other and to those of the main epidemic regions in northern, north-eastern and central China, and Yangzi River Valley (Zeng and Luo 2006). The present paper focuses on the epidemics in these regions.

Zeng (1988b, 2000) proposed a concept of a regional epidemic system according to the regional characteristics of disease development, and attempted to classify the major areas of stripe rust epidemics into 15 different epidemic regions in China, based on the understanding of characteristics of individual regions and the relationship in disease development between regions. Figure 1 shows the location of these epidemic regions of wheat stripe rust in China. The classification of these regions is based on their geographic, climatic, wheat planting and growing and disease epidemic features (Zeng and Luo 2006). The interregional long-distance disease spread and epidemiological features in each region have been intensively reviewed (Zeng and Luo 2006).

Thus, epidemiological studies of wheat stripe rust in China must consider the following features: (1) the epidemics in a region may be caused by endogenous inoculum, exogenous inoculum or both, depending on characteristics of the specific epidemic region and its role in interregional disease pandemics (Zeng and Luo 2006), (2) the pathogen pathway among different regions is critical to predict disease intensity, potential risk of epidemics and their possible influences on other regions, (3) strategies of resistance gene deployment in different regions would greatly influence the racial shifts of the pathogen between the epidemic regions that determines the intensity of disease pandemics, and (4) understanding spatial and temporal disease development at both field and regional scales is essential for disease management at field and regional levels. These features caused Chinese scientists to work on the almost all aspects of this pathosystem, which undoubtedly increased the difficulty in epidemiological research on this disease.

The research on wheat stripe was rust started by the epidemiology group at the China Agricultural University (CAU, former Beijing Agricultural University) at the end of 1950s. Basic field experiments, dealing with the quantitative field-scale analysis of Van der Plank's (Van der Plank 1960, 1963) apparent infection rate (r) in relation to precipitation and

Fig. 1 Wheat stripe rust epidemic regions in China based on geographic, climatic, wheat growing and disease epidemic features. Fifteen epidemic regions were classified (see Zeng and Luo 2006, for definition). *Grey lines* are borderlines of provinces and *black lines* are borderlines of epidemic regions. *Arrows* indicate the possible trend directions of pathogen dispersal in spring and fall across these epidemic regions

temperature, were conducted in early 1960s and published in 1962 (Zeng 1962). Table 1 lists *r* values obtained from different geographic locations and the corresponding weather conditions. The universally susceptible cv. Bima #1 was planted in all locations in that time period, and CYR 1 was the predominant race during these years. Experimental data were used to generate a mathematical model to estimate the

Table 1 Examples of disease apparent infection rates (*r* values) of wheat stripe rust obtained from epidemics in multiple locations in 1960 and 1961, and disease incidences during the corresponding time period of the seasons

Location and year	Duration and incidence	Apparent infection rate (*r*)	Rain days	Duration	Precipitation (mm)	Duration and average temperature (°C)
Huangchuan 1961	3/15–4/15 0.078–77.9%	0.34	12	3/1–3/31	116.6	3/1–4/16 12.6
Luohe 1961	4/10–5/10 0.0038–9.41%	0.30	6	3/26–4/26	33.7	3/26–5/10 19.8
Zhengzhou 1960	3/17–4/16 0.008–13.6%	0.29	11	3/1–3/31	72	3/1–4/16 10.5
Zhengzhou 1961	3/10–4/10 0.002–0.631%	0.23	5	3/26–4/26	28	3/26–5/10 19
Jieshan 1960	3/17–4/15 0.002–0.631%	0.21	7	3/1–3/31	34.7	3/1–4/16 10.3
Shuitou 1961	3/30–5/1 0.00029–0.045%	0.20	3	3/10–4/15	14.3	3/10–5/1 16.3
Shuiji 1961	3/30–4/30 0.012–0.25%	0.14	6	3/10–4/15	9.3	3/16–5/1 16.4
Ningjin 1960	3/21–421 0.0013–0.028%	0.13	4	3/5–4/11	7.1	3/5–4/21 10.3

The relevant weather conditions during the corresponding time period were used to calculate the *r* values.

distance of disease spread (Zeng 1963). Based on many surveys at different geographical scales, from field to region, more knowledge about region-level disease epidemics was obtained. In the 1970s, the group considered systems analysis (Jeffers 1978; Robert 1975; Zadoks 1971) as a useful tool to study the epidemiology of stripe rust in China, since realistic experiments were not always feasible and since the necessary information about epidemics and regional gene deployment was difficult to obtain and interpret. The group performed field experiments for parameter estimation and developed different types of models for field-level and region-level disease development. The group applied the systems analysis methods to stripe rust research by following the Zadoks (Zadoks 1971) methodology of systems analysis and computer simulation (de Wit and Goudriaan 1978; de Wit 1982; Kranz and Hau 1980; Teng et al. 1980; Waggoner 1978). Temporal and spatial development of disease was studied intensively in field experiments, and several simulation models were developed. These models were upgraded to become tools for addressing questions about stripe rust epidemiology and control at various levels, from field to region. The present review discusses major achievements of the group on stripe rust research by using systems analysis approaches from the mid-1950s to about the 2000s, briefly summarised in Table 2. Figure 2 summarizes the linkages between the successive models and their relationships in the hierarchy of model construction, and Table 3 lists the symbols used in this paper.

Simulation of temporal disease development at field level

Daily multiplication factor (DMF)

Work began with the determination of the Daily Multiplication Factor (DMF) using the number of diseased leaves as a basic unit. The DMF of stripe rust, defined as the daily increment of newly-diseased daughter leaves per infectious mother leaf, is a function of their sporulation and of weather variables. From 1979 to 1981, studies were made to assess the

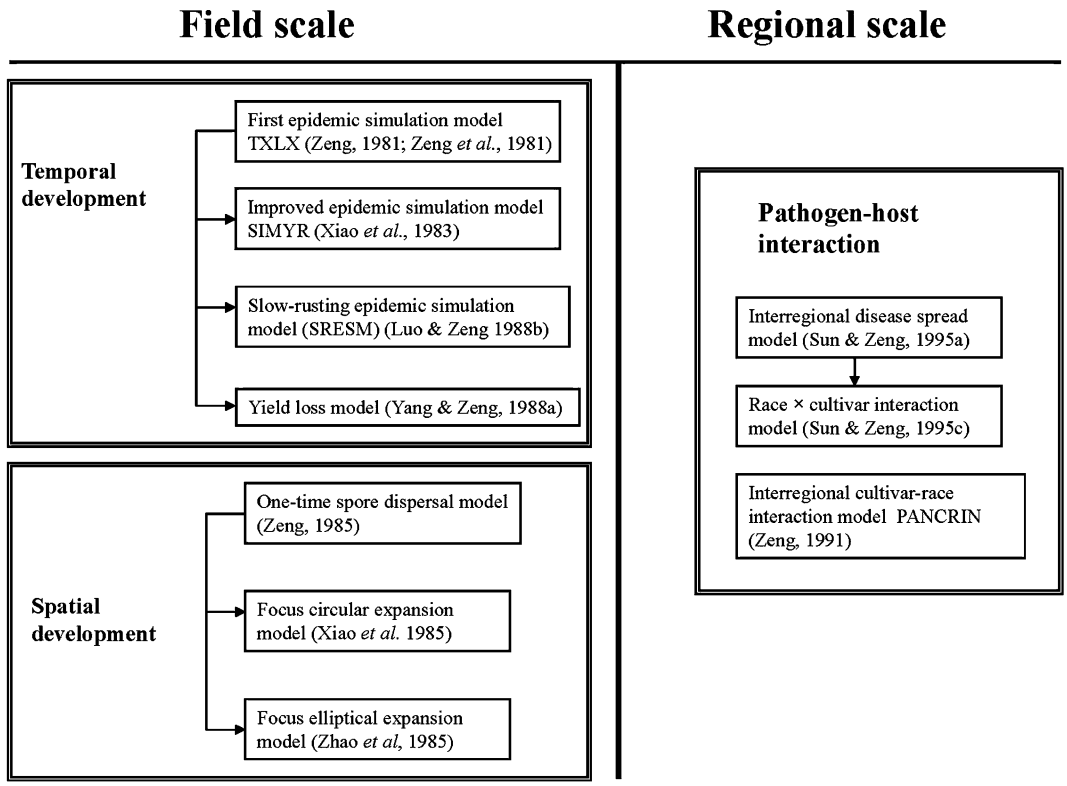

Fig. 2 Summary of main simulation models and their relationships in hierarchy of systems analysis of wheat stripe rust. The models were completed by the epidemiology group at China Agricultural University from mid 1970s to early 2000s

Table 2 Chronological summary of studies on systems analysis of wheat stripe rust epidemics conducted by the epidemiology group at the China Agricultural University (former Beijing Agricultural University), 1960 to 2004

Year	Description of studies	Description of model(s)	Model application	Reference
1960–1964	Estimation of epidemic rate at field and regional levels	Epidemic rate, $R_{cd}=f$ (monthly dew days, monthly precipitation, monthly average temperature)	Prediction of R_{cd} Estimation of maximum R_{cd} Detection of interregional disease spread	Zeng (1962) Zeng and Luo (2006)
1960–1963	Disease spread at field level	Distance of spread=f (quantity of inoculum in focus, precipitation)	Prediction of focus expansion	Zeng (1963)
		Distance of spread=f (inoculum density, wind)	Determination of the area for fungicide spray to eradicate disease foci	Zeng (1981)
		Disease distribution in the field due to one-day spore dispersal	Simulation and prediction of daily expansion of disease foci	Zhang and Zeng (1986)
		Gradient of disease spread		Zeng (1988a)
		Circular and elliptic disease spread models		Xiao et al. (1985); Zhao et al. (1985)
1960–1966 and 1975–1988	Quantitative analysis of disease spread at regional level	Average disease index of target region caused by exogenous inoculum=f (disease intensity of source region, distance between the source and target regions)	Estimation of possible distance of interregional disease spread	Zeng (1988b)
1975–1988	Experiments and modelling for epidemics at field level	Daily multiplication factor, DMF=f (dew temperature, dew period, leaf area with sporulation)	Simulation of daily disease development	Xiao et al. (1983); Zeng (1980)
		Disease apparition rate, APP=f (accumulated effective temperature)	Simulation of slow-rusting epidemics	Luo and Zeng (1988a, b, 1989)
		Lesion expansion rate, LE=f (temperature, solar radiation)	Estimation of yield loss	Yang and Zeng (1988a, b)
		Disease index=f (DMF, APP, LE)	Risk analysis of disease epidemics	Luo et al. (1993); Luo and Zeng (1990a)
		Yield loss=f (disease index, photosynthesis reduction)	Deployment of resistant genes, analysis of local and exogenous inoculum sources and effects of chemical control	Luo et al. (1993); Luo and Zeng (1989)
1989–2003	Parasitic fitness of races on cultivars	Relative parasitic fitness=$r_{i,j}/r_{ck}$	Understanding the quantitative interactions between cultivars and races	Zeng (1996a, 2003a)
		$r_{i,j}$: epidemic rate of the race j on cultivar i r_{ck}: epidemic rate of the universally susceptible combination	Prediction of dynamics of races and breakdown of resistance	Sun and Zeng (1993, 1995a, b, c)
1989–2004	Interregional pandemics and cultivar–race interactions	Simulation model PANCRIN	Regional epidemic pattern	Zeng (1991, 1994)
		Regional classification for epidemics	Evaluation of durable resistance	Zeng (1996a)
		Disease progress in a region=f (R, EXO, F)	Estimation of dynamics of races based on cultivar deployment information	Zeng (1996a, 1998)
		R: predicted epidemic rate for the region	Study of pathogen oversummering	Zeng (2003b)
		EXO: estimated distance of interregional disease spread	Study of resistance deployment at regional level	Zeng (2003b)
		F: estimated parasitic fitness		

Table 3 Description of symbols or abbreviations in the text

Abbreviation or symbol dimension	Description
Epidemiological	
AC	Relative acreage of a cultivar
AD	Area of diseased leaves
APP	Daily apparition rate
D	Distance of disease spread
DI	Disease incidence
DII	Disease incidence×10% (average severity arbitrarily determined)
DIL	Dilution coefficient of long-distance disease dissemination
DMF	Daily multiplication factor
DS	Disease severity
DX	Disease index=DI×DS
EXO	Estimated distance of disease spread
F	Parasitic fitness of a race on a cultivar
IDX	Improved disease index
IF	Infection efficiency
IP	Infectious period
LAI	Relative leaf area index of a cultivar
LE	Lesion expansion rate
LP	Latent period
LS	Lesion size in mm^2.
P	Actual photosynthetic rate of the diseased leaf
PF	Relative parasitic fitness of pathogen races on combination of cultivars
P_{max}	Maximal photosynthetic rate under the optimal conditions
R	Regional apparent infection rate
r	Van der Planks's apparent infection rate
rr	Relative epidemic rate
R_{cd}	Calculated regional epidemic rate
RD	Number of days with rain >0.1 mm
RT	Proportion of a race in a particular region and month
S	Disease intensity (severity×total number of diseased leaves)
SP	Sporulation per unit of lesion per day
TT_{50}	Accumulated effective temperature that apparition of 50% infected sites requires
XE	Severely infected fields in hectare× average incidence×average severity
Meteorological	
DP	Dew period in h
DT	Dew temperature
PP	Monthly precipitation in mm
RD	Monthly number of rainy days
TE	Monthly average temperature
TT	Accumulated effective temperature
XT	Relative amount of disease on a cultivar caused by a race
Others	
PANCRIN	A simulation model for interregional cultivar–race interactions
SIMYR	Simulation model for spring epidemic of stripe rust
SRESM	Slow-rusting epidemic simulation model
TXLX	The first Chinese simulation model

necessary parameters. In the fall, inoculation experiments were conducted in Beijing, far away from the oversummering areas of the rust, to avoid spontaneous infections. The susceptible cv. Yanda #1817 was planted in October in 3×3 m plots. Pots containing seedlings with one or more diseased leaves per pot were placed at the centre of each plot for 24 h as inoculum sources. These field inoculations were done once a day for each plot with three replicates. Pots contained 1, 3, or 9 diseased leaves, representing different inoculum densities. The experiments lasted in 2–3 weeks. After one latent period (7–10 days), the newly-diseased leaves around the inoculation foci were counted. An exponential regression equation (Eq. 1) was obtained to estimate the DMF value:

$$\mathrm{DMF} = \exp\{0.55 \times \ln[(\mathrm{DP}-5)^2 \times \mathrm{DT} \times (\mathrm{DT}-1) \times (18-DT)] + 2.34 \times \ln(\mathrm{AD}) - 3.94\} \quad (1)$$

where DP is the dew period duration in h (with DP=5 when DP<5), DT is dew temperature (0<DT<18°C), and AD is the sporulating area of infectious mother leaves in cm^2. This regression equation was validated by means of a separate set of the multi-year experimental data, collected after the first set of experiments, and the results indicated that 70% of the observations were within the range of predicted value ±2 standard deviations (unpublished).

Disease apparition rate (APP)

The latency and daily rate of apparition of infections (Fig. 2) are important characteristics of a stripe rust epidemic (Zadoks 1961). The process of latently infected leaves beginning to sporulate, called appari-

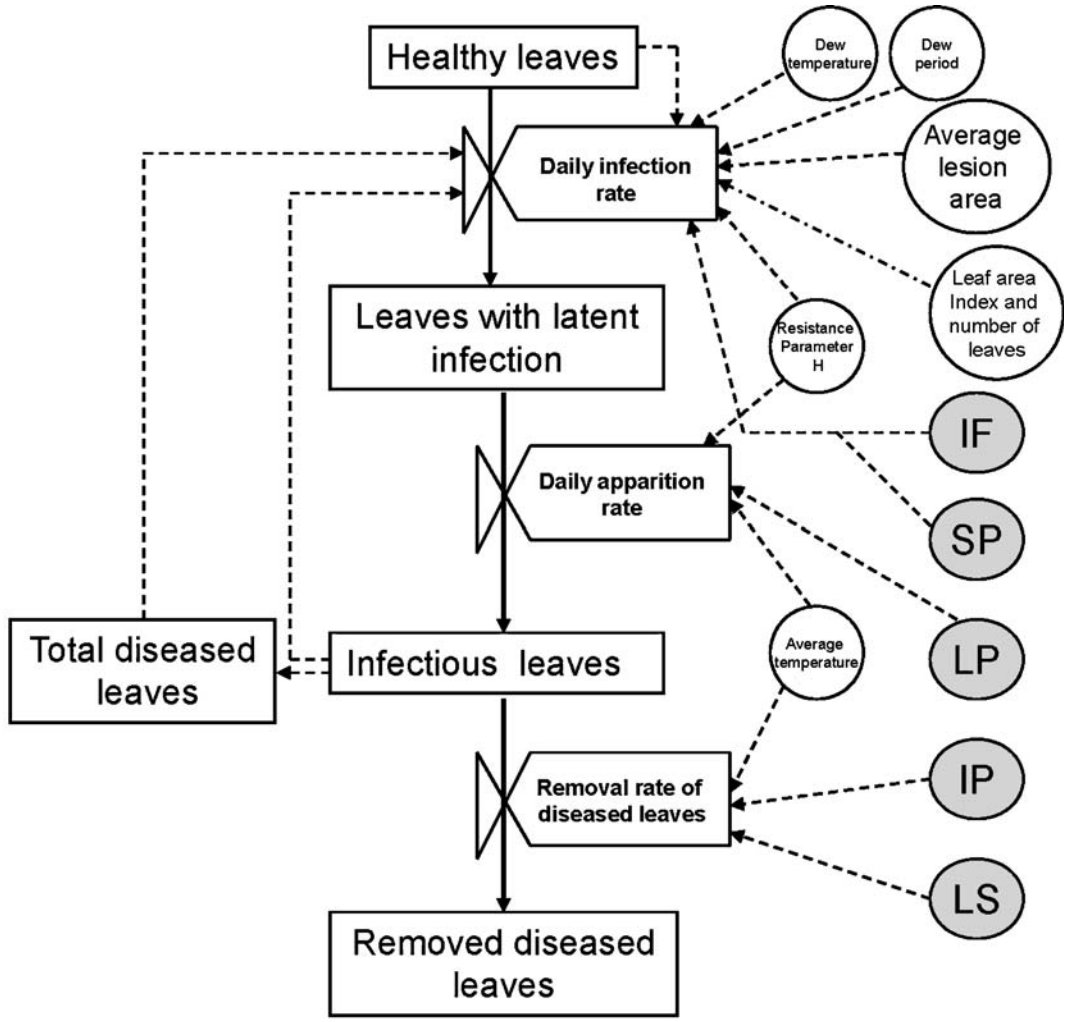

Fig. 3 The flow chart diagram of the preliminary simulation model of wheat stripe rust TXLX developed by Zeng (1981). The variables indicated in *circles with grey background* are parameters of slow-rusting resistance incorporated into this model as part of slow-rusting epidemic simulation model SRESM (Luo and Zeng 1988b)

tion, is a component of the disease simulation model. This process was quantified as a function of temperature. For leaves or sites infected simultaneously, the apparition process may last 3–7 days depending mainly upon daily temperature. The disease apparition rate for a certain day was calculated as the quotient of the number of just-appeared diseased leaves divided by the total number of diseased leaves when apparition was completed. This rate was made a function of accumulated effective temperature, defined by accumulated daily average temperatures above 0°C from the inoculation date to the corresponding disease recording date. Multi-year field experiments were conducted to obtain the quantitative relationships between accumulated effective temperature and APP. Wheat seedlings in the field were inoculated weekly in the spring. Newly-appearing sporulating leaves were counted daily from the inoculation day until the day when no more newly-sporulating leaves were detected. Data sets of APP versus the corresponding accumulated effective temperature were used to develop e.g. regression Eq. 2 (Xiao and Zeng 1985):

$$\mathrm{APP}_i = 1/\left(\left(1 + 3.822 \times 10^{12} \times \exp(-0.2126 \times \mathrm{TT}_i)\right)\right) \quad (2)$$

where APP_i is the daily apparition rate at day i, and TT_i is the accumulated effective temperature from the day of inoculation to day i.

Modelling stripe rust epidemics in spring

The first Chinese simulation model, TXLX (Fig. 2) (Zeng 1981; Zeng et al. 1981), simulating disease development, was driven by the number of diseased leaves using a time step of 1 day. The model calculated the number of newly-infected leaves per day using daily dew temperature and dew period. The number of latently infected leaves was used to model the apparition process. Next, the model computed the number of newly-sporulating leaves. Another module calculated the daily number of spores produced using a lesion expansion rate. The infectious period parameter (in days) was used to compute the number of leaves dying each day and the total number of dead leaves. The daily weather variables included average temperature, dew temperature and dew period. The model produced estimates of the daily disease incidence and severity (%) for a field of a certain size (about a hectare, 15 Chinese *mu*). Xiao et al. (1983) developed the model SIMYR, elaborating upon TXLX, improving some functions with more detailed information.

Simulation of slow-rusting

Stripe rust epidemics on wheat cultivars with slow-rusting resistance were also studied (Luo and Zeng 1988a). Multi-year field experiments were conducted with six wheat cultivars to assess their slow-rusting parameters (Parlevliet 1979) including infection frequency (IF), latent period (LP), sporulation (SP), lesion expansion rate (LE), and infectious period (IP). Compared with a universally susceptible cultivar, the slow-rusting cultivars expressed significantly lower IF, longer LP, less SP, slower LE, and shorter IP (Luo and Zeng 1988a). Elaborating upon the simulation models mentioned above, the slow-rusting epidemic simulation model (SRESM) was developed (Luo and Zeng 1988b, 1989) and subjected to validation tests using data from field experiments. The new model, as indicated in Fig. 3 incorporated parameters for the components of resistance into the corresponding modules of the older models using detected values from specific cultivars. For example, IF (0–1) was incorporated in the module simulating infection process, LP (TT_{50} (accumulated effective temperature that an apparition of 50% infected sites requires): 129–164) was incorporated in the module simulating latent period, IP (7.0–11.6 days) was incorporated in the module to determine infectious period, LS (0.06–0.19 cm^2 day^{-1} was incorporated in the module simulating lesion expansion, and SP (14,000–40,000 spores day^{-1} $leaf^{-1}$ defined sporulation capacity (Luo and Zeng 1988b). Simulation experiments (Luo and Zeng 1995) demonstrated that IF, SP and IP were the most important components of slow-rusting resistance.

Coupling a weather generator (Shen 1988) to SRESM, the model was used to estimate the risk (probability of occurrence of a certain disease incidence) of stripe rust epidemics on cultivars with different resistance levels under various weather conditions for different wheat-growing regions (Luo and Zeng 1990a, 1995). With three values of each component as inputs, a series of virtual cultivar model calculations was produced leading to a classification into low, moderate, and high risks of epidemics (probability of occurrence of a certain disease intensity). Incorporating a yield model (Yang and Zeng 1988b), yield loss was simulated for each cultivar with a certain combination of slow-rusting parameters. A criterion of yield loss was used to determine the usefulness of a cultivar if grown under different climatic conditions. For example, some cultivars may show a higher epidemic risk if grown in an area with more rain (Luo and Zeng 1995). The results were applied to assist in decision making on regional deployment of resistant cultivars using resistance level (Luo and Zadoks 1992; Luo and Zeng 1990b) the estimated strength of initial inoculum sources, and climatic conditions for the specific wheat-growing regions (Luo and Zeng 1990a, 1995) as model inputs. The model was coupled to a weather generator (Luo et al. 1993) to produce frequency distributions of yield loss applying a Monte-Carlo method. Risk assessment tables were produced to guide growers in disease management for different ecological systems in China. The tables contained estimated disease epidemic risk levels (0–1) of stripe rust for locations simply defined with dry, normal and humid climatic conditions. For instance, if the initial disease incidence is 0.0001 at early season, the probability of yield loss <3% is about 0.47 in a dry area, but about 0.22 in a humid area.

Simulation of spatial disease development at field level

Uredospores of *P. striiformis* can be dispersed from a few centimeters to over a thousand kilometers (Gregory

1968). The actual distance of spread of disease depends on the amount of inoculum, winds, and conditions for infection. We distinguish three steps in spatial disease development: appearance of isolated individual diseased leaves, development of disease foci, and spread of disease over the whole field, in a way similar to the scales defined by Heesterbeek and Zadoks (1987). Eradication of disease foci was broadly applied in China in the 1950s as a disease management strategy.

Relationship between inoculum quantity and distance of disease spread

Zeng (1963) observed that, weather conditions being equal, the radius of expanding disease foci (secondary spread) correlated positively with the disease intensity of the initial foci (initial source). Using data from multi-year surveys, several regression equations were obtained to describe the relationship between disease intensity of the initial source and distance covered by disease after one latent period in a focus under various precipitation levels. The distance of spread (Gregory 1968) (D) was measured as the maximum radius of the focus following one latent period (i.e., primary disease gradient; Gregory 1968). The initial disease intensity in the focus was determined as the number of sporulating leaves multiplied by their average rust severity. Three weather conditions, described as the total precipitation (mm) during the 10 days in which the primary infection was shedding spores, ≥20, 4–9 and ≤3, were used in these regression equations. The preliminary equation to predict distance of disease spread from 25 observations was obtained as:

$$\text{Log } D = 0.1794 + 0.5838 \text{ Log } S\left(r^2 = 0.95\ P < 0.01\right) \quad (3)$$

Where, D is disease spread distance in m, and S is disease intensity represented by disease intensity× total number of diseased leaves, similar to the conditional severity defined by Xu and Ridout (1998).

Simulation of focus expansion

Field experiments were conducted (Zhang and Zeng 1986) to determine the effects of wind speed and direction on spore dispersal and the corresponding disease spread. A pot with five to eight diseased seedlings was placed for 24 h in a disease-free field. Leaves in the area surrounding the focus were then randomly sampled. Spores were washed into a suspension sample from which the number of spores per leaf was determined under the microscope. The results demonstrated that, in a situation with a steady wind, spore dispersal showed an elliptical pattern. The gradient of spore dispersal was steep in the up-wind direction, but gentle in the down-wind direction. The number of spores dispersed in the cross-wind direction exhibited an approximately normal distribution. A simulation model (Zeng 1985) was developed to predict spore dispersal distance according to the spore distribution gradients. Spatial models to simulate circular (Xiao et al. 1985) and elliptical (Zhao et al. 1985) focus expansion in non-wind and prevailing wind situations were constructed.

In the early 1960s, most wheat-growing regions in China used the disease-focus elimination strategy to control stripe rust. The disease foci were identified in early spring and sprayed with fungicides. Unfortunately, these foci usually expanded dramatically with unexpected speed. Foci of several hundred cm^2 can expand to over 10 m^2 within a few days. When fungicides were applied in a 4- to 5-m radius around a focus, a number of leaves with latent infection might escape the treatment. Therefore, it was found more effective to combine the spore dispersal model, the disease spread prediction equation, and the disease development model. This enabled us to predict disease spread and the number of latent and sporulating leaves in the foci, and to provide guidance for the focus treatment strategy. Although some trials were conducted, more information and further quantitative analysis are needed.

Yield loss model and its applications

Yield loss caused by stripe rust was studied experimentally by Yang and Zeng (1988a). The critical wheat growth stage, at which yield loss was consistently significantly correlated with disease intensity, was the flowering stage. An empirical model was developed to describe the relationship between disease severity at this stage and the corresponding yield loss (%). Luo and Zeng (1995) applied the model to determine criteria for cultivar selection and cultivar deployment in different regions of China, with regional climate data as model inputs, and to estimate

possible risk of wheat stripe rust epidemics on these cultivars. Yang and Zeng (1988b) conducted experiments to determine the mechanism of yield loss and to quantify the relationship of stripe rust severity with reduction of photosynthesis in leaves. The photosynthetic rate of a diseased leaf P showed a linear and inverse correlation with disease severity (DS). The actual photosynthetic rate of the diseased leaf (P) was calculated as $P=P_{max}(1-0.83\times DS)$, where P_{max} was the maximal photosynthetic rate under optimal conditions of temperature and light intensity.

Pathogen–host interactions

Parasitic fitness of rust races on cultivars

Two key features of wheat stripe rust epidemics in China are the large fluctuations in epidemic intensity and the repeated breakdown of resistance. Studies on rust dynamics should consider the interactions between races and cultivars. The management of resistant cultivars plays an important role in race shifts (Leonard 1969; Johnson 1984). Simulation of this process requires measurable parameters such as selection pressure. We chose another parameter, parasitic fitness of the pathogen, to quantify the reproduction of specific races on specific cultivars. Although definitions of parasitic fitness, its dimension, and its measurement have been published (Leonard 1994) a more widely acceptable standard to measure parasitic fitness would be desirable. Parasitic fitness, determined by genetic features of both host and pathogen, can be expressed in terms of relative epidemic rate. Zeng (1996a, b, 1998) defined a variable, the relative epidemic rate, rr, to describe the parasitic fitness of a given race on a certain cultivar in a race×cultivar combination. Measurement of this parasitic fitness required field experiments in a region where exogenous inoculum does not appear late in the spring, as in the Beijing district. Plots of 3.3×3.3 m were used to test various race×cultivar combinations. One race×cultivar combination per plot was tested using central inoculation and focus expansion in the adult plant stage to assess the apparent infection rate r. An improved disease index (IDX) was recorded using the additional relationship between infection type and sporulation (Feng and Zeng 1990; Li and Zeng 1990) to distinguish infection levels. The IDXs recorded at two dates, the appearance of the first rust generation and the end of epidemic season, were used to calculate r (Van der Plank 1963; Zadoks and Schein 1979) per plot. The relative epidemic rate ($rr_{i,j}$) for a specific race i on cultivar j was calculated as $rr_{i,j}=r_{i,j}/r_{ck}$, where $r_{i,j}$ is the apparent infection rate of race i on cultivar j, and r_{ck} is the apparent infection rate of the race×cultivar combination with the highest r value, used as the universally susceptible check. Sun and Zeng (1995c) discussed the mathematical estimation of parasitic fitness in heterogeneous host populations and the application of a parasitic fitness matrix of race×cultivar interactions (Sun and Zeng 1995b). The following equation was used to establish a matrix and to calculate parasitic fitness for a certain cultivar×race combination: $F_{i,k}=S_{i,k}/S_{j,H}$, where, $F_{i,k}$ is the parasitic fitness of race i on cultivar k relative to the fittest race–cultivar combination in the race–cultivar system, $S_{i,k}$ is the disease intensity on cultivar k caused by race i at the end of an epidemic season, and $S_{j,H}$ is the disease intensity caused by race j on cultivar H, which satisfies: $S_{j,H}=\text{Max}\ \{S_{i,k}, i=1 \text{ to } n; k=1 \text{ to } m\}$. From this matrix, the resistance type of the tested cultivar, whether horizontal, vertical, or their combination, could be inferred.

Evaluation of these methods using experimental data is in progress. Although some race×cultivar combinations did not yet yield stable data, the relative epidemic rate is a promising method to estimate parasitic fitness complementing greenhouse and field tests. Parasitic fitness can also be a useful parameter to study the dynamics of pathogen races and the mechanism of resistance breakdown.

A simulation model for interregional cultivar–race interactions: PANCRIN

A simulation model PANCRIN was initially developed in late 1980s and published in 1991 (Zeng 1991). The main objective of this model was to study the regional epidemics of stripe rust in China by consideration of race×cultivar interactions in the various regions. The principle of modelling was based on the regional classification and features of regional epidemics (Zeng and Luo 2006). Eleven main stripe rust epidemic regions were considered based on geographic variation and disease epidemic features. Three major features were considered:

disease epidemic rate, pathogen interregional dispersal and fitness of pathogen races on cultivars with specific resistance.

The following empirical equation was used to calculate the apparent infection rate R at a region scale, different from Van der Plank's apparent infection rate r at a field scale:

$$R = (1.16\mathrm{RD} + 0.1\mathrm{PP} + 0.95\mathrm{TE} - 2.6)/100 \quad (4)$$

where 22>TE<1; RD is the number of days with rainfall >0.1 mm, PP is the monthly precipitation in mm, and TE is the monthly average of daily average temperature in °C. To account for the effect of the relative leaf area index (LAI) on R, R was modified by adding ln (LAI/30). During the winter, when TE is <1, R was computed as:

$$R = \ln\left(0.33^{(1-\mathrm{TE})}\right)\Big/30 \quad (5)$$

During the summer, when TE is >22, R=0.

To calculate the disease incidence (DI) on susceptible cultivars after one latency period in a target region due to long-distance dispersal of pathogen spores, two parameters were considered according to Zeng (1988a): the amount of immigrating inoculum from the source regions XE and the distance in km between the source and target regions D. The following equation was used to calculate the modified DII:

$$\mathrm{DII} = \mathrm{XE}/\exp[5.044 + 2.2698\ \ln(D+1)] \quad (6)$$

where DII is disease incidence (DI)×10% (average severity arbitrarily determined), XE=disease incidence×average severity of infected fields×area of wheat in the source region in Chinese *mu* (1 *mu*= 666.7 m^2). The dilution coefficient during long-distance disease dissemination (DIL) was roughly estimated as:1

$$\mathrm{DIL} = \mathrm{DII}/\mathrm{XE} \quad (7)$$

In consideration of interactions between pathogen races and resistant cultivars, the fitness of race j on cultivar i ($F_{i,j}$) was calculated as:

$$F_{i,j} = r_{i,j}/r_c, \quad (8)$$

where $r_{i,j}$ is the apparent infection rate of race j on the cultivar i, and r_c is the apparent infection rate on the most susceptible race–cultivar combination. These parameters were determined from specially designed field experiments (Zeng and Wang 1990). The relative amount of disease on cultivar i (XT_i) was summarized as:

$$\mathrm{XT}_i = \sum \mathrm{DX}_{i,j}, \quad (9)$$

Where, $\mathrm{DX}_{i,j}$ is the disease index (disease incidence (DI)×disease severity (DS)) on cultivar i caused by race j.

The proportion of the race subpopulation in a particular region and month RT_j, was estimated as:

$$\mathrm{RT}_j = \sum \left(\mathrm{DX}_{i,j} \times \mathrm{AC}_j \times \mathrm{LAI}_j\right) \Big/ \sum\sum \left(\mathrm{DX}_{i,j} \times \mathrm{AC}_j \times \mathrm{LAI}_j\right) \quad (10)$$

where AC_j, is the relative acreage of cultivar i and LAI_j is the relative leaf area index of cultivar i.

In simulations, eight nested cycles of calculations including year, month, target region, target cultivar, target race, source region, source cultivar, and source race, were involved. The two main components of the model include the computations of disease increments caused by local inoculum and those caused by exogenous inoculum for each region. Therefore, many various inoculum quantities of different races were considered in simulations. The outputs of the simulations included disease index on different cultivars in each region in different periods of time and frequencies of pathogen races (Zeng and Zhang 1990).

PANCRIN has been used recently to answer several important questions about regional disease epidemics and strategies for disease management in China (Zeng 2003b, 2004). One important regional disease management strategy which has been intensively discussed in China is the elimination of wheat cropping in the oversummering mountainous regions (Zeng and Luo 2006) (Fig. 1). To provide information about the determination of geographic boundaries of this strategy and its effects, PANCRIN was used to simulate disease development under different regional conditions, to help answer the question. Three major conclusions were: (1) the percentage of wheat fields abandoned will affect the strategy of disease control, (2) a minimum of 95% of the wheat fields should be abandoned, since conditions favourable to disease development could otherwise annihilate the effect of

abandonment, and (3) the geographic latitude (°NL) for stripe rust oversummering may vary with years. Another application of PANCRIN was to study strategies of regional cultivar deployment in order to delay the breakdown of resistance at a regional scale. Simulation results (Zeng 2004) demonstrated that (1) the planting area of each resistant cultivar should be <15% of the total planting area, (2) the number of resistant cultivars should be >6, (3) the planting area of susceptible cultivars should be <10%, and (4) when the new virulent race corresponding to the new vertically-resistant cultivar had a relative low aggressiveness, the new cultivar might exhibit a prolonged durability of resistance.

Improvement of PANCRIN may be implemented by including various weather variables, additional regions, cultivars, and races in order to consider more complicated scenarios of epidemics. For instance, to study the overwintering and oversummering of the pathogen, the divisions into epidemic regions could be refined according to the estimated oversummering and overwintering rates used as inputs. Intensive studies also made use of other models by Sun and Zeng (1994) to study interregional disease spread.

Modelling effects of cultivar deployment on disease management

Deployment of resistant cultivars at the regional scale as a management strategy can be studied by modelling to determine an efficient application of cultivar resistance. One method was to simulate pathogen multiplication for different races on a set of cultivars using parasitic fitness data. The frequency of the various races and the corresponding standard deviation of race frequency can be calculated after any given number of generations (Luo and Zadoks 1992). The optimal solution of cultivar deployment could be the strategy which involves proportions of cultivars that result in the greatest reduction of the standard deviation of pathogen frequencies after completion of a growing season. Such information is useful for decision making on cultivar deployment at the regional level. Data on frequencies of the major races in China from 1976 to 1989 and the percentages of six groups of cultivars grown in these years were used to generate a statistical model (Sun and Zeng 1993). In addition, Sun and Zeng (1995a) developed a model to simulate interregional disease spread duly considering cultivar–race interactions.

This review does not discuss the long-term dynamics of stripe rust races in China, an important topic to which other institutions in China contributed most information (Wang et al. 1986).

Conclusions and outlook

Although systems analysis used in this review allows us to gain an insight on epidemiological processes using available data as inputs to various simulation models, these models do not create new knowledge per se. However, the continuous improvement and testing of the models generates rationalisation and hierarchy of our knowledge and creates new ideas in an imaginative way. Decisive conclusions cannot always be expected, but new ideas and new research questions can be explored. Models can help new experiments. TXLX was a preliminary field-scale model which can be used to predict disease developments at a field level, to study the pathogen life cycle, fungicide applications and even slow-rusting epidemics. PANCRIN is a region-scale model which can be used to study interregional pandemics, shifts of pathogen races and regional strategies of resistance deployment. The inaccuracy in prediction of regional disease pandemics mainly came from a difficulty in the determination of the reasonable variation ranges of some parameters, such as the regional epidemic rate (R) (on susceptible cultivars), the relative parasitic fitness of races with combinations of cultivars (PF) and the disease dilution coefficient related to the long-distance aerial spore dispersal (DIL). Determination of PF requires field observations of the disease on specific cultivars caused by specific races to obtain disease dynamic data. Therefore under this circumstances, ‘field races’ (Zadoks 1961) are more meaningful than those resulting from race differentiation on seedlings. Improvement of the PANCRIN model needs further clarification of the applicable concept of PF and the methodology for its measurement in fields, since it involves the quantification of virulence and aggressiveness of pathogen races. It is still very difficult to determine DIL and its possible range by using current techniques. Although population genetics may help infer the pathway of pathogen dispersal, epidemiologists are still facing the difficulty to determine the quantitative dilution of the pathogen through long-distance dispersal. In long-term re-

search, as reported here on stripe rust epidemiology, old facts and new questions should be matched. To further develop the science of epidemiology, systems analysis should go hand-in-hand with scientific imagination and continued experimentation.

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