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Southern corn rust caused by *Puccinia polysora* Underw: a review

Qiuyu Sun, Leifu Li, Fangfang Guo, Keyu Zhang, Jiayu Dong, Yong Luo and Zhanhong Ma*

Abstract

Southern corn rust (SCR) caused by *Puccinia polysora* Underw is one of the most devastating maize diseases, resulting in substantial yield losses worldwide. The pathogen is an obligate biotrophic parasite that is difficult to culture on artificial media. In recent years, the disease has become prevalent—both globally and in China—and increasing difficult to control because of its wide distribution, long-distance migration, multiple physiological races and fast evolution, all of which have contributed to a considerable increase in the risks of associated epidemics. In this review, we summarize the current knowledge of *P. polysora*, with emphasis on its global distribution (particularly in China), life and disease cycle, population genetics, migration, physiological races, resistance genes in maize and management. Understanding the underlying factors and processes in SCR epidemics should facilitate management of the disease and breeding for resistant maize varieties.

Keywords: Southern corn rust, Distribution, Migration, Races, Resistant genes, Management

Background

Maize (*Zea mays* L.) is the most highly produced and economically important cereal crop globally. Owing to an increase in demand for maize in China, its production has surpassed by far those of rice and wheat (Wang et al. 2019). In 2019, the world's total yield of maize was 1,148 million tons, whereas rice and wheat yields were only 755 million and 766 million tons, respectively (FAO 2019). However, the annual yield loss attributed to southern corn rust (SCR) has significantly increased because of the lack of maize varieties with a high degree of resistance to SCR (Futrell et al. 1975; Brewbaker et al. 2011; Liu et al. 2016). Consequently, SCR poses a great threat to global maize production and, therefore, to global food security. In this review, we focus on the symptoms of SCR and the biological characteristics of the pathogen causing this disease, its distribution and migration trends, physiological races, resistance genes in maize and disease management. In general, our review aims to enhance our understanding and management of SCR.

Global distribution of SCR

Puccinia polysora, the causal agent of SCR, was first described and named by Underwood in 1897 (Underwood 1897), after it was first identified in 1891 on a herbarium specimen of eastern grama grass (*Tripsacum dactyloides* L.) in Alabama, USA (Underwood 1897). However, in 1879, Cummins had observed that SCR was widespread in Central and South America as well as in Massachusetts, USA, i.e., much earlier than the reported identification in 1891 (Krattiger et al. 1998). The pathogen shows enormous destructive potential in maize (Melching 1975). It was later reported that the pathogen can infect teosintes, including two *Erianthus* species and four *Tripsacum* species (Cammack 1959a). However, in early days, the role of *P. polysora* in epidemiology was unknown.

In 1949 and 1950, for the first time, SCR epidemics were reported in Sierra Leone, Liberia, Ivory Coast, Gold Coast, Dahomey, Togoland and Nigeria in West Africa, resulting in yield losses of up to 50% (Rhind et al. 1952).

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By 1952, SCR had reached Tanganyika, Uganda, Nyasaland and Kenya, and the east coast of Africa including the Republic of Mauritius which is in the Indian Ocean and is the most eastern point of disease migration, thereby posing a threat to maize production in Asia and Australia (Orian 1954); it was also reported in Canada in 1954 (Orian 1954). In 1953, it caused 80–84% yield losses in the Philippines (Reyes 1953). SCR was first noticed in south India in October 1957, with a mild infection in maize (Payak 1992). In 1959, SCR infestations were observed in Australia (Barker 1969). SCR emerged in Thailand in 1967 (Brewbaker et al. 2011). At the same year, the first authentic specimen of the disease was found in Hawaii (Raabe et al. 1981). In the 1900s, disease surveys carried out by the Brazilian Agricultural Research Corporation (Embrapa) revealed that SCR was one of the most important maize diseases and caused 40% losses in susceptible maize varieties (Bulow 1966; Krattiger et al. 1998). The epiphytotic of SCR extended to the main part of the maize belt in the United States during 1972–1974, causing 30%–50% yield losses (Futrell et al. 1975). By 1997, SCR had also become widespread in the Okinawa, Kyushu, Shikoku and Yamaguchiken prefectures of Japan (Hirayae et al. 1998). In addition, in 1951, a disease similar to SCR was described in Kumamoto, Japan (Nishi et al. 1998). In China, the disease was first identified in Hainan Province in 1972 (Duan and He 1984). According to an SCR distribution map published by the Center for Agriculture and Bioscience International (CABI), the disease has been found in more than 110 countries spanning Africa, Asia, the Americas and Australasia (CABI 2021; Fig. 1).

Temperature and humidity are the main factors influencing cereal rust spore germination, penetration, establishment and spread (Pavgi 1972). Potential future distribution of SCR was modeled using the CLIMEX software based on current geographic locations and temperature and humidity conditions. According to the results, SCR occurs in all continents; furthermore, future trends might incline towards a general reduction in incidence in the Southern Hemisphere concomitant with general increase in incidence in the Northern Hemisphere (Ramirez-Cabral et al. 2017).

SCR in China

In China, maize is widely cultivated in areas ranging from eastern coastal provinces to western provinces and from Hainan Province in the south to Heilongjiang Province in the north (Guo 2010). The area under maize cultivation in China can be divided into six regions (spring maize in the northeast area, summer maize in the Huanghuaihai area, southwestern mountainous area, southeastern maize area, tropical area and subtropical area). To increase production, numerous high-yield varieties are bred, such as Zhengdan 958, Xundan 20 and Xianyu 335, which have become the main cultivated corn varieties in

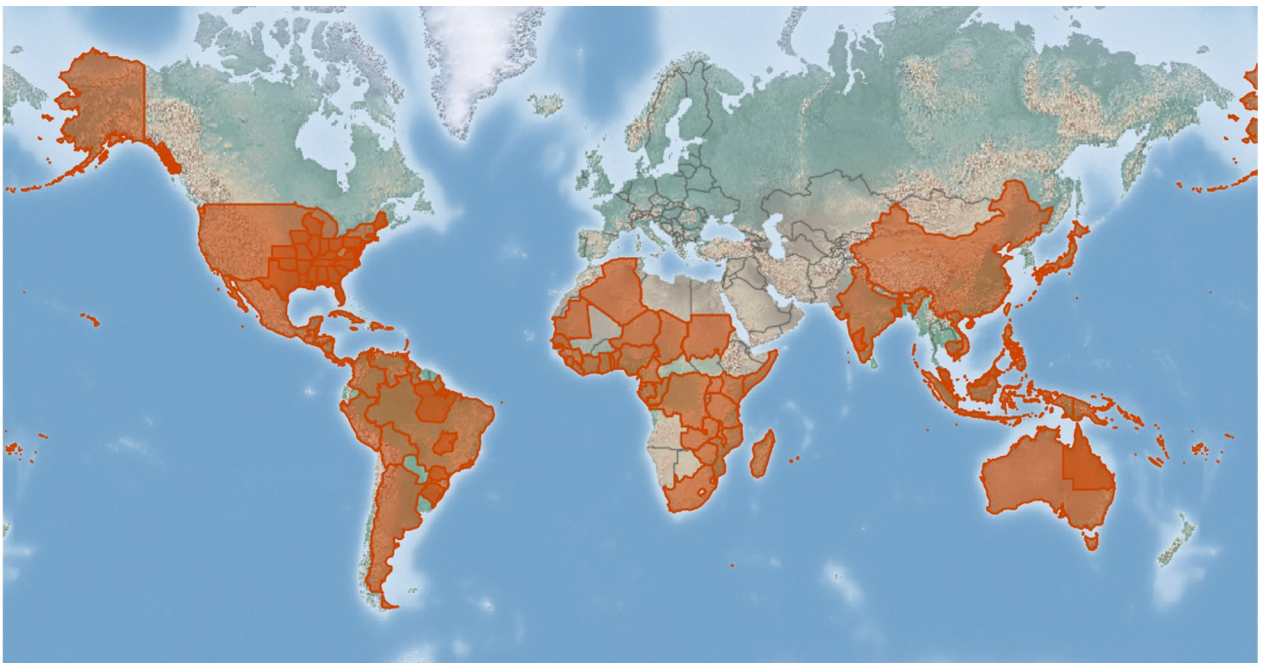

Fig. 1 Worldwide distribution of southern corn rust. The red areas represent the occurrence regions of southern corn rust (CABI 2021)

China; however, all these three varieties are susceptible to SCR (Liu et al. 2009). Indeed, Wang et al. (2006) investigated the resistance of 178 corn varieties to SCR, and reported that only 25(14%) varieties were highly resistant or resistant to SCR, whereas the majority (86%) were susceptible. In 1998, the disease outbroke in Hebei, Jiangsu, Shandong and Henan provinces. This was the first report of the disease in the northern areas of China since it was first identified in Hainan Province in 1972 (Duan and He 1984; Liu et al. 1998). At present, SCR has become a serious disease in more than 20 provinces/cities, including Hainan, Guangdong, Guangxi, Fujian, Jiangxi, Hunan, Guizhou, Yunnan, Chongqing, Hubei, Zhejiang, Anhui, Jiangsu, Henan, Shannxi, Shanxi, Shanghai, Shandong, Hebei, Tianjin, Beijing and Liaoning (Fig. 2). In our surveys, we observed that SCR occurred earlier in southern areas than in northern areas (unpublished data). Typhoons can efficiently enhance long-distance spore dispersal of *P. polysora*, and the landing pathway and duration of typhoon will determine the range and severity of SCR (Wang et al. 2020a). Owing to the cultivation of susceptible varieties over large areas and a favorable climate for disease development, SCR displays an aggravating tendency in China (Liu et al. 2018). In 2015, the affected area and yield losses caused by SCR were 5.289 million hm^2 and 756 million kg, respectively, which were 4.5-fold and 8.8-fold great than the annual averages of 2008–2014 (Liu et al. 2016; Fig. 3).

Symptoms and biological characteristics

P. polysora is an obligate parasite that infects, grows and reproduces only on living maize plant tissues (Unartngam et al. 2011). The pathogen infects all the aboveground parts of maize plants including leaves, sheaths and stems (Fig. 4). Its damage is considerably more devastating and lethal than common rust damage caused by *Puccinia sorghi* Schw. (Scott et al. 1984). Pustules of *P. polysora* are primarily produced on the upper leaf surfaces, distinguishing them from common rust whose pustules develop on both sides (Crouch and Szabo 2011). Urediniospores of SCR are one-celled, yellowish to golden, echinulate, with 4–5 equatorial pores; whereas teliospores are two-celled, chestnut brown, angular to ellipsoid or oblong (Krattiger et al. 1998). When urediniospores of these two fungi are treated with 95% sulfuric acid or 75% hydrochloric acid, the protoplast of *P. polysora* contracts into the shape of a ball but that of *P. sorghi* contracts to resemble several balls—a difference that can be used to distinguish between these two species (Huang et al. 2020). The sporulation of urediniospores from a single pustule could last 18–20 days, which can release 1,500–2,000 and 600–1,150 spores daily from susceptible and resistant plants, respectively (Cammack 1962). Teliospores are produced in the late season or not at all and are covered by the epidermis, rendering the observation more difficult (King and Scott 1982; Crouch and Szabo 2011).

SCR thrives under high temperature and relative humidity (Shurtleff 1980). The optimal temperatures for the germination of urediniospores and disease development are 26–28 °C and 24–27 °C, respectively; the temperature range for disease occurrence and development is 15–31 °C (Yang et al. 2015). Li et al. (2019) reported that the density of urediniospores in the air was significantly and negatively correlated with temperatures above 27 °C and sunshine duration. Similarly, Godoy et al. (2003) found that the epidemic rate of SCR in Brazil was positively correlated with the mean daily temperature but negatively correlated with sunshine hours when relative humidity was > 90%. In addition, analysis of the biochemical parameters of the pathogen revealed that SCR is a high-sugar disease (Huang et al. 2012).

Life cycle and disease cycle

The life cycles of most cereal rusts are well understood (Jin et al. 2010). However, the life cycle of *P. polysora* remains unclear (Cammack 1959a; Guo et al. 2013). Teliospores are rarely produced in mature plants or not produced at all (Crouch and Szabo 2011), and are sometimes covered with an epidermis and do not dehisce, which makes it difficult to find them (Crouch and Szabo 2011). Series experiments to induce teliospore germination have been unsuccessful (Cammack 1962; Brewbaker et al. 2011). Therefore, the function of teliospores in the fungal life cycle is unclear, although teliospores might play a role in pathogen survival. Urediniospores serve as the primary inoculum sources for maize infection, known as the mini cycle of *P. polysora* (Brewbaker et al. 2011), but the aecidial and pycnidial stages of *P. polysora* remain unknown (Cammack 1959a).

In China, SCR can occur in most summer maize areas where ambient temperature favors the disease development. We found that the causing pathogen, *P. polysora*, can overwinter and survive throughout the year with a continuity of the uredo-stage in the southern areas of China, where maize can be planted throughout the year (Fig. 5). The wind transports urediniospores from such southern areas to the major summer maize-producing area in the Huanghuaihai region of China, where the pathogen cannot overwinter.

Population genetics

General knowledge regarding the levels of genetic diversity in *P. polysora* populations remains limited. In Japan, at least two genetic groups were found based on genetic diversity analysis of 14 isolates using restricted

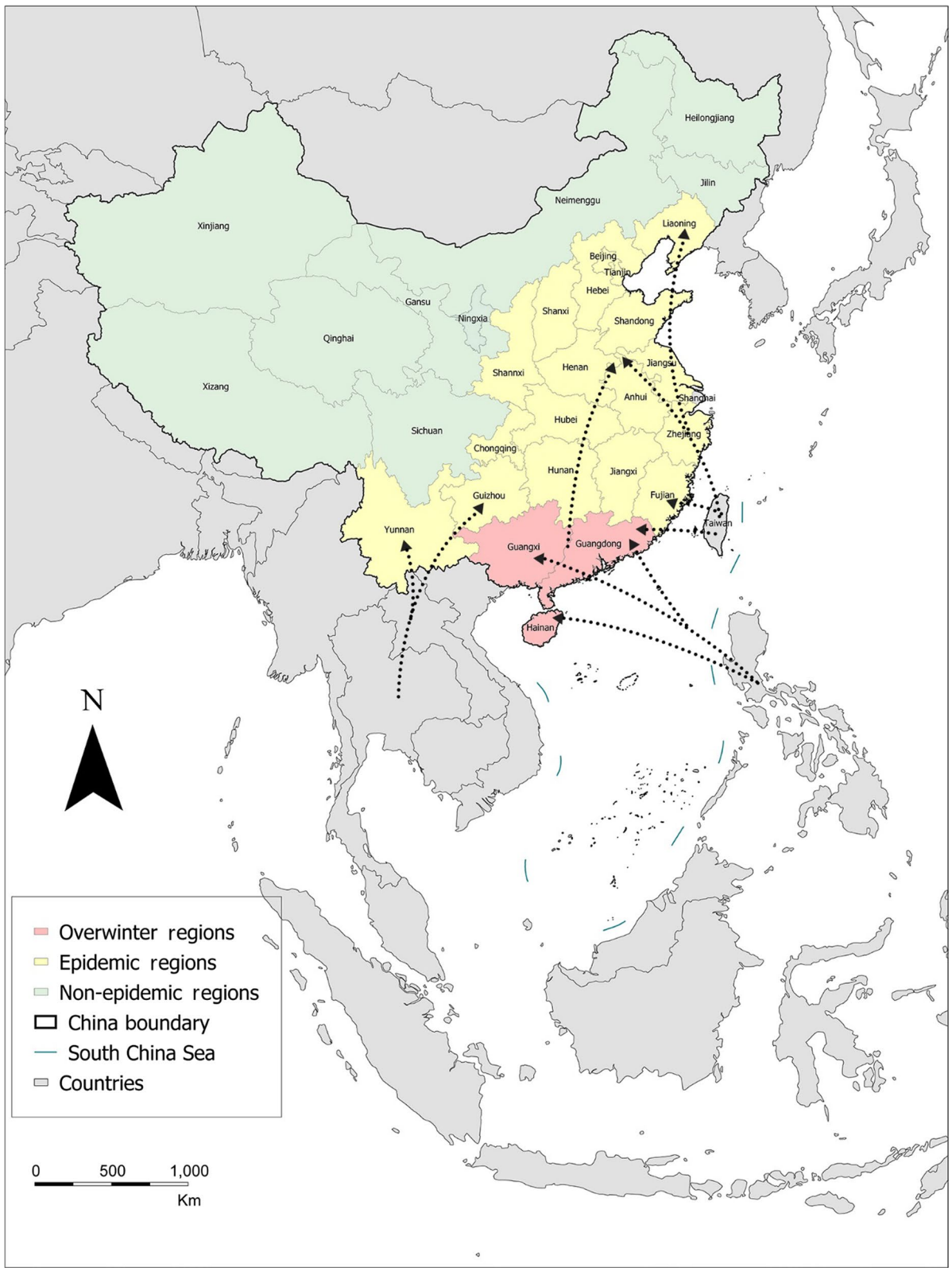

Fig. 2 The distribution of *Puccinia polysora* and the possible resources of primary infection of *P. polysora* causing southern corn rust in China. Broken black arrows indicate the possible pathway of urediniospores dispersed by the wind (Liu et al. 2018; Wang et al. 2020a)

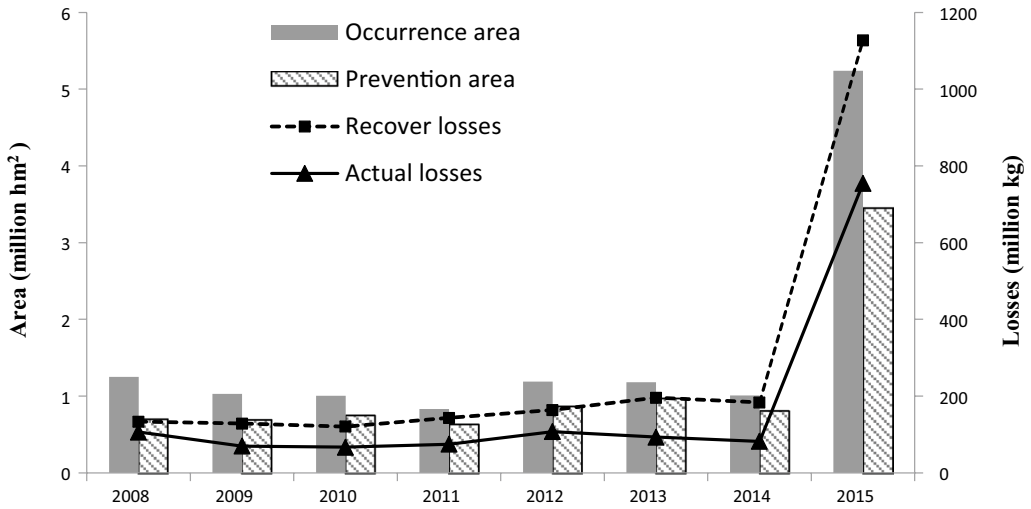

Fig. 3 The occurrence area and prevention area of southern corn rust, and actual losses and recover losses of maize in China during 2008–2015 (Liu et al. 2016)

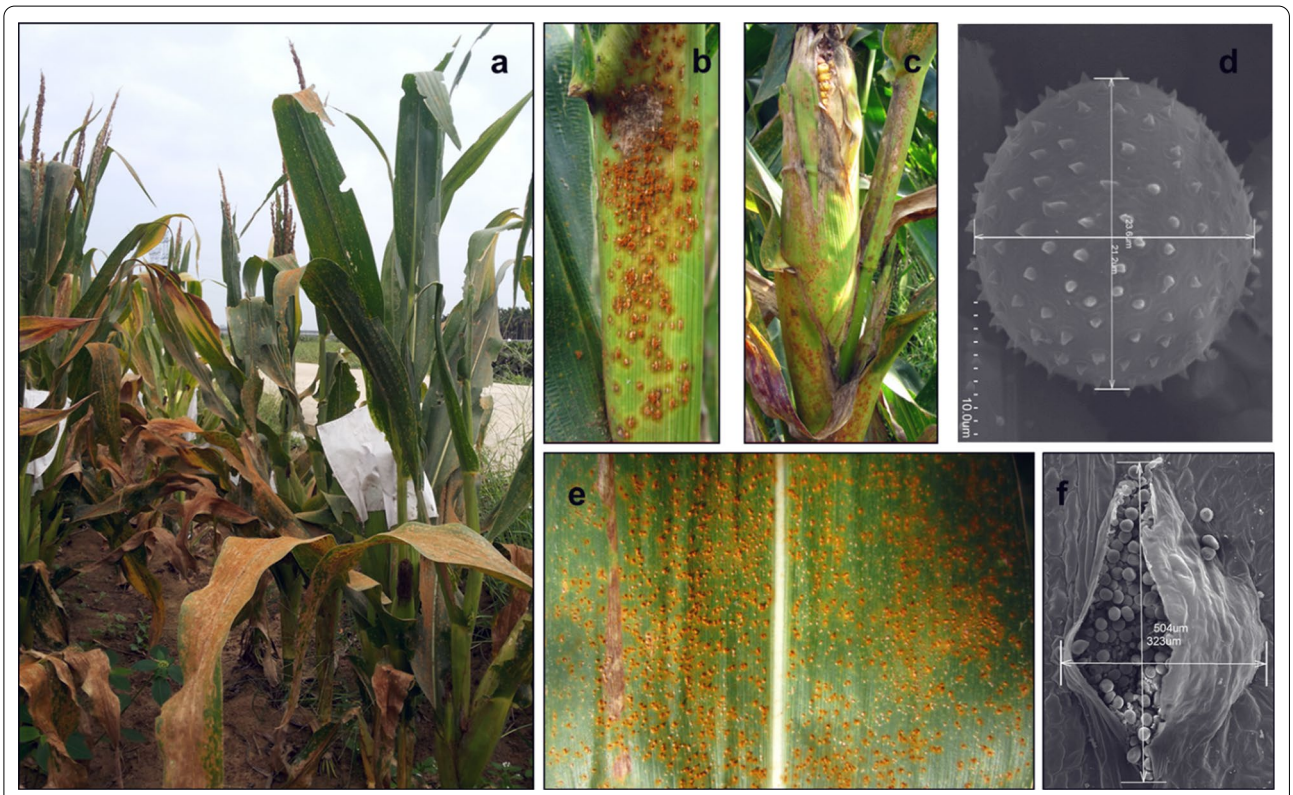

Fig. 4 Southern corn rust symptoms. **a** Urediniums on adult maize plants. **b** Urediniums on the sheath. **c** Urediniums on the sheaths, bracts and stem. **d** A urediniospore under a scanning electron microscope (SEM). **e** Urediniums on a maize leaf. **f** A uredinium under a SEM

fragment length polymorphisms (RFLP) of the internal transcribed spacer 1 (ITS1) and ITS2 regions (Hirayae et al. 1998). Similarly, in Thailand, Unartngam et al. (2011) divided 38 specimens from different provinces into 13 groups at 75% Dice's similarity coefficient based on the results of genetic diversity analyses of *P. polysora*

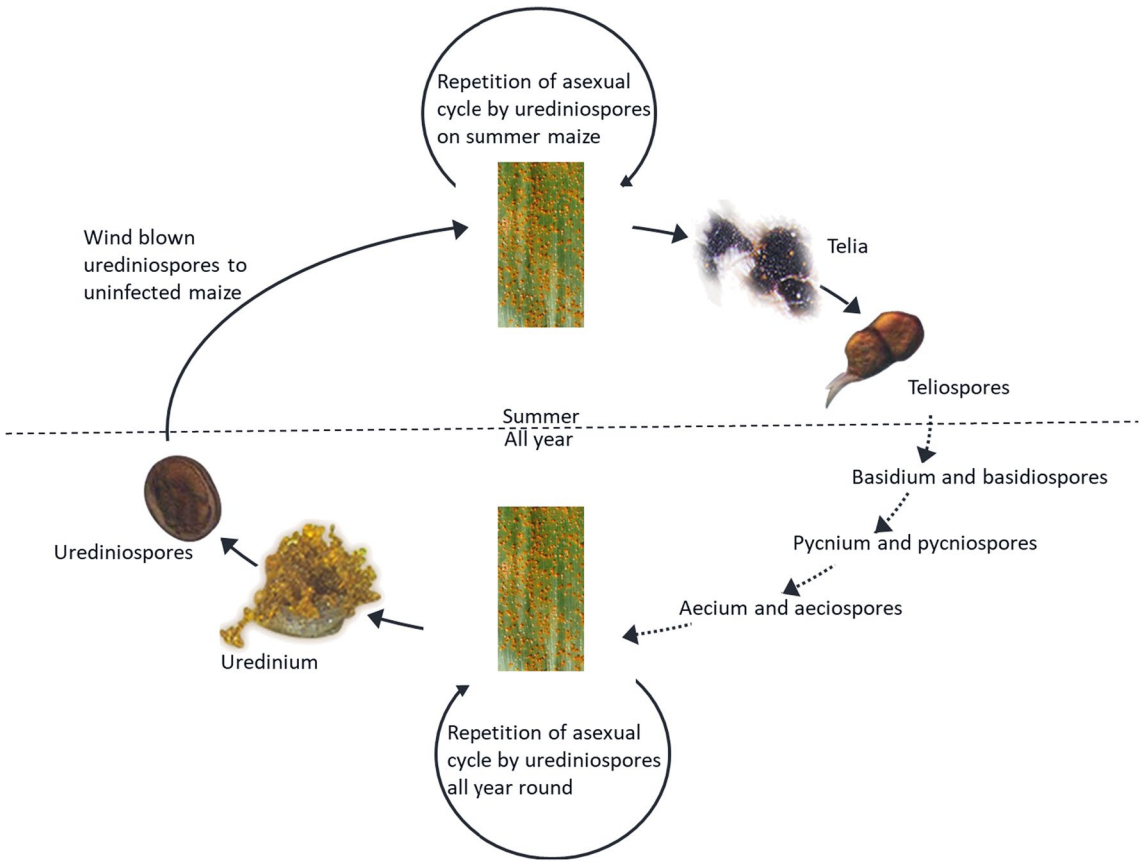

Fig. 5 Disease cycle of southern corn rust in China. Two types of disease symptoms were observed on maize, the uredinial stage with corresponding urediniospores and the telial stage with teliospores (Crouch and Szabo 2011). The disease symptoms at the basidial, pycnial and aecial stages appearing on an alternate host are unknown. The pathogen can survive and repeat its asexual cycle by urediniospores throughout the year in south. In summer, the wind transports urediniospores from south to north where the pathogen cannot over-winter

using five inter-simple sequence repeat (ISSR) markers. They found that isolates from different provinces were presented in the same group and those from each province were clustered into more than one group, indicating that the genetic diversity of the isolates has no correlation with their geographical distribution. In turn, in China, the similarity of the ITS and *β-tubulin* sequences of the isolates sampled in Hainan, Henan and Chongqing was 99.12%–100% and 98.97%–99.91%, respectively (Xing 2011). However, in another study, a total of 72 isolates collected from 12 provinces in China were clustered into 2 groups and 5 subgroups of 10 branches, with high genetic diversity, based on 18 polymorphic ISSR primers (Guo et al. 2013). The results showed that all the isolates collected in 2011 and eight isolates collected in 2012 were clustered in one group, but the other isolates collected in 2012 were clustered in another group, suggesting some relationships in the subgroup population based on collected loci and years.

P. polysora migration

P. polysora urediniospores can be dispersed by wind currents to elevations higher than 15,000 ft and over long distance (Orian 1954). Cammack (1958) identified two main size groups of urediniospore—namely, a small size group in Southeast Asia and neighboring islands and a larger-size group in the West Indies, Africa and the South Indian Ocean—consisting with two directions (eastwards and westwards) of migration from their origin in the region of the Caribbean. However, the examination lacked data on cross-inoculation studies, which indicated that it is uncertain if distinct forms of *P. polysora* exist. Cammack (1959b) speculated that urediniospores were the only source of infection dispersing from the Caribbean to Africa. In Thailand, specimens collected from different provinces were clustered in the same group because of urediniospore migration (Unartngam et al. 2011).

In China, no significant sub-population differentiation has been found among Hainan, Henan and Chongqing, implying potential frequent gene flows among these places (Xing 2011). However, Guo et al. (2013) reported low gene flows among provincial populations, and the pathogens in Hainan, Guangdong and Guangxi were not the original sources of the disease in summer maize in the Huanghuaihai area, indicating that the pathogen in China may originate from other areas outside the mainland of China. Conversely, Liu et al. (2018) inferred that *P. polysora* emergence in Tianjin was likely attributable to long-distance wind dispersal from southern China. In addition, according to Wang et al. (2020a), Taiwan, China, is the original source of the pathogen found in the Huanghuaihai region, and Liaoning, Zhejiang, Fujian, and Guangdong provinces; the Philippines is the source of the pathogen in Guangdong, Guangxi and Hainan; and Thailand and other neighboring countries are the sources of the pathogen occurring in Yunnan and Guizhou provinces (Fig. 2). Based on the previous studies and our surveys of *P. polysora*, we inferred that the pathogen in the southern area in China where the disease can over-winter could be the source of SCR occurred in summer maize in the Huanghuaihai area of China (Liu et al. 2018; Wang et al. 2020a; Fig. 2).

Physiological races

Several physiological races of *P. polysora* have been identified. In 1952, EA1, the first physiological race of *P. polysora*, was recognized from urediniospore collections sampled in Zanzibar, Tanganyika and Uganda (Storey and Howland 1957). The EA2 race was subsequently identified in Zanzibar, Northern Tanganyika and Uganda using four maize lines in 1955 (Ryland and Storey 1955), whereas the EA3 race was found in Kenya in 1961 (Storey and Howland 1967a). Six novel physiological races, PP.3, PP.4, PP.5, PP.6, PP.7 and PP.8, distinct from the East African races, were identified in Georgia, Nicaragua, Colombia, Mexico, Florida, Nicaragua and Puerto Rico of North and Central America, using 11 maize cultivars as differentials (Robert 1962). The tenth race, PP.9, was identified in South Africa in 1965 (Ullstrup 1965). In Brazil, 17 virulence patterns were identified in 60 single pustule isolates using six maize cultivars (Casela and Ferreira 2002). SCR occurs globally; however, race identification of *P. polysora* has been performed using maize differentials with different genetic sources (Storey and Howland 1957; Robert 1962; Ullstrup 1965; Casela and Ferreira 2002). Owing to the lack of unique differentials and a high variability of the causing pathogen, the identification and application of resistant genes against SCR in maize plants have been limited. Further, in tropical regions, the constant failure of studies on race-specific resistance to SCR has been attributed to the occurrence of multiple races of the pathogen in tropical areas (Casela and Ferreira 2002). According to Keller et al. (2001), race-specific resistance is ineffective, unstable and short-lived owing to the rapid evolution of the pathogen. Therefore, there is an urgent need to identify and classify the physiological races and virulence patterns of *P. polysora.*

SCR-resistant genes

The identification of resistance genes can facilitate maize breeding and production programs (Babu et al. 2004). The identification of SCR-resistant genes began as early as the 1950s. In 1957, *Rpp1* and *Rpp2,* two genes conferring resistance to two physiological races of *P. polysora* EA1 and EA2 were identified from AFRO.29 (Colombia 2) and AFRO.24 (SLP 20-4A) maize lines originating in Columbia and Mexico, respectively (Storey and Howland 1957, 1959). *Rpp1* is completely dominant and confers high resistance to EA1, but not to EA2, conversely, *Rpp2* is incompletely dominant and confers equal resistance to these two races (Storey and Howland 1957). In addition, neither *Rpp1* nor *Rpp2* exhibited resistance to EA3. Backcross segregation, repulsion and coupling studies on seedlings inoculated with EA1 and EA2 revealed that *Rpp1* and *Rpp2* are linked, with an estimated recombination rate of 12.23% (Storey and Howland 1959). *Rpp3*–*Rpp8* were identified by Robert (1962) using a series of maize lines. *Rpp9,* which is closely linked to *Rpp1,* was from PT186208, with resistance to the PP.9 race isolated in Indiana (Scheffer and Ullstrup 1965). *Rpp10* and *Rpp11* were isolated from AFRO.761 (Andaqui, Colombia) and AFRO.600 (Zapalote Chico, Mexico) maize lines, respectively, of which *Rpp10* is fully dominant and exhibited high resistance to both EA1 and EA3, whereas *Rpp11* is incompletely dominant and exhibited incomplete resistance to EA1 and EA3 (Storey and Howland 1967b).

Recently, numerous studies have shown that major quantitative trait loci (QTL) and SCR resistant genes are mapped to the short arm of chromosome 10 (Table 1) (Liu et al. 2003; Chen et al. 2004; Zhang et al. 2009; Yao et al. 2013; Zhang 2013; Wu et al. 2015; Wang et al. 2020b; Meng et al. 2021). Effective identification of the loci for SCR resistance-related genes will facilitate the breeding of SCR-resistant maize varieties. *RppP25*, a major resistance gene from the inbred maize line P25 (China) was roughly mapped on chromosome 10S with a 5.8 cM genetic distance from the simple sequence repeat (SSR) marker phi059 (Liu et al. 2003). In addition, a single dominant gene, *RppQ*, was identified from the Qi319 (China) maize line, and was mapped on the short arm of chromosome 10S between the SCAR marker MA7 and the AFLP marker M-CCG/E-AGA157 with a genetic distance of 0.46 and 1.71 cM, respectively. (Chen et al. 2004;

Table 1 Summary of SCR-resistant genes

Gene	Race of *P. polysora*	Maize variety/line	Country	Chromosome	References
Rpp1	EA1	AFRO.29	Columbia		Storey and Howland (1959)
Rpp2	EA1, EA2	AFRO.24 (SLP 20-4A)	Mexico		Storey and Howland (1959)
Rpp3–Rpp8	PP.3–PP.8				Robert (1962)
Rpp9	PP.9	PT186208	South African		Scheffer and Ullstrup (1965)
Rpp10	EA1, EA3	AFRO.761	Colombia		Storey and Howland (1967b)
Rpp11	EA1, EA3	AFRO.600	Mexico		Storey and Howland (1967b)
RppP25		P25	China	10S	Liu et al. (2003)
RppQ		Qi319	China	10S	Chen et al. (2004)
RppD		W2D	China	10S	Zhang et al. (2009)
RppC		CML470	CIMMYT	10S	Yao et al. (2013)
Rpp12		Jiku12	China	10S	Zhang (2013)
RppS		SCML205	China	10S	Wu et al. (2015)
RppM		Kangxiujing2416 (Jing2416k)	China	10S	Wang et al. (2020b)
RppCML496		CML496	CIMMYT	10S	Meng et al. (2021)

Zhou et al. 2007). Additionally, genetic analysis revealed that the resistance of the inbred line W2D (China) was controlled by a dominant *RppD*, which was mapped on the short arm of chromosome 10S flanked by the SSR marker UMC1291 and the CAPS marker CAPS858 with a genetic distance of 2.9 and 0.8 cM, respectively (Zhang et al. 2009). Furthermore, in the inbred line CML470 developed by CIMMYT, a dominant *RppC* was identified, which was mapped on the short arm of chromosome 10S in the region between the SSR markers UMC1380 and UMC1291 with a genetic distance of 3.5 and 8.8 cM, respectively. (Yao et al. 2013). Similarly, the Jiku12 (China) resistance gene was identified as a dominant *Rpp12* distinct from *RppQ* and mapped on the chromosome 10S with a genetic distance of 4.2 cM away from the adjacent molecular marker PHI063 (Zhang 2013). In turn, the tropical inbred line SCML205 (China), harbors a single dominant resistance gene, *Rpps*, which was mapped on the distal arm of chromosome 10S (Wu et al. 2015). In addition, Wang et al. (2020b) identified and mapped a resistance gene, *RppM*, from the near-isogenic line Kangxiujing2416 (Jing2416K) to a 3.69 Mb region on chromosome 10S, and the *RppCML496* resistance gene was detected in CML496 (CIMMYT) and delimited to a 128 kb interval on chromosome 10 (Meng et al. 2021). Furthermore, two major resistance QTLs, *qSCR6.01* and *qSCR10.01*, were identified from the Qi319 (China) maize line (Lu et al. 2020). Particularly, unlike the previous identified major genes/QTLs for SCR resistance on chromosome 10, *qSCR6.01* is the first identified major QTL associated with resistance to SCR on chromosome 6.

In addition to the major genes/QTLs abovementioned, QTLs with partial resistance to SCR have been reported based on linkage or association mapping, which offer additional genetic resources and information essential for maize breeding (Chen et al. 2019; Deng et al. 2019; Meng et al. 2021). For example, using recombinant inbred lines derived from a cross between Ye 478 and Qi319, Lu et al. (2020) identified three QTLs designated as *qSCR3.04*, *qSCR5.07* and *qSCR9.03* on chromosomes 3, 5 and 9, respectively, which effectively explained 2.84% to 3.11% of the total phenotypic variation in each QTL. In addition, Deng et al. (2020) reported a major QTL named Ascr4.01 on chromosome 4, which can explain 48% to 65% of the total phenotypic variation, based on a recombinant inbred line derived from a cross between CIMBL83 and Lx9801. QTLs with minor effects or partial resistance to SCR have been detected on all maize chromosomes, which provide more valuable source for SCR resistance (Brewbaker et al. 2011; Wanlayaporn et al. 2013; Zhou et al. 2018; de Souza Camacho et al. 2019; Chen et al. 2019; Deng et al. 2019, 2020; Lu et al. 2020).

The resistance genes *Rpp1–Rpp11* appear race-specific, whereas the other resistance genes exhibit complete resistance to SCR. The pathogen evolves rapidly, so that new race overcomes the resistance gene like *Rpp9,* which is used to breed resistant maize varieties in Southern US. These findings highlight the need for identifying novel resistance resources (Brewbaker et al. 2011).

Disease control and integrated management

The use of resistant crop varieties is considered an effective and environmentally friendly strategy for disease management. In China, several maize varieties, such as Nongda108, Zhengda619, Chengdan22 and Jundan20, have been identified to be resistant or highly resistant to SCR (Yuan et al. 2010). Intercropping can also reduce the severity of the disease. Liu et al. (2013) compared infection severity and defense-related enzyme activity in mono-cropping and inter-cropping systems inoculated with SCR, and according to the results, inter-cropping significantly enhanced defense and resistance to SCR.

Disease prediction models and monitoring networks are used as early warning systems in disease management schemes (Isard et al. 2011; Liu et al. 2015). Additionally, a molecular detection system for monitoring SCR has also been established (Leng et al. 2012), and spectral disease indices (SDIs)-based monitoring models have been developed for evaluating infected leaves and classifying the severity of SCR damage. Such models have provided a sound theoretical basis for remote sensing of SCR in the field (Meng et al. 2020). Additionally, a prediction model has been established for the prediction of SCR disease index (Li et al. 2019). Indeed, Spurlock et al. (2020) observed a significant positive correlation between relatively high normalized difference vegetation index (NDVI) and SCR severity based on remote sensing analysis, which facilitated the development of a more reliable SCR scouting model.

Chemical control is used in preventing the epidemic of plant diseases. Although, there is no SCR-specific fungicide, in the maize booting stage, 12.5% epoxiconazole SC effectively controlled SCR damage by up to 85.54%, which was only 20%–40% under conventional fungicides, such as triadimefon and zineb (Hu et al. 2003). However, Faske and Emerson (2021) found that the application of quinone outside inhibitor (Qol) or Qol + demethylation inhibitor (DMI) fungicides at tasseling stage were most effective when SCR was present and environmental conditions favored rust development. In addition, a combination of triazole and estrobirulina was feasible to reduce SCR severity (Moratelli et al. 2015).

Conclusions and future perspectives

SCR is a major maize disease reported globally. The wide occurrence of this disease and the resulting yield losses are causes for deep concern from all stakeholders, including researchers. The analysis of the biological characteristics, distribution, races and genetic structure of the causing pathogen, *P. polysora*, in various regions has offered key insights into its historical and contemporary emergence, revolution and also migration trends. Considering it is highly invasive and potentially catastrophic, globally concerted efforts are needed to prevent pathogen incidence and spread. In the past, physiological races of *P. polysora* were identified using maize differential lines. However, due to the loss of two maize lines, the classification of races was suspended (Guo et al. 2013), resulting in the discovery and mapping of resistant genes based on complete resistance to SCR rather than race-specific resistance. The establishment of a novel differential host system is required for the identification of new physiological races, evaluation of spatiotemporal dynamics of races, and cultivation of resistant varieties. Studies on *P. polysora* migration based on population genetics are scarce now. Furthermore, the lack of useful genetic markers for *P. polysora* impedes research on the migration of the disease. Nevertheless, the development of SSR and single nucleotide polymorphism (SNP) markers would facilitate an understanding of pathogen population diversity and migration. Integrated management strategies are required for SCR. Furthermore, a convenient, cost-effective, specific and rapid diagnostic tool for predicting and monitoring disease incidence should be developed. Currently, our understanding of the alternate host of *P. polysora* and the roles of teliospores in host–pathogen interactions is inadequate. Considering that mining resistance genes are critical for breeding SCR-resistant maize varieties, the maize germplasm available globally should be screened to identify novel SCR-resistant genetic sources.

Abbreviations

CIMMYT: Centro Internacional de Mejoramiento de Maíz y Trigo/ International Maize and Wheat Improvement Center; DMI: Demethylation inhibitor; ISSR: Inter-simple sequence repeat; ITS: Internal transcribed spacer; NDVI: Normalized difference vegetation index; Qol: Quinone outside inhibitor; QTL: Quantitative trait loci; SCR: Southern corn rust; RFLP: Restricted fragment length polymorphisms; SDIs: Spectral disease indices; SNP: Single nucleotide polymorphism; SSR: Simple sequence repeat.

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Authors' contributions

QS, YL and ZM conceived and designed the study. QS collected the data and wrote the paper. LL, FG, KZ and JD helped in collecting the data. All authors read and approved the final manuscript.

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Availability of data and materials

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Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication
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Competing interests
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References

Babu R, Nair SK, Prasanna BM, Gupta HS. Integrating marker-assisted selection in crop breeding-prospects and challenges. Curr Sci. 2004;87:607–19.
Barker SJ. Testing of maize hybrids resistant to *Puccinia polysora* on the Atherton Tableland, Queensland. Queensl J Agric Anim Sci. 1969;26:319–27.
Brewbaker JL, Kim SK, So YS, Logrono M, Moon HG, Ming R, et al. General resistance in maize to southern rust (*Puccinia polysora* Underw.). Crop Sci. 2011;51:1393–409. https://doi.org/10.2135/cropsci2010.06.0327.
Bulow JFWV. As ferrugens *(Puccinia sorghi, P. polysora*, *Physopella zeae*) do milho (*Zea mays*). I Revisão Bibliográfica Pesqui Agropecu Bras. 1966;1:249–62.
CABI. *Puccinia polysora*. In: Invasive species compendium. Wallingford, UK:CAB International. 2021. https://www.cabi.org/isc.
Cammack RH. *Puccinia polysora*: A review of some factors affecting the epidemiology in West Africa. In: Proceedings of sixth commonwealth mycology conference 1962;134–8.
Cammack RH. Studies on *Puccinia polysora* Underw: III. Description and life cycle of *P. polysora* in West Africa. Trans Br Mycol Soc. 1959a;42(1):55–8. https://doi.org/10.1016/S0007-1536(59)80067-X.
Cammack RH. Studies on *Puccinia polysora* underw: I. The world distribution of forms of *P. polysora*. Trans Br Mycol Soc. 1958;41(1):89–94. https://doi.org/10.1016/S0007-1536(58)80011-X.
Cammack RH. Studies on *Puccinia polysora* underw: II. A consideration of the method of introduction of *P. Polysora* into Africa. Trans Br Mycol Soc. 1959b;42(1):27–32. https://doi.org/10.1016/S0007-1536(59)80062-0.
Casela CR, Ferreira AS. Variability in isolates of *Puccinia polysora* in Brazil. Fitopatol Bras. 2002;27(4):414–6. https://doi.org/10.1590/S0100-41582002000400015.
Chen CX, Wang ZL, Yang DE, Ye CJ, Zhao YB, Jin DM, et al. Molecular tagging and genetic mapping of the disease resistance gene *RppQ* to southern corn rust. Theor Appl Genet. 2004;108:945–50. https://doi.org/10.1007/s00122-003-1506-7.
Chen WJ, Lu L, Zhang XJ, Li WC, Sun SL, Zhu ZD, et al. QTL mapping for resistance to southern corn rust in maize. J Plant Genet Resour. 2019;20(3):521–9. https://doi.org/10.13430/j.cnki.jpgr.20180831001 (in Chinese).
Crouch JA, Szabo LJ. Real-time PCR detection and discrimination of the southern and common corn rust pathogens *Puccinia polysora* and *Puccinia sorghi*. Plant Dis. 2011;95(6):624–32. https://doi.org/10.1094/PDIS-10-10-0745.
de Souza Camacho LR, Coan MMD, Scapim CA, Pinto RJB, Tessmann DJ, Contreras-Soto RI. A genome-wide association study for partial resistance to southern corn rust in tropical maize. Plant Breed. 2019;138(6):770–80. https://doi.org/10.1111/pbr.12718.
Deng C, Li H, Li Z, Tian Z, Chen J, Chen G, et al. New QTL for resistance to *Puccinia polysora* Underw in maize. J Appl Genet. 2019;60:147–50. https://doi.org/10.1007/s13353-019-00488-w.
Deng C, Lv M, Li X, Zhao X, Li H, Li Z, et al. Identification and fine-mapping of *qSCR4.01*, a novel major QTL for resistance to *Puccinia polysora* Underw in maize. Plant Dis. 2020;104(7):1944–8. https://doi.org/10.1094/PDIS-11-19-2474-RE.
Duan DR, He HZ. Description of a rust *Puccinia polysora* on corn in Haian island. Acta Mycol Sin. 1984;32:125–6. https://doi.org/10.1334/j.mycosystema.1984.02.011 (in Chinese).
FAO. FAOSTAT-Agriculture, Food and Agricultural Organizations of the United Nations, 2019. http://faostat3.fao.org/brpwse/Q/QC/E. Accessed on 19 July 2021.
Faske TR, Emerson M. Multiyear evaluation of fungicide efficacy and application timing for control of southern rust in hybrid corn in Arkansas. Plant Dis. 2021;105(4):1108–14. https://doi.org/10.1094/PDIS-06-20-1247-RE.
Futrell MC, Hooker AL, Gene SE. Resistance in maize to corn rust, controlled by a single dominant gene. Crop Sci. 1975;15(4):597–9. https://doi.org/10.2135/cropsci1975.0011183X001500040044x.
Godoy CV, Amorim L, Filho AB, Silva HP, Silva WJ, Berger RD. Temporal progress of southern rust in maize under different environmental conditions. Fitopatol Bras. 2003;28(3):273–9. https://doi.org/10.1590/S0100-41582003000300008.
Guo QH. The development and evolution of the major maize producing areas in China. J Maize Sci. 2010;18(1):139–45. https://doi.org/10.13597/j.cnki.maize.science.2010.01.034 (in Chinese).
Guo YY, Chen MG, Sun SL, Wu XF, Jiang K, Zhu ZD, et al. Genetic diversity of *Puccinia polysora* Underw. in China. Sci Agric Sin. 2013;46(21):4523–33. https://doi.org/10.3864/j.issn.0578-1752.2013.21.015 (in Chinese).
Hirayae K, Kawase A, Umeda Y, Nakatani D, Yamaguchi T, Nishi K. Genetic variation of southern rust fungus of corn in Japan. Kyushu Plant Prot Res. 1998;44:12–4. https://doi.org/10.4241/kyubyochu.44.12 (in Japanese).
Hu WY, Zheng MX, Ruan YL, Pan FY, Yu BJ, Yu YF, et al. Preliminary studies on the incidences rules of Puccinia polysora. China Plant Prot. 2003;12:9–12. https://doi.org/10.3969/j.issn.1672-6820.2003.12.004 (in Chinese).
Huang FY, Yu K, Li WC, Zhu ZD, Li HJ, Wu XF, et al. Relationship of biochemical parameters of corn inbred lines and their resistance to southern corn rust. J Maize Sci. 2012;20(2):138–43. https://doi.org/10.13597/j.cnki.maize.science.2012.02.030 (in Chinese).
Huang LQ, Zhang KY, Dong JY, Sun QY, Li LF, Ma ZH. A fast method for distinguishing southern rust pathogen *Puccinia polysora* from common rust pathogen *Puccinia sorghi*. J Plant Prot. 2020;47(6):1385–6. https://doi.org/10.1380/j.cnki.zwbhxb.2020.2020096 (in Chinese).
Isard SA, Barnes CW, Hambleton S, Ariatti A, Russo JM, Tenuta A, et al. Predicting soybean rust incursions into the north American continental interior using crop monitoring, spore trapping, and aerobiological modeling. Plant Dis. 2011;95(11):1346–57. https://doi.org/10.1094/PDIS-01-11-0034.
Jin Y, Szabo LJ, Carson M. Century-old mystery of *Puccinia striiformis* life history solved with the identification of *Berberis* as an alternate host. Phytopathology. 2010;100:432–5. https://doi.org/10.1094/PHYTO-100-5-0432.
Keller B, Feuillet C, Messmer M. Genetics of disease resistance. In: Slusarenko AJ, Fraser RSS, van Loon LC, editors. Mechanisms of resistance to plant diseases. Dordrecht: Springer; 2001. p. 100–60. https://doi.org/10.1007/978-94-011-3937-3_5.
King SB, Scott GE. Development of southern rust on maize at different stages of maturity. Plant Dis. 1982;66:477–81. https://doi.org/10.1094/PD-66-477.
Krattiger A, Kulisek ES, Casela C. Introduction and overview: diagnosing maize diseases with proprietary biotechnology applications transferred from pioneer Hi-Bred international to Brazil and Latin America. In: Casela C, Renfro B, Krattiger AF, editors. Diagnosing maize diseases in Latin America. ISAAA Briefs No. 9. Ithaca, NY and EMBRAPA, Brasilia: ISAAA, 1998. pp 1–4
Leng WF, Li GZ, Ma ZH. Distribution and molecular detection of corn rust in China. China Plant Prot. 2012;32(12):18–20 (in Chinese).
Li XH, Zhang KY, Li LF, Zhang SM, Luo Y, Ma ZH. Correlation between *Puccinia polysora* Underw. spore number and disease index and meteorological factors. Acta Phytopathol Sin. 2019;49(3):362–9.
Liu J, Jiang Y, Zeng J, Ji G, Liu L, Qiu K, et al. Analysis of the severe occurrence characteristics and causes of the southern corn rust in China in 2015. China Plant Prot. 2016;36(5):44–7 (in Chinese).
Liu J, Ma Q, Yu K, Wang XM. Distribution of southern corn rust and corn varieties resistance to the rust in China. Crops. 2009;3:71–5 (in Chinese).
Liu Q, Gu YL, Wang SH, Wang CC, Ma ZH. Canopy spectral characterization of wheat stripe rust in latent period. J Spectrosc. 2015;2015:126090. https://doi.org/10.1155/2015/126090.
Liu TX, Zhao CL, Wang XP, Zhao ZJ, Li CH. Inter-cropping with southern corn rust resistance maize genotype improved maize (*Zea mays* L.) defense response. Life Sci J. 2013;10(1):2861–8.
Liu XF, Xu JY, Gu YL, Sun QY, Yuan WY, Ma ZH. Occurrence of *Puccinia polysora* causing southern corn rust in the northeast Huanghuaihai region of China. Plant Dis. 2018;102(4):826. https://doi.org/10.1094/PDIS-05-17-0646-PDN.
Liu YY, Shi J, Wang QL. Southern corn rust occurred in Hebei province in 1998. Plant Prot. 1998;25:53 (in Chinese).
Liu ZX, Wang SC, Dai JR, Huang LJ, Cao HH. Studies of genetic analysis and SSR linked marker location of gene resistance to southern rust in inbred line P25 of maize. Acta Genet Sin. 2003;30(8):706–10.

Lu L, Xu ZN, Sun SL, Du Q, Zhu ZD, Weng JF, et al. Discovery and fine mapping of qSCR6.01, a novel major QTL conferring southern rust resistance in maize. Plant Dis. 2020;104(7):1918–24. https://doi.org/10.1094/PDIS-01-20-0053-RE.
Melching JS. Corn rust: type, races and destructive potential. In: Proc 30th Annual Corn Sor Res Conf. Washington: The American Seed Trade Association. 1975. pp. 90–115.
Meng LV, Deng CE, Li XY, Zhao XD, Li HM, Li ZM, et al. Identification and fine-mapping of *RppCML496*, a major QTL for resistance to *Puccinia polysora* in maize. Plant Genome. 2021;14: e20062. https://doi.org/10.1002/tpg2.20062.
Meng R, Lv ZG, Yan JB, Chen GS, Zhao F, Zeng LL, et al. Development of spectral disease indices for southern corn rust detection and severity classification. Remote Sens. 2020;12:3233. https://doi.org/10.3390/rs12193233.
Moratelli G, Kaefer KAC, Ertel F, Vogt RT, Ferreira SD, Egewarth VA, et al. Effect of fungicide application times in the control management of leaf foliar diseases in maize. Afr J Agric Res. 2015;10:3686–95. https://doi.org/10.5897/AJAR2015.9984.
Nishi K, Kawase A, Namiki F, Hirayae K. Seasonal prevalence of southern rust of corn in Kumamoto Prefecture. Kyushu Pl Prot Res. 1998;44:9–11. https://doi.org/10.4241/kyubyochu.44.9 (in Japanese).
Orian G. Occurrence of *Puccinia polysora* Underwood in the Indian Ocean Area. Nature. 1954;4402:505. https://doi.org/10.1038/173505a0.
Pavgi M. Morphology and taxonomy of the *Puccinia* species on corn and sorghum. Mycopathologia. 1972;47:207–20. https://doi.org/10.1007/BF02051659.
Payak MM (1992) Interception of *Puccina polysora* southern rust of maize in India. New Delhi: National Bureau of Plant Genetic Resources, Indian Agricultural Research Institute
Raabe RD, Connors IL, Martinez AP, Nelson SC. Checklist of plant diseases in Hawaii. Honolulu: University of Hawaii at Manoa; 1981.
Ramirez-Cabral NYZ, Kumar L, Shabani F. Global risk levels for corn rusts (*Puccinia sorghi* and *Puccinia polysora*) under climate change projections. J Phytopathol. 2017;165:563–74. https://doi.org/10.1111/jph.12593.
Reyes GM. An epidemic outbreak of the maize rust in eastern and central Visayas, Philippines. Philipp J Agric. 1953;18:115–28.
Rhind D, Waterston JM, Deighton FC. Occurrence of *Puccinia polysora* Underw in West Africa. Nature. 1952;169:631. https://doi.org/10.1038/169631a0.
Robert AL. Host ranges and races of the corn rusts. Phytopathology. 1962;52:1010–2.
Ryland AK, Storey HH. Physiological Races of *Puccinia polysora* Underw. Nature. 1955;17:655–6. https://doi.org/10.1038/176655b0.
Scheffer RP, Ullstrup AJ. A host-specific toxic metabolite from *Helminthosporium carbonum*. Phytopathology. 1965;55:1037–8.
Scott GE, King SB, Armour JW. Inheritance of resistance to southern corn rust in maize populations. Crop Sci. 1984;24:265–7. https://doi.org/10.2135/cropsci1984.0011183X002400020013x.
Shurtleff MC. Compendium of corn diseases. St Paul: APS Press; 1980.
Spurlock TN, Stark CR, Bailey JD, Tolbert AC, Hoyle RC. Detection, spread and economic impact of southern rust in Arkansas corn fields using remote sensing and spatial analysis technologies. In: Ford V, Kelley J, McKinney N, editors. Corn and grain sorghum research studies. Arkansas: University of Arkansas System; 2020. p. 22–33.
Storey H, Howland AK. Resistance in maize to the Tropical American rust fungus, *Puccinia polysora*. II: Linkage of genes *Rpp1* and *Rpp2*. Heredity. 1959;13:61–5. https://doi.org/10.1038/hdy.1959.4.
Storey H, Howland AK. Resistance in maize to the tropical American rust fungus, *Puccinia polysora* Underw. I. Genes *Rpp1* and *Rpp2*. Heredity. 1957;11:289–301. https://doi.org/10.1038/hdy.1957.26.
Storey HH, Howland AK. Resistance in maize to a third East African race of *Puccinia polysora* Underw. Ann Appl Biol. 1967a;60(2):297–303. https://doi.org/10.1111/j.1744-7348.1967.tb04481.x.
Storey HH, Howland AK. Inheritance of resistance in maize to the virus of streak disease in East Africa. Ann Appl Biol. 1967b;59(3):429–36. https://doi.org/10.1111/j.1744-7348.1967.tb04459.x.
Ullstrup AJ. Inheritance and linkage of a gene determining resistance in maize to an American race of *Puccinia polysora*. Phytopathology. 1965;55:425–8.
Unartngam J, Janruang P, To-anan C. Genetic diversity of *Puccinia polysora* in Thailand based on inter simple sequence repeat (ISSR) markers analysis. Int J Agric Technol. 2011;7(4):1125–37.
Underwood LM. Some new fungi chiefly from Alabama. Bull Torrey Bot Club. 1897;24(2):81–6. https://doi.org/10.2307/2477799.
Wang S, Zhang RY, Shi Z, Zhao YX, Su AG. Identification and fine mapping of *RppM*, a southern corn rust resistance gene in maize. Front Plant Sci. 2020a;11:1057. https://doi.org/10.3389/fpls.2020.01057.
Wang XM, Liu J, Guo YY, Duan CX, Zhu ZD, Sun SL, et al. Multiorigins of initial infection sources of *Puccinia polysora* causing southern rust of maize in China. J Maize Sci. 2020b;28(3):1–14 (in Chinese).
Wang XM, Jin QM, Shi J, Wang ZY, Li X. The status of maize diseases and the possible effect of variety resistance on disease occurrence in the future. Acta Phytopathol Sin. 2006;36(1):1–11 (in Chinese).
Wang YQ, Gao F, Gao GY, Zhao JY, Wang XG, Zhang R. Production and cultivated area variation in cereal, rice, wheat and maize in China (1998–2016). Agronomy. 2019;9(5):222. https://doi.org/10.3390/agronomy9050222.
Wanlayaporn K, Authrapun J, Vanavichit A, Tragoonrung S. QTL mapping for partial resistance to southern corn rust using RILs of tropical sweet corn. Am J Plant Sci. 2013;4(4):878–89. https://doi.org/10.4236/ajps.2013.44108.
Wu XJ, Li N, Zhao PF, He Y, Wang SC. Geographic and genetic identification of *RppS*, a novel locus conferring broad resistance to southern corn rust disease in China. Euphytica. 2015;205:17–23. https://doi.org/10.1007/s10681-015-1376-5.
Xing GZ. Genetic diversity and ultrastructural studies of southern corn rust in China. MA Dissertation. Zhengzhou: Henan Agricultural University; 2011 (in Chinese)
Yang X, Ding XL, He ZL, Ma ZH. Determination of temperature required by southern corn rust. Plant Prot. 2015;41(5):145–7 (in Chinese).
Yao JG, Shan J, Cao B, Cui LG, Du SL, Han ZJ, et al. Mapping the maize southern rust resistance gene of inbred line CML470. J Plant Genet Resour. 2013;14(3):518–22 (in Chinese).
Yuan HX, Xing P, Li CH, Li HL. Resistance comparisons to southern corn rust in different corn varieties. J Maize Sci. 2010;18(2):107–9 (in Chinese).
Zhang XL. Study on the resistance of maize to northern corn leaf blight and southern corn rust. Ph.D. Dissertation, Chinese Academy of Agricultural Sciences, Beijing; 2013 (in Chinese)
Zhang Y, Xu L, Zhang DF, Dai JR, Wang SC. Mapping of southern corn rust-resistant genes in the W2D inbred line of maize (*Zea mays* L.). Mol Breed. 2009;25:433–9. https://doi.org/10.1007/s11032-009-9342-3.
Zhou CJ, Chen CX, Cao PX, Wu SW, Sun JW, Jin DM, et al. Characterization and fine mapping of *RppQ*, a resistance gene to southern corn rust in maize. Mol Genet Genomics. 2007;278:723–8. https://doi.org/10.1007/s00438-007-0288-z.
Zhou GF, Hao DR, Mao YX, Zhu QL, Chen GQ, Lu HH, et al. Identification of genetic loci conferring partial resistance to southern corn rust through a genome-wide association study. Eur J Plant Pathol. 2018;150:1083–90. https://doi.org/10.1007/s10658-017-1351-1.

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