Plant Immune Response

Subjects: Plant Sciences

Contributor: María Fiamma Grossi Vanacore , Melina Sartori , Francisco Giordanino , Germán Barros , Andrea Nesci , Daiana García

In a world with constant population growth, and in the context of climate change, the need to supply the demand of safe crops has stimulated an interest in ecological products that can increase agricultural productivity. This implies the use of beneficial organisms and natural products to improve crop performance and control pests and diseases, replacing chemical compounds that can affect the environment and human health. Microbial biological control agents (MBCAs) interact with pathogens directly or by inducing a physiological state of resistance in the plant. This involves several mechanisms, like interference with phytohormone pathways and priming defensive compounds. In Argentina, one of the world's main maize exporters, yield is restricted by several limitations, including foliar diseases such as common rust and northern corn leaf blight (NCLB).

maize phyllosphere

1. Introduction

Maize (*Zea mays* L.) is one of the most important crops in the world and is used for human and animal consumption, as well as being a source of biofuel. This cereal is composed mainly of starch, but also supplies proteins and fatty acids and vitamins and minerals of great nutritional value [1]. Furthermore, it provides high levels of phenolic acids, flavonoids and carotenoids with antioxidant properties [2]. Argentina is one of the world's largest exporters of maize. In 2021/2022, this country produced over 52 Mtn of maize [3].

One of the main factors contributing to reduced crop productivity is the occurrence of diseases, combined with an improper management [4]. Several maize diseases, particularly foliar ones, have a negative impact on photoassimilates production, which results in lower grain yield. In most cases, the extent of the disease depends on the environmental conditions, the pathogens involved, the host's susceptibility and human intervention. In particular, the main factors that benefit fungal disease development are changes in sowing dates, reduced tillage, irrigation, intense and frequent precipitations during the summer months, poor monitoring and the presence of volunteer maize from previous harvests [5][6][7][8].

One of the most important foliar fungal diseases in maize is northern corn leaf blight (NCLB), caused by *Exserohilum turcicum*, a teleomorph of *Setosphaeria turcica* (Leonard and Suggs). In years with serious occurrence of NCLB, yield of the susceptible hybrids has been documented to decrease by about 40–50% [9]. In the field, mycelia and conidia of *E. turcicum* overwinter in crop residues and can be transported for long distances [10]. This pathogen is a hemibiotroph microorganism that spreads biotrophically at early stages of infection before

shifting to a necrotrophic lifestyle [11][12]. Symptoms appear as grey elliptical lesions beginning on the lower leaves of the plant. As the disease progresses, susceptible plants become covered with the necrotic lesions that converge, giving it the appearance of being “burnt”. NCLB reduces maize yield by destroying the photosynthetically active area. Yield is also affected indirectly during the harvest because of stem breakage and rot, since the decrease in photosynthetically active area causes remobilization of carbohydrates from the stems to provide for the cob [10][13][14].

Cultural management of NCLB includes the application of fungicides, the selection of hybrids with genetic resistance, crop rotation and changes in the sowing date. The latter is often avoided in years when drought is forecasted, as an early date increases the risk of drought matching critical periods of the crop in the central area of Argentina [8]. The prevalent method for disease management in maize and other crops is the use of chemical fungicides. The fungicides used to control NCLB are mixtures of strobilurins and triazoles e.g., pyraclostrobin + epoxiconazole; azoxystrobin + cyproconazole; picoxystrobin + cyproconazole, among others [15]. A proper application should be performed at the initial stages of the disease in such a way that the critical stages of the crop are protected [8][16]. However, these chemicals are moderately hazardous, Class II and, to be effective, must constantly protect new plant leaves, increasing costs [17]. In addition, a chemical control practice may cause environmental problems [18] and health problems [19]. In addition, the economic damage thresholds for NCLB are only recent, and maize fungicide applications are generally decided on subjective criteria [9].

There is a need to generate new preventive strategies for the management of foliar diseases in crops of interest, like NCLB, in order to reduce fungicide application. This implies that we should adhere to an eco-friendly model of sanitary practices using natural substances typical of the ecosystem to be controlled. In this sense, new preventive strategies could study the capacity of native phyllosphere microorganisms with biofungicide potential. The phyllosphere can be considered an ephemeral habitat in which microorganisms are expected to multiply and use niches as the leaves expand [20]. These microorganisms interact in several ways, with each other and with the plant, by competition, mutualism, commensalism, antibiosis or plant hormone generation [21].

For this, biological control is an alternative strategy to the use of chemical compounds and involves the use of beneficial microorganisms for disease control. The term microbiological control agent (MBCA) applies to the use of antagonist organisms or natural products extracted from them to suppress disease [22]. There are several examples of MBCA application against phytopathogenic moulds [23][24][25][26][27]. In particular, refs. [26][27] searched for microorganisms that are antagonists of *E. turcicum* and can be obtained from the maize plant phyllosphere for the control of NCLB. These authors selected two of these antagonist isolates and applied them to maize plants during a field assay for blight control. Application of *Bacillus* spp. showed a reduction in the disease caused by *E. turcicum*, which was higher than 50% during 40 days with a significant increase in the grain yield compared to the untreated plants [26][27]. Therefore, the use of MBCA becomes a powerful management alternative aimed at minimising the yield losses caused by fungi, including those that cause foliar diseases, improving plant resistance to diseases. The mechanisms by which MBCA can be useful in disease control can be direct, by antagonism of the pathogen, by competition for nutrients or space, antibiosis, mycoparasitism or biofilm formation, or indirect, by inducing a state of resistance on the plant that enhances its defences through biochemical changes against further

infections [28]. The latter represents a convenient strategy for the protection of new leaves. Unlike chemical pesticides with known modes of action, there are difficulties in understanding interactions involving MBCA, plants and pathogens.

To develop a successful foliar biofungicide, it is necessary to understand the mechanisms by which the biocontrol is executed in order to achieve effectiveness. In this sense, the leaf microbiome helps the plant against the attack of pathogens in an indirect manner by activating its defence mechanisms. In plants, there is an innate nonspecific immune system and an acquired or adaptive one, which are differentiated in specificity and memory of the response to the attacking agent [29]. The latter responds to changes or disturbances in the cellular structure caused by pathogens, symbiotic or free-living microorganisms, the application of exogenous chemical substances that act as elicitors, in fertilisation or against abiotic stress [30][31].

In the study of MBCAs controlling diseases, several biochemical indicators can be monitored to determine the physiological activities triggered in the host by the MBCA and/or pathogen that make disease development incompatible. For example, ref. [32] reviewed the modes of action of MBCA against the diseases in general, emphasising screening techniques, risk assessments and practical use. More recently, ref. [33] explored the progress made in the use of the biocontrol agents against fungal plant diseases. Regarding the phyllosphere habitat, in 2012, the authors of [34] compiled knowledge about microbial life in the phyllosphere. Other reviews, such as refs. [35][36] revised the investigations carried out into the epiphytic microbial communities.

2. Plant Immune Response

A plant's induced defences are stimulated once the pathogen enters the plant, and involve two staggered mechanisms: PTI (pattern-triggered immunity) and ETI (effector-triggered immunity) [37][38]. Defensive response may comprise hypersensitive responses and cell death, reactive oxygen species (ROS) generation, stomatal closure, cell wall reinforcement, production of secondary metabolites and pathogenesis-related (PR) proteins [39][40]. Natural selection may favour pathogens that avoid plant immunity, thus leading to a compatible interaction [38].

The different defence mechanisms in plants may be grouped as innate constitutive and basal resistance. In the event that a pathogen successfully avoids the constitutive defences and colonisation takes place, plants rely on inducible immune responses to avoid the disease progress [41]. This sort of defence requires pathogen recognition before deploying active response against the attacker [42]. Following the early signalling events activated by the pathogen attack, elicitor signals are often amplified through the generation of secondary signal molecules, such as salicylic acid (SA), ethylene (ET) and jasmonic acid (JA). In addition, the defence response in the plant–fungal interactions is also closely related to the accumulation of many secondary metabolites, such as flavonoids, phenolic compounds and phytoalexins [43][44]. Pathogen identification relies on the detection of conserved pattern-associated molecular patterns (PAMPs), particular broadly conserved molecules associated with a large range of pathogens, such as flagellin and chitin, by pattern-recognition receptors (PRR) set on the extracellular face of the host cell. This leads to PAMP-triggered immunity (PTI), a basal immune response effective against a broad spectrum of pathogens. PTI limits the pathogen growth by callose accumulation, cell wall strengthening, defence-

related gene activation, ROS production, rapid calcium influx and phosphorylation cascades [35][43][45][46]. The activation of PTI also results in a growth inhibition, revealing the balance between growth and defence.

During coevolution, pathogens develop mechanisms to overcome plant defences and allow parasitism. In these cases, pathogens secrete effector molecules that inhibit or weaken PTI, enabling infection. At the same time plants have evolved the ability to recognise specific pathogen effectors using resistance (R) proteins that activate effector-triggered immunity (ETI), and normally result in ROS and calcium accumulation followed by hypersensitive response and cell death [38][47].

ETI also triggers the biosynthesis of SA and the expression of PR proteins, activating systemic acquired resistance (SAR) and linking the basal to the inducible resistance [41][48]. SAR consists of priming events, mainly associated with large amounts of transcriptional reprogramming once the plant has been exposed to certain pathogens that lead to a much faster and stronger defence response both locally and systemically [49]. SAR induction involves the production of mobile signals that translocate to distant non-attacked tissues to prepare against further infections [50]. Non-pathogenic microbes can also mediate the plant defence response through induced systemic resistance (ISR) [51]. Both SAR and ISR constitute long-term systemic resistance against a broad spectrum of pathogens, but normally their actions are antagonistic, and their range of pathogens may differ. Their signalling pathways are often antagonistic, as SAR depends on the SA pathway, but ISR relies on ET and JA [27][41][52][53]. However, since both pathogens and MBCA are often detected by similar mechanisms in the host, the difference between SAR and ISR is not clear [33].

References

1. Reynolds, T.L.; Nemeth, M.A.; Glenn, K.C.; Ridley, W.P.; Astwood, J.D. Natural variability of metabolites in maize grain: Differences due to genetic background. J. Agric. Food Chem. 2005, 53, 10061–10067.
2. Masisi, K.; Beta, T.; Moghadasian, M.H. Antioxidant properties of diverse cereal grains: A review on in vitro and in vivo studies. Food Chem. 2015, 196, 90–97.
3. Bolsa de Comercio de Rosario. Available online: https://www.bcr.com.ar/es/mercados/gea/estimaciones-nacionales-de-produccion/estimaciones (accessed on 1 June 2023).
4. Sweets, L.E.; Wright, S. Integrated Pest Management: Corn Diseases. Plant Prot. Programs 2008, 22. Available online: http://hdl.handle.net/10355/7284 (accessed on 1 June 2023).
5. de Souza, J. Enfermedades del maíz en Entre Ríos. In Actualización Técnica Maíz, Girasol y Sorgo; INTA EEA Paraná: Paraná, Argentina, 2007; Volume 44, pp. 80–85.

6. Couretot, L. Principales Enfermedades del Cultivo de Maíz en la Zona Norte de la Prov. de Bs. As. Campaña 2009/10. Available online: http://www.inta.gov.ar/pergamino (accessed on 1 June 2023).
7. Díaz, C. Evolución e impacto de enfermedades foliares en el cultivo de maíz: Cercospora y Tizones. In Actas IX Congreso Nacional de Maíz; Rosario, Argentina, 2010; pp. 200–204.
8. Formento, A. Enfermedades Foliares Reemergentes del Cultivo de Maíz: Royas (Puccinia sorghi y Puccinia polysora), Tizón foliar (Exserohilum turcicum) y Mancha ocular (Kabatiella zeae); INTA Paraná: Paraná, Argentina, 2010.
9. De Rossi, R.; Guerra, F.; Plazas, M.; Vuletic, E.; Brücher, E.; Guerra, G.; Reis, E. Crop damage, economic losses, and the economic damage threshold for northern corn leaf blight. Crop. Prot. 2021, 154, 105901.
10. Pratt, R.C.; Gordon, S.G. Breeding for Resistance to Maize Foliar Pathogens. In Plant Breeding Reviews; John Wiley & Sons, Ltd.: Hoboken, NJ, USA, 2010; Volume 27, pp. 119–173.
11. Hurni, S.; Scheuermann, D.; Krattinger, S.G.; Kessel, B.; Wicker, T.; Herren, G.; Fitze, M.N.; Breen, J.; Presterl, T.; Ouzunova, M.; et al. The maize disease resistance gene Htn1 against northern corn leaf blight encodes a wall-associated receptor-like kinase. Proc. Natl. Acad. Sci. USA 2015, 112, 8780–8785.
12. Sucher, J.; Boni, R.; Yang, P.; Rogowsky, P.; Büchner, H.; Kastner, C.; Kumlehn, J.; Krattinger, S.G.; Keller, B. The durable wheat disease resistance gene Lr34 confers common rust and northern corn leaf blight resistance in maize. Plant Biotechnol. J. 2016, 15, 489–496.
13. Chen, G.; Wang, X.; Long, S.; Jaqueth, J.; Li, B.; Yan, J.; Ding, J. Mapping of QTL conferring resistance to northern corn leaf blight using high-density SNPs in maize. Mol. Breed. 2015, 36, 4.
14. Sibiya, J.; Tongoona, P.; Derera, J. Combining ability and GGE biplot analyses for resistance to northern leaf blight in tropical and subtropical elite maize inbred lines. Euphytica 2012, 191, 245–257.
15. Couretot, L.; Ferraris, G.; Mousegne, F.; Russian, H. Control químico de roya común del maíz (Puccinia sorghi). In Proceedings of the 1° Congreso Argentino de Fitopatología, HM-25, Córdoba, Argentino, 28–30 May 2008; p. 211.
16. Carmona, M.; Melo Reis, E.; Trezzi Casa, R. Identificación y manejo de las principales enfermedades del maíz. Horiz. A 2008, 583, 44.
17. Bayer. 2008. Available online: http://www.bayercropscience.com.pe/web/articulo=407 (accessed on 5 March 2023).
18. Batista, A.C.; Domingues, C.E.d.C.; Costa, M.J.; Silva-Zacarin, E.C.M. Is a strobilurin fungicide capable of inducing histopathological effects on the midgut and Malpighian tubules of honey

bees? J. Apic. Res. 2020, 59, 834–843.

19. Samarghandi, M.R.; Mohammadi, M.; Karami, A.; Tabandeh, L.; Dargahi, A.; Amirian, F. Residue Analysis of Pesticides, Herbicides, and Fungicides in Various Water Sources Using Gas Chromatography-Mass Detection. Pol. J. Environ. Stud. 2017, 26, 2189–2195.
20. Baldotto, L.E.B.; Olivares, F.L. Phylloepiphytic interaction between bacteria and different plant species in a tropical agricultural system. Can. J. Microbiol. 2008, 54, 918–931.
21. Kadivar, H.; Stapleton, A.E. Ultraviolet radiation alters maize phyllosphere bacterial diversity. Microb. Ecol. 2003, 45, 353–361.
22. Pal, K.K.; Gardener, B.M. Biological control of plant pathogens. Curr. Opin. Biotechnol. 2006, 7, 343–347.
23. Atehnkeng, J.; Ojiambo, P.; Ikotun, T.; Sikora, R.; Cotty, P.; Bandyopadhyay, R. Evaluation of atoxigenic isolates of Aspergillus flavus as potential biocontrol agents for aflatoxin in maize. Food Addit. Contam. Part A 2008, 25, 1264–1271.
24. Etcheverry, M.G.; Scandolara, A.; Nesci, A.; Ribeiro, M.S.V.B.; Pereira, P.; Battilani, P. Biological interactions to select biocontrol agents against toxigenic strains of Aspergillus flavus and Fusarium verticillioides from maize. Mycopathologia 2009, 167, 287–295.
25. Huang, C.; Yang, K.; Liu, Y.; Lin, Y.; Chen, C. Suppression of southern corn leaf blight by a plant growth-promoting rhizobacterium Bacillus cereus C1L. Ann. Appl. Biol. 2010, 157, 45–53.
26. Sartori, M.; Nesci, A.; Montemarani, A.; Barros, G.; García, J.; Etcheverry, M. Preliminary Evaluation of Biocontrol Agents against Maize Pathogens Exserohilum turcicum and Puccinia sorghi in Field Assays. Agric. Sci. 2017, 8, 1003–1013.
27. Sartori, M.; Nesci, A.; García, J.; Passone, M.A.; Montemarani, A.; Etcheverry, M. Efficacy of Epiphytic Bacteria to Prevent Northern Leaf Blight Caused by Exserohilum turcicum in Maize. Rev. Argent. Microbiol. 2017, 49, 75–82.
28. Legein, M.; Smets, W.; Vandenheuvel, D.; Eilers, T.; Muyshondt, B.; Prinsen, E.; Samson, R.; Lebeer, S. Modes of Action of Microbial Biocontrol in the Phyllosphere. Front. Microbiol. 2020, 11, 1619.
29. Ronald, P.C.; Beutler, B. Plant and animal sensors of conserved microbial signatures. Science 2010, 330, 1061–1064.
30. Bent, A.F.; Mackey, D. Elicitors, effectors, and R genes: The new paradigm and a lifetime supply of questions. Annu. Rev. Phytopathol. 2007, 45, 399–436.
31. Spoel, S.H.y.; Dong, X. How do plants achieve immunity? Defence without specialized immune cells. Nat. Rev. Immunol. 2012, 12, 89–100.

32. Köhl, J.; Kolnaar, R.; Ravensberg, W.J. Mode of action of microbial biological control agents against plant diseases: Relevance beyond efficacy. Front. Plant Sci. 2019, 10, 845.
33. Palmieri, D.; Ianiri, G.; Del Grosso, C.; Barone, G.; De Curtis, F.; Castoria, R.; Lima, G. Advances and perspectives in the use of biocontrol agents against fungal plant diseases. Horticulturae 2022, 8, 577.
34. Vorholt, J.A. Microbial life in the phyllosphere. Nat. Rev. Microbiol. 2012, 10, 828–840.
35. Bashir, I.; War, A.F.; Rafiq, I.; Reshi, Z.A.; Rashid, I.; Shouche, Y.S. Phyllosphere microbiome: Diversity and functions. Microbiol. Res. 2021, 254, 126888.
36. Leveau, J.H. A brief from the leaf: Latest research to inform our understanding of the phyllosphere microbiome. Curr. Opin. Microbiol. 2019, 49, 41–49.
37. Pruitt, R.N.; Gust, A.A.; Nürnberger, T. Plant immunity unified. Nat. Plants 2021, 7, 382–383.
38. Jones, J.D.G.; Dangl, J.L. The plant immune system. Nature 2006, 444, 323–329.
39. Castro-Moretti, F.R.; Gentzel, I.N.; Mackey, D.; Alonso, A.P. Metabolomics as an emerging tool for the study of plant–pathogen interactions. Metabolites 2020, 10, 52.
40. Pieterse, C.M.J.; Leon-Reyes, A.; Van der Ent, S.; Van Wees, S.C.M. Networking by small-molecule hormones in plant immunity. Nat. Chem. Biol. 2009, 5, 308–316.
41. Molina, A.; Sánchez Vallet, A.; Sánchez-Rodríguez, C. Inmunidad innata en plantas y resistencia a patógenos: Nuevos conceptos y potenciales aplicaciones en protección vegetal. Phytoma España Rev. Prof. Sanid. Veg. 2007, 192, 43–46.
42. Thaler, J.S.; Humphrey, P.T.; Whiteman, N.K. Evolution of jasmonate and salicylate signal crosstalk. Trends Plant Sci. 2012, 17, 260–270.
43. Lanubile, A.; Ferrarini, A.; Maschietto, V.; Delledonne, M.; Marocco, A.; Bellin, D. Functional genomic analysis of constitutive and inducible defense responses to Fusarium verticillioides infection in maize genotypes with contrasting ear rot resistance. BMC Genom. 2014, 15, 710.
44. Wang, F.; Wang, C.; Yan, Y.; Jia, H.; Guo, X. Overexpression of cotton GhMPK11 decreases disease resistance through the gibberellin signaling pathway in transgenic Nicotiana benthamiana. Front. Plant Sci. 2016, 7, 689.
45. Hacquard, S.; Spaepen, S.; Garrido-Oter, R.; Schulze-Lefert, P. Interplay between Innate Immunity and the Plant Microbiota. Annu. Rev. Phytopathol. 2017, 55, 565–589.
46. Serrano, I.; Audran, C.; Rivas, S. Chloroplasts at work during plant innate immunity. J. Exp. Bot. 2016, 67, 3845–3854.
47. Withers, J.; Dong, X. Post-translational regulation of plant immunity. Curr. Opin. Plant Biol. 2017, 38, 124–132.

48. Ngou, B.P.M.; Jones, J.D.; Ding, P. Plant immune networks. Trends Plant Sci. 2021, 27, 255–273.
49. Zeier, J. Metabolic regulation of systemic acquired resistance. Curr. Opin. Plant Biol. 2021, 62, 102050.
50. Shine, M.B.; Xiao, X.; Kachroo, P.; Kachroo, A. Signaling mechanisms underlying systemic acquired resistance to microbial pathogens. Plant Sci. 2019, 279, 81–86.
51. Groen, S.C.; Whiteman, N.K. The Evolution of Ethylene Signaling in Plant Chemical Ecology. J. Chem. Ecol. 2014, 40, 700–716.
52. Ton, J.; Van Pelt, J.A.; Van Loon, L.C.; Pieterse, C.M.J. Differential Effectiveness of Salicylate-Dependent and Jasmonate/Ethylene-Dependent Induced Resistance in Arabidopsis. Mol. Plant-Microbe Interact. 2002, 15, 27–34.
53. Van der Ent, S.; Van Wees, S.C.; Pieterse, C.M. Jasmonate signaling in plant interactions with resistance-inducing beneficial microbes. Phytochemistry 2009, 70, 1581–1588.