Wheat and Fungal Pathogens

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Photosynthesis is a universal process in the plant kingdom that occurs in various green organs, such as leaves, young stems, green fruits, and ears before maturity, providing a material basis and energy supply for multiple physiological metabolic processes in plants. Plant organs that can perform photosynthesis are considered photosynthetic source organs, which mainly include the leaves of plants, while the storage organs of the organic matter synthesized by photosynthesis represent photosynthetic sink organs, which include mainly stalks, roots, and fruits. At different growth and development stages, the photosynthetic sources and sinks can change accordingly.

Keywords: photosynthesis ; wheat diseases ; immune defense ; ROS ; gene expression

1. Introduction

Photosynthesis is a universal process in the plant kingdom that occurs in various green organs, such as leaves [1], young stems [2], green fruits [3], and ears before maturity [4], providing a material basis and energy supply for multiple physiological metabolic processes in plants [5]. Plant organs that can perform photosynthesis are considered photosynthetic source organs, which mainly include the leaves of plants, while the storage organs of the organic matter synthesized by photosynthesis represent photosynthetic sink organs, which include mainly stalks, roots, and fruits [6]. At different growth and development stages, the photosynthetic sources and sinks can change accordingly. For instance, when plants are in the seedling stage, leaves and stems act as both sources and sinks of photosynthesis because photosynthates are supplied for their own growth and development. Photosynthesis is disrupted by a variety of complex factors, including abiotic stresses caused by water [7], temperature [8], light [9], and mechanical damage [10] and biological stresses caused by insects and pathogens [11][12]. In contrast, photosynthetic changes during this process may be related to responses to these factors.

2. Wheat and Its Fungal Pathogens Constitute Ideal Pathogen Systems for Studying the Role of Photosynthesis in the Process of Resistance Development

Wheat is one of the most important crop species and has been a staple food of humans for thousands of years worldwide [13]. The growth period of wheat is longer than that of other food crop species such as maize and rice, making wheat more susceptible to various pathogens and diseases during its growth and development [14]. Among these diseases, the wheat powdery mildew caused by the obligate biotrophic fungus Blumeria graminis f. sp. tritici (Bgt), stripe rust caused by the biotrophic parasite Puccinia striiformis f. sp. tritici (Pst), and Fusarium head blight (FHB), which is caused mainly by Fusarium graminearum species, are the three most severe diseases affecting both yield and quality [15][16]. The basic characteristics of the three diseases are listed in **Table 1** . Wheat powdery mildew is an epidemic disease worldwide, especially in regions such as southwestern China, whose environment is temperate and rainy during the wheat growing season [17]. Furthermore, powdery mildew can occur throughout the whole growth period of wheat, and the optimal temperature for its occurrence is 15–20 °C [18]. For the infected position, Bgt pathogens infect mainly the leaves and sometimes also infect green awns, glumes, and stalks [19]. These organs differ largely in the position of the plant, but they are usually considered photosynthetic source organs at the developmental stage because of their strong photosynthetic competence [20]. Therefore, wheat powdery mildew is a typical source disease from the perspective of photosynthetic function. Wheat stripe rust is also one of the most threatening diseases in wheat production worldwide [21], especially in temperate climates. Stripe rust can occur during the whole growth period from seedling emergence to maturity, and the optimum temperature for its occurrence is 13–16 °C [22][23]. The tissue specificity of stripe rust is stronger than that of wheat powdery mildew, which mainly parasitizes leaves, the main source of wheat photosynthesis, and seldom occurs in wheat stems and glumes [24]. Therefore, wheat stripe rust is also a typical source disease from the perspective of source–sink relationships.

Table 1. Characteristics of the three major fungal diseases of wheat.

Major Diseases in Wheat	Pathogen	Position of Infection	Infection Period	Optimal Temperature	Classification of Diseases
Powdery mildew	*Blumeria graminis* f. sp. *tritici* (*Bgt*)	leaf	whole growth period	15–20 °C [32]	source disease
Stripe rust	*Puccinia striiformis* f. sp. *tritici* (*Pst*)	leaf	whole growth period	13–16 °C [36,37]	source disease
Fusarium head blight	*Fusarium graminearum* species complex	ear	adult stage	20–25 °C [39]	sink disease

It is well known that the occurrence of FHB in wheat is extremely harmful to the quality of wheat because the causal agent produces the mycotoxin deoxynivalenol (DON), which is poisonous to both humans and animals [25]. At the same time, FHB can also cause great loss of wheat yield, especially under the environment conditions suitable for F. graminearum growth and breeding [26][27]. F. graminearum infects mainly the ears of wheat at the adult stage, and they are generally considered sink organs due to their role as a storage location for photosynthates in wheat. Therefore, in contrast to wheat powdery mildew and stripe rust, FHB is a typical sink disease. Measurements of photosynthesis parameters of FHB-resistant/susceptible sister-line wheat after inoculation with F. graminearum showed that the photosynthesis rate of susceptible wheat leaves did not change significantly, while the photosynthesis rate of resistant wheat leaves decreased significantly in the early stage after pathogen infection, but the resistant genotype produced a larger yield than the susceptible genotypes did [28]. This indicates that the decrease in photosynthesis rate could play a key role in inducing systemic resistance to maintain the ultimate yield.

The mechanism through which both stripe rust and powdery mildew cause yield losses could be different from that of FHB because of the difference in the infection positions. A variety of wheat leaf diseases can reduce the amount of green leaf area, resulting in a decrease in chlorophyll contents in the infected parts [29], which directly reduces the photosynthesis capacity and both the synthesis and the accumulation of organic matter in photosynthetic source organs, leading to a decline in wheat yield [30]. For instance, the chloroplast envelope of wheat mesophyll cells is disrupted after infection with Bgt , and the thylakoid becomes enlarged [30]. The effector protein of wheat stripe rust fungus can inhibit chloroplast function [31], and Pst infection can also reduce the chlorophyll content in wheat leaves [32]. However, there may be several reasons for wheat yield loss caused by FHB. Studies have indicated that damaged photosynthetic sink organs may inhibit photosynthesis at the source via feedback mechanisms, which could be the important reason to explain the yield loss caused by FHB [28][33].

In addition, a few reports have indicated that photosynthesis may be involved in wheat immune defense responses to fungal pathogens and may influence resistance formation because changes in photosynthesis may provide an important signal for maintaining source—sink balance during interactions between wheat and pathogens [12][34]. However, there are few relevant research reports on the role of photosynthesis in influencing the formation of wheat resistance, and those studies have focused mainly on photosynthetic changes after inoculation with pathogens, so knowledge about the influence of photosynthesis is very fragmented or not systematic [35]. Both wheat powdery mildew and stripe rust are source organ diseases, and FHB is a sink organ disease. Hence, wheat and its fungal pathogens constitute an ideal pathogen system for exploring the role of photosynthesis in the development of wheat resistance.

3. Relationships between Yield and Photosynthetic Changes Caused by Pathogen Infection in Wheat

Decreases in wheat yield caused by pathogen infection have always been a focus of wheat breeders and physiologists [36]. A large number of studies have reported adverse effects of wheat leaf diseases (such as powdery mildew and stripe rust) and ear diseases (such as FHB) on wheat yield [37][38]. Infection of wheat powdery mildew at the seedling stage can affect the growth and development of wheat plants [39] and can further lead to a decrease in grain filling and grain weight at the adult stage [19]. Wheat stripe rust infection not only can reduce the number of tillers at the tillering stage [40] but also can significantly reduce the grain number per spike and 1000-grain weight at the adult stage [41]. Wheat stripe rust and powdery mildew have very similar effects on wheat yield because they both directly act on the source organs of wheat photosynthesis and cause wheat yield losses through long-term inhibition of photosynthesis in wheat leaves [38][42][43]. In addition, both Bgt and Pst are parasitic fungi that rely on host metabolism to provide carbohydrates, amino acids, and inorganic nutrients [44][45]. The growth and reproduction of a large number of pathogens directly consume nutrients in photosynthetic source organs, further reducing wheat yields [44].

The production of the mycotoxin DON in diseased wheat ears is the main negative effect caused by FHB but could also lead to severe yield losses due to a damaged photosynthetic sink and a disrupted source–sink balance [25][28]. It has been reported that FHB can cause 10–70% yield losses in years in which epidemics occur [30]. Unfortunately, how the damage of photosynthetic sink organs causes the changes of photosynthetic source organs remains to be further explored. It has been reported that the activity of photosynthesis-related enzymes and the expression of associated gene transcripts are modified by sink demand [46]. Stomatal closure is a plant's first line of defense against pathogens [47]. Recent studies have suggested that the reduced photosynthetic efficiency of susceptible wheat leaves is regulated by stomatal factors after the occurrence of FHB symptoms, while the Gs of resistant wheat leaves did not show a significant decrease under the same treatment [28][36]. These reports suggest that when pathogens attack the photosynthetic sinks of wheat, there is some kind of feedback regulatory mechanism that alters the balance between photosynthetic sources and sinks by adjusting photosynthesis parameters.

Infection of wheat pathogenic fungi in the leaves and ears can cause a decrease in photosynthesis in wheat leaves. The decline in photosynthesis in the early stage of various fungal pathogens invasion in wheat are listed in **Table 2** . In incompatible reactions, the decrease in photosynthesis capacity of the host may be due to the material and energy needed for defense [48], while in compatible reactions, the decrease in photosynthesis capacity may be due to the damage caused by pathogen infection of the host [49][50]. Unlike the transcriptomic and photosynthetic changes in sister wheat lines after inoculation with Bgt. , it was found that the inhibition of photosynthesis in resistant wheat paralleled the global downregulation of photosynthesis-related genes to actively regulate the immune response, but the decrease in photosynthesis in susceptible wheat lines is caused by stomatal closure and did not regulate the immune response [51]. The infection of wheat stripe rust also causes a phenomenon similar to that caused by powdery mildew. The Pn of the resistant cultivar CN19 carrying the gene Yr41 [52] and the susceptible cultivar Sy95-71 decreased significantly at 72 h after inoculation with Pst compared with no inoculation [32]. By exploring the resistance mechanism of wheat resistance genes, it was also found that, by inhibiting photosynthesis, the stripe rust resistance gene Yr36 could provide broad-spectrum resistance to Pst races in wheat at the adult stage [53]. Studies on the signals of photosynthetic changes caused by wheat leaf diseases have also suggested that the resistant genotypes of wheat could actively regulate photosynthetic changes to mediate specific immune defenses against the invasion of pathogens, albeit at a cost of greatly reduced photosynthesis capacity in the initial stage of pathogen infection [54]. Taken together, these results indicate that the changes in photosynthesis parameters in the early stage of stripe rust and powdery mildew infection in wheat were related to the development of resistance.

Table 2. Changes in photosynthesis and antioxidant enzyme activity in response to the three main fungal pathogens in wheat at the early stage of infection (within 72 h).

Major Diseases in Wheat	Parameter (Leaf)	Wheat Cultivar/Line	Resistance	0 h	12 h	24 h	48 h	72 h
Powdery mildew	**Pn [25]**	**L658**	**R**	**CG**	↘	↗	→	↘
		L958	**S**	**CG**	↘	↗	→	↘
	SOD [25]	**L658**	**R**	**CG**	→	→	→	↘
		L958	**S**	**CG**	↗	→	↘	↘
	CAT [25]	**L658**	**R**	**CG**	↘	↗	↘	↗
		L958	**S**	**CG**	→	↗	↘	↘
Stripe rust	**Pn [24]**	**CN19**	**R**	**CG**	–	–	–	↘
		Sy95-71	**S**	**CG**	–	–	–	↘
	Pn [73]	***psbo-A1* Mutant**	**R**	–	–	–	↘	–
		Kronos	**S**	–	–	–	**CG**	–
Fusarium head blight	**Pn [74]**	**L693**	**R**	**CG**	–	↘	→	→
		L661	**S**	**CG**	–	→	→	→
	SOD [74]	**L693**	**R**	**CG**	–	↘	→	→
		L661	**S**	**CG**	–	↗	→	→
	CAT [74]	**L693**	**R**	**CG**	–	↗	→	→
		L661	**S**	**CG**	–	↘	↘	↘

CP: control group; ↘: indicates that the current time has decreased significantly from the previous point in time; ↗: indicates that current time has increased significantly from the previous point in time; →: indicates that current time had no significant change from the previous point in time; ─: indicates that no available data were detected.

Interestingly, photosynthesis-related parameters in the ears and leaves of resistant and susceptible wheat were found to be significantly different after inoculation with F. graminearum [55]. The photosynthesis of wheat spikes increased at the early stage of inoculation in both resistant and susceptible plants, although the spikes were directly infected by F. graminearum ; however, there was a larger increase in the ears and a larger decrease in the leaves in the resistant plants than in the susceptible plants [55]. Therefore, changes in the photosynthesis parameters of different genotypes of wheat may provide important insight into the mediation of immune defense responses to wheat sink diseases.

4. ROS Produced by Photosynthesis Relay Compatibility or Incompatibility Signals

Photosynthesis can regulate the immune defense response in many ways, the most important of which is by inducing the production of ROS and regulating changes in hormones [12][34][48]. ROS, including singlet oxygen (1O 2), superoxide anion radicals (O 2•−), hydrogen peroxide (H 2O 2), and hydroxyl radicals (•OH) are produced mainly during interactions between metabolic intermediates and oxygen during photosynthesis [56][57]. The concentration of ROS in plants is maintained usually at low levels due to rapid and precise regulation by various antioxidant enzymes, mainly catalase (CAT), superoxide dismutase (SOD), ascorbate peroxidase (APX), and peroxidase (POD) [57]. The metabolism of ROS has a dual role in plant growth and development. On the one hand, high concentration of ROS accumulation in plant cells can ultimately damage proteins, lipids, and nucleic acids and can even disrupt photosynthesis, and ROS are toxic to many cellular processes in plants [58][59][60]. On the other hand, a role for ROS in the immune defense has gradually been elucidated, indicating that ROS play a crucial role in plant immune defense [61]. The production of ROS is one of the earliest cellular reactions following pathogen recognition [62]. Many studies have revealed that low concentrations of ROS can activate the expression of defense-related genes and induce various defense responses [63], while a high accumulation of ROS can be used as a defense weapon to resist pathogen invasion [64]. In addition, ROS can regulate immune defense through interactions with plant hormones [65][66], and the synergistic effect of ROS and salicylic acid (SA) plays an important role in mediating the hypersensitive response (HR) [67][68]. In interactions between plants and pathogens, the ROS that accumulate are generated mainly by photosystem I (PSI) and PSII during photosynthesis [69]. Therefore, the changes in ROS caused by photosynthetic changes could be an important factor explaining why early photosynthesis is involved in the development of plant disease resistance, which ultimately results in compatibility or incompatibility between pathogens and hosts.

A large number of studies have suggested that the accumulation of ROS, particularly H 2O 2, constitutes an important signal that is transmitted and may determine the compatibility between pathogenic fungi and wheat in the early stage of infection [50][70][71]. For instance, transgenic experiments confirmed that, after being transferred to a resistance gene, the powdery mildew resistance of susceptible wheat variety Yangmai 158 was significantly improved at the seedling and mature stages and accumulated more ROS at the Bgt infection position [72]. The results indicate that ROS could improve the resistance of wheat to powdery mildew. By measuring the changes in ROS and photosynthesis in resistant/susceptible sister wheat lines after inoculation with Bgt , Hu et al. [51] reported that two stages of H 2O 2 bursts occurred during the incompatible reaction process and that a single low-amplitude and transient H 2O 2 outbreak in susceptible wheat lines was not sufficient to induce the HR. In addition, ROS signaling plays a crucial role in the immune response to stripe rust caused by the parasitic fungus Pst . Overexpression of LSD-1-like zinc-finger protein (TaLOL) , which actively regulates the ROS signaling pathway, can enhance resistance to stripe rust by inducing the production and accumulation of ROS and cell death, while silencing TaLOL2 increases sensitivity to avirulent races of Pst and reduces ROS production in wheat [70]. These results indicate that the accumulation of ROS acts as an important signal of immune defense in wheat leaf diseases caused by parasitic fungi.

FHB resistance is an extremely complex trait that is controlled by multiple quantitative trait loci (QTLs) and includes the additive effect of several genes [73]. Wheat resistance to FHB consists of two main types: resistance to the initial infection (type I resistance) and resistance to the spread of the disease within the wheat head (type II resistance) [74]. F. graminearum , the causal agent of wheat FHB, is a hemibiotroph that can absorb nutrients from dead tissues, so the HR mediated by the accumulation of ROS may not easily effectively resist FHB [75]. The results of some studies on the changes in ROS levels during FHB infection are consistent with our views. For instance, comparative transcriptome analysis suggested that ROS may accumulate in susceptible mutants of the wheat cultivar Wangshuibai after inoculation with F. graminearum but not in FHB-resistant cultivars of Wangshuibai [76]. Another example is the FHB-resistant line L693, which exhibits temporary infection symptoms due to insufficient accumulation of ROS after inoculation with F.

graminearum , after which systemic acquired resistance (SAR) is induced in distal tissues via the SA pathway to resist pathogen invasion [55]. These results indicate that type II resistance to spreading within the wheat head mediated by the jasmonic acid (JA) or SA pathway may determine the compatibility between pathogens and wheat instead of mediating a strong immune defense response based on ROS at the site of parasitic fungal infection [55][76][77]. Therefore, it is difficult to determine the compatibility of F. graminearum with wheat by outbreak of ROS in the infected position, but ROS produced in a pathogen-infected position may play a role in signaling pathways because ROS are closely related to the SA pathway.

References

1. Smith, W.K.; Vogelmann, T.C.; Delucia, E.H.; Bell, D.T.; Shepherd, K.A. Leaf form and photosynthesis. BioScience 199 7, 47, 785–793.
2. Nilsen, E.T. Stem photosynthesis: Extent, patterns, and role in plant carbon economy. In Plant Stems; Gartner, B.L., E d.; Academic Press: San Diego, CA, USA, 1995; pp. 223–240.
3. Cipollini, M.L.; Levey, D.J. Why some fruits are green when they are ripe: Carbon balance in fleshy fruits. Oecologia 19 91, 88, 371–377.
4. Kriedemann, P. The photosynthetic activity of the wheat ear. Ann. Bot. 1966, 30, 349–363.
5. Göhre, V.; Jones, A.M.; Sklenář, J.; Robatzek, S.; Weber, A.P. Molecular crosstalk between PAMP-triggered immunity a nd photosynthesis. Mol. Plant Microbe Interact. 2012, 25, 1083–1092.
6. Paul, M.J.; Foyer, C.H. Sink regulation of photosynthesis. J. Exp. Bot. 2001, 52, 1383–1400.
7. Chaves, M.; Flexas, J.; Pinheiro, C. Photosynthesis under drought and salt stress: Regulation mechanisms from whole plant to cell. Ann. Bot. 2008, 103, 551–560.
8. Hou, W.; Sun, A.H.; Chen, H.L.; Yang, F.S.; Pan, J.L.; Guan, M.Y. Effects of chilling and high temperatures on photosyn thesis and chlorophyll fluorescence in leaves of watermelon seedlings. Biol. Plant. 2016, 60, 148–154.
9. Takahashi, S.; Badger, M.R. Photoprotection in plants: A new light on photosystem II damage. Trends Plant Sci. 2011, 16, 53–60.
10. Quilliam, R.S.; Swarbrick, P.J.; Scholes, J.D.; Rolfe, S.A. Imaging photosynthesis in wounded leaves of Arabidopsis tha liana. J. Exp. Bot. 2005, 57, 55–69.
11. Meyer, G.A.; Whitlow, T.H. Effects of leaf and sap feeding insects on photosynthetic rates of goldenrod. Oecologia 199 2, 92, 480–489.
12. Lu, Y.; Yao, J. Chloroplasts at the crossroad of photosynthesis, pathogen infection and plant defense. Int. J. Mol. Sci. 2 018, 19, 3900.
13. Kumar, R.R.; Rai, R.D. Can wheat beat the heat: Understanding the mechanism of thermotolerance in wheat (Triticum aestivum L.). Cereal Res. Commun. 2014, 42, 1–18.
14. Duveiller, E.; Singh, R.P.; Nicol, J.M. The challenges of maintaining wheat productivity: Pests, diseases, and potential e pidemics. Euphytica 2007, 157, 417–430.
15. He, Z.; Lan, C.; Chen, X.; Zou, Y.; Zhuang, Q.; Xia, X. Progress and perspective in research of adult-plant resistance to stripe rust and powdery mildew in wheat. Sci. Agric. Sin. 2011, 44, 2193–2215.
16. Bai, G.; Shaner, G. Management and resistance in wheat and barley to fusarium head blight. Annu. Rev. Phytopathol. 2 004, 42, 135–161.
17. Conner, R.; Kuzyk, A.D.; Su, H. Impact of powdery mildew on the yield of soft white spring wheat cultivars. Can. J. Plan t Sci. 2003, 83, 725–728.
18. Yang, M.J.; Huang, K.Y.; Han, Q.D. Research progresses on wheat powdery mildew and its resistance. Mol. Plant Bree d. 2016, 14, 1244–1254.
19. Gao, H.; Niu, J.; Li, S. Impacts of wheat powdery mildew on grain yield & quality and its prevention and control method s. Am. J. Agric. 2018, 6, 141–147.
20. Wang, Z.-M.; Wei, A.-L.; Zheng, D.-M. Photosynthetic characteristics of non-leaf organs of winter wheat cultivars differi ng in ear type and their relationship with grain mass per ear. Photosynthetica 2001, 39, 239–244.
21. Schwessinger, B. Fundamental wheat stripe rust research in the 21st century. New Phytol. 2017, 213, 1625–1631.
22. Doodson, J.K.; Manners, J.G.; Myers, A. Some effects of yellow rust (Puccinia striiformis) on the growth and yield of a s pring wheat. Ann. Bot. 1964, 28, 459–472.

23. Line, R.F. Stripe rust of wheat and barley in North America: A retrospective historical review. Annu. Rev. Phytopathol. 2002, 40, 75–118.

24. Chen, W.; Wellings, C.; Chen, X.; Kang, Z.; Liu, T. Wheat stripe (yellow) rust caused by Puccinia striiformis f. sp. tritici. Mol. Plant Pathol. 2014, 15, 433–446.

25. Parry, D.W.; Jenkinson, P.; Mcleod, L. Fusarium ear blight (SCAB) in small-grain cereals—A review. Plant Pathol. 2007, 44, 207–238.

26. Cowger, C.; Sutton, A.L. The southeastern US Fusarium head blight epidemic of 2003. Plant Health Progress. 2005, 6, 4.

27. Wegulo, S.N.; Baenziger, P.S.; Nopsa, J.H.; Bockus, W.W.; Hallen-Adams, H. Management of Fusarium head blight of wheat and barley. Crop Prot. 2015, 73, 100–107.

28. Yang, S.; Li, X.; Chen, W.; Liu, T.; Zhong, S.; Ma, L.; Zhang, M.; Zhang, H.; Yu, D.; Luo, P. Wheat resistance to fusariu m head blight is associated with changes in photosynthetic parameters. Plant Dis. 2016, 100, 847–852.

29. Rosyara, U.R.; Subedi, S.; Duveiller, E.; Sharma, R.C. The effect of spot blotch and heat stress on variation of canopy t emperature depression, chlorophyll fluorescence and chlorophyll content of hexaploid wheat genotypes. Euphytica 201 0, 174, 377–390.

30. Aboukhaddour, R.; Fetch, T.; Mccallum, B.D.; Harding, M.W.; Beres, B.L.; Graf, R.J. Wheat diseases on the prairies: A Canadian story. Plant Pathol. 2020, 69, 418–432.

31. Xu, Q.; Tang, C.; Wang, X.; Sun, S.; Zhao, J.; Kang, Z.; Wang, X. An effector protein of the wheat stripe rust fungus targ ets chloroplasts and suppresses chloroplast function. Nat. Commun. 2019, 10, 5571.

32. Chen, Y.E.; Cui, J.M.; Su, Y.Q.; Yuan, S.; Yuan, M.; Zhang, H.Y. Influence of stripe rust infection on the photosynthetic c haracteristics and antioxidant system of susceptible and resistant wheat cultivars at the adult plant stage. Front. Plant S ci. 2015, 6, 779.

33. Spanic, V.; Viljevac Vuletic, M.; Drezner, G.; Zdunic, Z.; Horvat, D. Performance indices in wheat chlorophyll a fluoresce nce and protein quality influenced by FHB. Pathogens 2017, 6, 59.

34. Kangasjärvi, S.; Tikkanen, M.; Durian, G.; Aro, E.M. Photosynthetic light reactions—An adjustable hub in basic producti on and plant immunity signaling. Plant Physiol. Biochem. 2014, 81, 128–134.

35. Francesconi, S.; Balestra, G.M. The modulation of stomatal conductance and photosynthetic parameters is involved in Fusarium head blight resistance in wheat. PLoS ONE 2020, 15, e0235482.

36. Walters, D.R.; Bingham, I.J. Influence of nutrition on disease development caused by fungal pathogens: Implications fo r plant disease control. Ann. Appl. Biol. 2007, 151, 307–324.

37. Serrago, R.A.; Carretero, R.; Bancal, M.O.; Miralles, D.J. Grain weight response to foliar diseases control in wheat (Triti cum aestivum L.). Field Crop. Res. 2011, 120, 352–359.

38. Singh, R.P.; Singh, P.K.; Rutkoski, J.; Hodson, D.P.; He, X.; Jørgensen, L.N.; Hovmøller, M.S.; Huerta-Espino, J. Diseas e impact on wheat yield potential and prospects of genetic control. Annu. Rev. Phytopathol. 2016, 54, 303–322.

39. Niu, J.; Wang, H.; Hong, D. Advance in studies on molecular basis for wheat powdery mildew resistance. J. Henan Agri c. Univ. 2006, 40, 678–682.

40. Hendrix, J.W.; Fuchs, E. Influence of fall stripe rust infection on tillering and yield of Wheat. Plant Dis. Rep. 1970, 54, 3 47–349.

41. Yang, F.P.; Yang, W.X.; Shang, X.W. A study on the effect of wheat stripe rust on wheat yield. J. Gansu Agric. Univ. 200 8, 2, 51–55.

42. Wright, D.P.; Baldwin, B.C.; Shephard, M.C.; Scholes, J.D. Source-sink relationships in wheat leaves infected with pow dery mildew. I. Alterations in carbohydrate metabolism. Physiol. Mol. Plant Pathol. 1995, 47, 237–253.

43. Li, X.; Liu, T.-G.; Chen, W.; Zhong, S.; Zhang, H.; Tang, Z.; Chang, Z.; Wang, L.; Zhang, M.; Li, L.; et al. Wheat WCBP1 encodes a putative copper-binding protein involved in stripe rust resistance and inhibition of leaf senescence. BMC Pla nt Biol. 2015, 15, 239.

44. Sutton, P.N.; Gilbert, M.J.; Williams, L.E.; Hall, J.L. Powdery mildew infection of wheat leaves changes host solute trans port and invertase activity. Physiol. Plant. 2007, 129, 787–795.

45. Chen, X.M. Epidemiology and control of stripe rust [Puccinia striiformis f. sp. tritici] on wheat. Can. J. Plant Pathol. 200 5, 27, 314–337.

46. Mccormick, A.J.; Cramer, M.D.; Watt, D.A. Changes in photosynthetic rates and gene expression of leaves during a so urce-sink perturbation in sugarcane. Ann. Bot. 2008, 101, 89–102.

47. Bharath, P.; Gahir, S.; Raghavendra, A.S. Abscisic acid-induced stomatal closure: An important component of plant defense against abiotic and biotic stress. Front Plant Sci. 2021, 12, 324.

48. Kangasjärvi, S.; Neukermans, J.; Li, S.; Aro, E.M.; Noctor, G. Photosynthesis, photorespiration, and light signalling in defence responses. J. Exp. Bot. 2012, 63, 1619–1636.

49. Nogueira, A.F., Jr.; Ribeiro, R.V.; Appezzato-Da-Glória, B.; Soares, M.K.M.; Rasera, J.B.; Amorim, L. Phakopsora euvitis causes unusual damage to leaves and modifies carbohydrate metabolism in grapevine. Front. Plant Sci. 2017, 8, 1675.

50. Ma, L.X.; Zhong, S.F.; Liu, N.; Chen, W.Q.; Liu, T.G.; Li, X.; Zhang, M.; Ren, Z.L.; Yang, J.Z.; Luo, P.G. Gene expression profile and physiological and biochemical characterization of hexaploid wheat inoculated with Blumeria graminis f. sp. tritici. Physiol. Mol. Plant Pathol. 2015, 90, 39–48.

51. Hu, Y.; Zhong, S.; Zhang, M.; Liang, Y.; Gong, G.; Chang, X.; Tan, F.; Yang, H.; Qiu, X.; Luo, L.; et al. Potential role of photosynthesis in the regulation of reactive oxygen species and defence responses to Blumeria graminis f. sp. tritici in wheat. Int. J. Mol. Sci. 2020, 21, 5767.

52. Luo, P.G.; Hu, X.Y.; Ren, Z.L.; Zhang, H.Y.; Shu, K.; Yang, Z.J. Allelic analysis of stripe rust resistance genes on wheat chromosome 2BS. Genome 2008, 51, 922–927.

53. Wang, S.; Li, Q.-P.; Wang, J.; Yan, Y.; Zhang, G.-L.; Yan, Y.; Zhang, H.; Wu, J.; Chen, F.; Wang, X.; et al. YR36/WKS1-mediated phosphorylation of PsbO, an extrinsic member of photosystem ii, inhibits photosynthesis and confers stripe rust resistance in wheat. Mol. Plant 2019, 12, 1639–1650.

54. Bolton, M.D. Primary metabolism and plant defense—Fuel for the fire. Mol. Plant Microbe Interact. 2009, 22, 487–497.

55. Li, X.; Zhong, S.; Chen, W.; Fatima, S.A.; Huang, Q.; Li, Q.; Tan, F.; Luo, P. Transcriptome analysis identifies a 140 kb region of chromosome 3B containing genes specific to fusarium head blight resistance in wheat. Int. J. Mol. Sci. 2018, 19, 852.

56. Rychter, A.M. Antioxidants and reactive oxygen species in plants. Ann. Bot. 2006, 98, 1114.

57. Sharma, P.; Jha, A.B.; Dubey, R.S.; Pessarakli, M. Reactive oxygen species, oxidative damage, and antioxidative defense mechanism in plants under stressful conditions. J. Bot. 2012, 2012, 217037.

58. Apel, K.; Hirt, H. Reactive oxygen species: Metabolism, oxidative stress, and signal transduction. Annu. Rev. Plant Biol. 2004, 55, 373–399.

59. Torres, M.A. ROS in biotic interactions. Physiol. Plant. 2010, 138, 414–429.

60. Li, C.-Y.; Zhang, N.; Guan, B.; Zhou, Z.-Q.; Mei, F.-Z. Reactive oxygen species are involved in cell death in wheat roots against powdery mildew. J. Integr. Agric. 2019, 18, 1961–1970.

61. Tsuda, K.; Katagiri, F. Comparing signaling mechanisms engaged in pattern-triggered and effector-triggered immunity. Curr. Opin. Plant Biol. 2010, 13, 459–465.

62. Torres, M.A.; Jones, J.D.; Dangl, J.L. Reactive oxygen species signaling in response to pathogens. Plant Physiol. 2006, 141, 373–378.

63. Qi, J.; Song, C.P.; Wang, B.; Zhou, J.; Kangasjärvi, J.; Zhu, J.K.; Gong, Z. Reactive oxygen species signaling and stomatal movement in plant responses to drought stress and pathogen attack. J. Integr. Plant Biol. 2018, 60, 805–826.

64. Bowling, S.A.; Guo, A.; Cao, H.; Gordon, A.S.; Klessig, D.F.; Dong, X. A mutation in Arabidopsis that leads to constitutive expression of systemic acquired resistance. Plant Cell 1994, 6, 1845–1857.

65. Shetty, N.P.; Jørgensen, H.J.L.; Jensen, J.D.; Collinge, D.B.; Shetty, H.S. Roles of reactive oxygen species in interactions between plants and pathogens. Eur. J. Plant Pathol. 2008, 121, 267–280.

66. Wasternack, C. Jasmonates: An update on biosynthesis, signal transduction and action in plant stress response, growth and development. Ann. Bot. 2007, 100, 681–697.

67. Overmyer, K.; Brosché, M.; Kangasjärvi, J. Reactive oxygen species and hormonal control of cell death. Trends Plant Sci. 2003, 8, 335–342.

68. Shah, J. The salicylic acid loop in plant defense. Curr. Opin. Plant Biol. 2003, 6, 365–371.

69. Asada, K. Production and scavenging of reactive oxygen species in chloroplasts and their functions. Plant Physiol. 2006, 141, 391–396.

70. Qi, T.; Guo, J.; Liu, P.; He, F.; Wan, C.; Islam, M.A.; Tyler, B.M.; Kang, Z.; Guo, J. Stripe rust effector PstGSRE1 disrupts nuclear localization of ROS-promoting transcription factor TaLOL2 to defeat ROS-induced defense in wheat. Mol. Plant 2019, 12, 1624–1638.

71. Zurbriggen, M.D.; Carrillo, N.; Hajirezaei, M.R. ROS signaling in the hypersensitive response: When, where and what for? Plant Signal. Behav. 2010, 5, 393–396.
72. Wang, Z.; Cheng, J.; Fan, A.; Zhao, J.; Yu, Z.; Li, Y.; Zhang, H.; Xiao, J.; Muhammad, F.; Wang, H.; et al. LecRK-V, an L-type lectin receptor kinase in Haynaldia villosa, plays positive role in resistance to wheat powdery mildew. Plant Biotechnol. J. 2018, 16, 50–62.
73. Kumar, A.; Karre, S.; Dhokane, D.; Kage, U.; Hukkeri, S.; Kushalappa, A.C. Real-time quantitative PCR based method for the quantification of fungal biomass to discriminate quantitative resistance in barley and wheat genotypes to fusarium head blight. J. Cereal Sci. 2015, 64, 16–22.
74. Mesterhazy, A. Types and components of resistance to Fusarium head blight of wheat. Plant Breed. 1995, 114, 377–386.
75. Ding, L.; Xu, H.; Yi, H.; Yang, L.; Kong, Z.; Zhang, L.; Xue, S.; Jia, H.; Ma, Z. Resistance to hemi-biotrophic F. graminearum infection is associated with coordinated and ordered expression of diverse defense signaling pathways. PLoS ONE 2011, 6, e19008.
76. Xiao, J.; Jin, X.; Jia, X.; Wang, H.; Cao, A.; Zhao, W.; Pei, H.; Xue, Z.; He, L.; Chen, Q.; et al. Transcriptome-based discovery of pathways and genes related to resistance against Fusarium head blight in wheat landrace Wangshuibai. BMC Genom. 2013, 14, 197.
77. Walter, S.; Nicholson, P.; Doohan, F.M. Action and reaction of host and pathogen during Fusarium head blight disease. New Phytol. 2010, 185, 54–66.