Grapevine defence mechanisms when challenged by pathogenic fungi and oomycetes

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Contents

Abstract

Traditional cultivated grapevine (*Vitis vinifera*) is susceptible to many fungal and oomycete pathogens causing devastating diseases including powdery mildew, downy mildew, grey mould, black rot and trunk diseases. These infections trigger various defence mechanisms such as reinforcement of the cell wall structure, production of phytoalexins and pathogenesis-related proteins, and localized cell death. In *V. vinifera* susceptible varieties, these defences are not effective, while in resistant grapevine, recognition of the pathogen induces effective mechanisms that stop the infection. Breeding programmes are conducted to take advantage of this genetic resistance. Moreover, a range of exogenous defence stimulators can be used to obtain a so-called "induced resistance" in susceptible

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Advances in Botanical Research
ISSN 0065-2296, https://doi.org/10.1016/bs.abr.2024.02.013

varieties. This chapter presents the recently acquired knowledge on the molecular mechanisms involved in genetic and induced resistances, and further consider other mechanisms such as ontogenic resistance. It also suggests how to exploit these resistances to durably protect vineyards against the different fungal diseases.

1. Introduction

All living organisms must find their place in an ecosystem if they are to survive, and so they interact with one another. In the case of plant-pathogen interaction, a complex process takes place involving molecules derived from both the pathogen and the plant. Molecules derived from pathogens are key factors in determining their pathogenicity and virulence. On the plant side, recognition of the pathogen triggers defence responses contributing to its level of resistance. Plant defences have been the subject of extensive research for several decades, and the molecular dialogue between plant and pathogens, as well as the mechanisms of resistance are increasingly well described (Andersen, Ali, Byamukama, Yen, & Nepal, 2018).

Grapevines are a major crop worldwide, with about 10,000 varieties, and 33 of which covers 50% of the world's vineyards (OIV, 2017). This culture is confronted to important diseases due to fungi and oomycetes. All grapevine varieties are not equally susceptible to these pathogens and have defence systems of varying effectiveness. Therefore, improving their defences to the main vine diseases by breeding and/or stimulating with exogenous biotic or abiotic substances acting as plant defence stimulators (PDS), is a major challenge for the conservation of this crop in a context of global change and limited fungicide treatments.

This chapter first reviews the recently acquired knowledges on molecular plant-pathogen interaction and lists the main diseases of grapevine due to fungal and fungal-like (oomycete) pathogens. In a second part, we describe the panel of defence mechanisms occurring in resistant *Vitis* species and varieties, and then we present the current state of knowledge concerning perception of the pathogens, defence responses in susceptible varieties, elicitor-induced resistance, basis of genetic resistance and, finally, ontogenetic resistance/tolerance. Each situation will lead to the induction of defences with different efficiencies against the pathogens.

1.1 Molecular dialogue in plant-pathogen interactions

All plants are steadily subject to an environment rich in potentially pathogenic microbes, such as bacteria, fungi, oomycetes or viruses. Microbes

(either pathogenic or symbiotic) infect plants to pump nutrients for their growth and development. Nevertheless, plants are resistant to most microbes due to an efficient immune system, combining constitutive and inducible defence responses (Fig. 1).

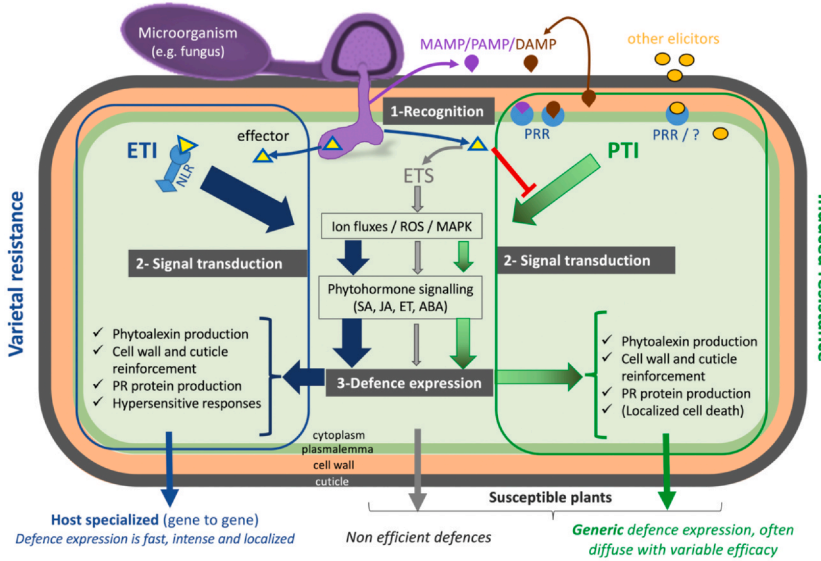

Fig. 1 Overview of the three main mechanisms of grapevine defences after an attack by microorganisms (*e.g.* fungi). The dark grey boxes give the major steps of defence mechanisms. **Recognition:** Plant perception of molecules produced by the microbe/pathogen (Microbe/pathogen-associated molecular pattern, MAMPs/PAMPs), molecules derived from the degradation of the plant's (Damaged associated molecular pattern/DAMPs), other elicitors (green dot) or fungal effectors (yellow triangles) *via* extra or intra cellular various receptors (usually in the plasma membrane or in the cytosol). Recognition of PAMPs, MAMPs/DAMPs and elicitors leads to the PAMP-triggered immunity (**PTI**, in green). Effectors from the pathogen can partially block the plant defence mechanisms and ultimately conduct to effector-triggered susceptibility (**ETS**, in grey). Finally, recognition of effectors through NBS-LRR type receptors results in effector-triggered immunity (**ETI**, in blue). **Signal transduction:** through calcium and ion fluxes, reactive oxygen species (ROS) formation, mitogen activated protein kinase (MAPK) activities and phytohormones (salicylic acid (SA), jasmonate (JA), ethylene (ET) and abcissic acid (ABA) that relay the signal). **Defence expression:** activation of genes involved in the phytoalexin production, in parietal reinforcements (in cuticle/cell wall with callose and/or lignine as examples), in pathogenesis-related proteins synthesis and cell death. The hypersensitive response (HR) is rather specific to **ETI** but, occasionally and depending on the elicitor perceived, a cell death process may occur for **PTI** (induced resistance). Ontogenic resistance is not represented here, but is thought to be similar to PTI-like with an age-dependent manner.

The constitutive defences are formed by physical and chemical barriers, such as cuticle, cell walls, and antimicrobial phytoanticipins. If a microorganism manages to cross this first line of defence, the plant can detect it and responds by activating defence responses. Immune receptors indeed detect a variety of molecules recognized as "non-host" or "danger" signals and switch on complex system of defence tools. These molecule signals are therefore called elicitors, as they elicit host responses (Jones & Dangl, 2006). Mechanisms of these immune responses are similar to the innate immunity described in animals. This is an ancient, broad-spectrum defence strategy with germ-line encoded components. Unlike jawed vertebrate animals, plants lack the adaptive immunity capable of the specific antibody production. On the other hand, all plant cells (not only specialized immune cells) can activate innate immunity in an autonomous manner through their evolved surveillance system (Cook, Mesarich, & Thomma, 2015; Jones, Vance, & Dangl, 2016; Albert, Hua, Nürnberger, Pruitt, & Zhang, 2020).

The main and evolutionary older layer of this inducible immunity is based on the external recognition of conserved microbial signatures called microbe/pathogen-associated molecular patterns (MAMPs/PAMPs) that are generated during microbial attack. The early external recognition is also achieved with the host-derived damage-associated molecular patterns (DAMPs) produced as a consequence of enzymatic microbial activities and toxins (Boller & Felix, 2009; Dodds & Rathjen, 2010; Monaghan & Zipfel, 2012; Gust, Pruitt, & Nürnberger, 2017). MAMPs and DAMPs are recognized by the plasma-membrane localized pattern recognition receptors (PRRs) and induce a broad variety of defence responses commonly referred to as PAMP-triggered immunity (PTI) or even basal immunity (Zipfel, 2014; Couto & Zipfel, 2016; Boutrot & Zipfel, 2017).

However successful pathogens can secrete effectors, pathovar-specific microbial molecules that are delivered into host cells to suppress or interfere with PTI responses, resulting in facilitated host colonization and effector-triggered susceptibility (ETS). These effectors are secreted either in the extracellular space within the plant tissues (apoplastic effectors) or in the host cells (cytoplasmic effectors) to directly manipulate host cellular process (Lovelace et al., 2023). Most known pathogen effectors are small proteins but secondary metabolites and small RNAs were more recently also identified as effectors manipulating plant immunity (Weiberg et al., 2013; Collemare, O'Connell, & Lebrun, 2019). In an ongoing arms-race between the host and attacking microorganism, another more specialized layer of microbial detection evolved more recently, termed effector-triggered immunity (ETI). In ETI, host-specific

intracellular receptors known as resistance (R) proteins detect the presence or activities of effectors. Host can sense effector activity by monitoring perturbations in a few key cellular processes. As the effector recognition reduces the pathogen virulence, effectors are also referred to as avirulence (Avr) products (Jones & Dangl, 2006). The bases for formulating the ETI model were already set by Flor proposing the gene-for-gene resistance (Flor, 1971). According to this concept, the resistance or the disease outcome is controlled by corresponding gene pairs, encoding the R and Avr proteins in the plant or the pathogen, respectively. Upon the co-evolution of a host with its pathogen, the ETI can be broken by new effector(s) leading again to ETS. The co-evolution of host immunity along with pathogen's effectors can be suitably illustrated by the zig-zag model (Jones & Dangl, 2006).

Interactions between plants and pathogenic microbes can be classified according to the mechanisms of the "molecular dialog" and the disease outcome. A non-host interaction is established between a plant and a non-adapted pathogen that lacks specialized effectors to disrupt immunity of a given plant. In this case, PTI responses are sufficient to block pathogen and result in a lack of disease. We speak about the incompatible interaction, when a pathogen overcomes PTI but is stopped by the recognition its effector(s) by the plant. The resulting ETI leads to a resistance. In contrast, the compatible interaction occurs when the effective ETI is missing or was overcome by novel effectors, finally leading to disease.

MAMPs are primarily molecular motifs with essential functions for microorganisms, making them conserved in many organisms of the same family (Newman, Sundelin, Nielsen, & Erbs, 2013). Thus, they constitute an effective and sustainable warning system for the plant species targeted by these microorganisms. Among bacterial MAMPs, there are, for example, peptidoglycans (PGN), exopolysaccharides (EPS) and flagellin, a protein constituting the structure of the flagellar filament necessary for the mobility of bacteria, which is mainly recognized *via* its flg22 epitope perceived by many plant species (Felix, Duran, Volko, & Boller, 1999; Gomez-Gomez & Boller, 2000; Gust et al., 2007; Sanabria, Huang, & Dubery, 2010; Abdalla et al., 2021). In fungi, chitin, a polymer of N-acetyl-D-glucosamines constituting fungal cell walls is the MAMP that has been the most studied. Interestingly for application in crop protection, chitin induces an elicitor effect on many plant species (Miya et al., 2007; Kishimoto et al., 2010; Burketová, Trdá, Ott, & Valentová, 2015; Bozsoki et al., 2017; Leppyanen et al., 2017; Brulé et al., 2019). In addition to fungal cell walls, certain enzymes released by pathogenic fungi to destroy plant cell walls can also be

detected by the plant, regardless of the DAMPs they produce. This is particularly the case for endo-xylanases and endo-polygalacturonases, which retain an elicitor activity despite a loss of function, *i.e.* the production of xylans and oligogalacturonates (OGs). These two enzymes excreted by fungi can therefore also be considered as MAMPs (Poinssot et al., 2003; Ron & Avni, 2004; Zhang et al., 2014).

The cell walls of oomycetes are different in their composition to those of fungi but generally contain β-1,3-glucans, cellulose, and some chitin, all possessing an elicitor activity (Aziz et al., 2007; Mélida et al., 2018; Rebaque et al., 2021). To carry out their infectious cycle, these pathogens also secrete xylanases or glucoside hydrolases that can be detected as MAMPs (Enkerli, Felix, & Boller, 1999; Furman-Matarasso et al., 1999; Wang, Xu et al., 2018; Sun et al., 2022). A membrane sphingolipid, extracted from the oomycete *Phytophthora infestans*, has also been described as a MAMP (Kato et al., 2022).

In addition to MAMPs, some plant-specific molecules can be released in case of injury and recognized by other PRRs to induce defensive reactions in plants. These endogenous molecules, or DAMPs, are mainly oligosaccharides or proteins, but can also take the form of small molecules such as extracellular cofactors (eATP/eNAD$^+$), extracellular DNA/RNA, or extracellular hydrogen peroxide (eH_2O_2) (Gust et al., 2017; Wu et al., 2020). The oligosaccharides are plant cell wall fragments, including cello-dextrins (CDs) and cellobioses resulting from cellulose degradation (Aziz et al., 2007; Souza et al., 2017), oligogalacturonates (OGs) resulting from the degradation of pectin-derived homogalacturonans (Poinssot et al., 2003; Ferrari et al., 2007; Ferrari et al., 2013; Benedetti et al., 2015), xyloglucans (Claverie et al., 2018) and mixed-bond β-1,3/1,4-glucans from hemicellulose (Rebaque et al., 2021). Among protein DAMPs, there are many peptides from proteolytic cleavages and secreted during injuries or following the detection of PAMPs. Plant-eliciting peptides (PEPs) are derived from precursors, PROPEPs, located in the basal state at the tonoplast and released under the action of metacaspases (Yamaguchi & Huffaker, 2011; Bartels et al., 2013; Hander et al., 2019). Related to PEPs, PAMP-induced peptides (PIPs) are formed in a similar way but have the particularity of being matured only after being secreted in the apoplasm in order to amplify the PTI (Hou et al., 2014; Shen et al., 2020). Rapid alkalinization factors (RALFs) are another group of endogenous peptides also excreted following the perception of a PAMP. Unlike the peptides mentioned above, RALFs can both stimulate and suppress immunity by acting as positive or negative regulators of PTI (Pearce, Moura, Stratmann, & Ryan, 2001; Stegmann et al., 2017).

Specific receptors, i.e. PRRs, located in the plasma membrane can perceive both MAMPs and DAMPs. PRRs can be serine/threonine receptor-like kinases (RLKs), allowing them to transduce the signal inside the cell, or they may not have a kinase domain and they are called RLPs (receptor-like proteins). The genome of the model plant *Arabidopsis thaliana* would include 417 RLKs against only 57 RLPs (Shiu & Bleecker, 2001; Shiu et al., 2004). We distinguish PRRs with LRR domain (leucine rich repeats) which perceive peptides or small molecules, PRRs with LysM domain (lysin motifs) which perceive rather oligosaccharides, PRRs with malectin domain which perceive peptides, PRRs with EGF-like domain (epidermal growth factor-like domain) which can perceive peptides or oligosaccharides, and finally lectin domain PRRs that can perceive some molecular motifs mentioned above (Ngou, Ding, & Jones, 2022). Finally, chitin oligomers are recognized by a heterodimeric receptor complex involving the lysin motif (LysM)-RLKs AtCERK1/LYK1 and AtLYK5 (Miya et al., 2007; Wan et al., 2008; Petutschnig, Jones, Serazetdinova, Lipka, & Lipka, 2010; Cao et al., 2014).

Once the elicitors or effectors are recognized by the plant, plant defences are activated and finely regulated by phytohormones, namely salicylic acid (SA), jasmonic acid (JA), and ethylene (ET) (for review, see Nawaz et al., 2023) to ensure a basal resistance to pathogens.

Beyond the recognition of the pathogen, the resistance of the plant also depends on the speed and intensity of the establishment of defence mechanisms. The acceleration and strengthening of basal defence responses then give the plant increased resistance to biotic stresses. The priming of defence reactions can be induced by pathogens (Sticher, MauchMani, & Metraux, 1997), by beneficial organisms such as rhizobacteria (Pieterse et al., 1998) or mycorrhizae (Pozo et al., 2002), by herbivores or injury (Ton et al., 2007; Chassot, Buchala, Schoonbeek, Métraux, & Lamotte, 2008), by a spectrum of natural or synthetic compounds such as lipopolysaccharides (LPS; Newman, Von Roepenack-Lahaye, Parr, Daniels, & Dow, 2002), SA (Mauch-Mani & Metraux, 1998), β-aminobutyric acid (BABA) (Zimmerli, Jakab, Metraux, & Mauch-Mani, 2000) or sulphated laminarin (PS3; Trouvelot et al., 2008; Gauthier et al., 2014). Although the mechanisms and genetic basis of priming remain largely unknown, it has been proposed that sensitization of the plant by a priming activator may be associated with an accumulation of proteins in an inactive form. Then, under biotic or abiotic stress, these proteins would be activated, thus amplifying the signal transduction leading to an induction of faster and/or

more intense defence responses (Conrath et al., 2006). Moreover, in plants and mammals, defence priming can also be associated with elevated levels of PRRs and with histone modifications in defence gene promoters, finally leading to faster plant immune responses (Conrath, Beckers, Langenbach, & Jaskiewicz, 2015).

1.2 Diseases caused by fungi and oomycetes on grapevine

Grapevine is confronted by many diseases caused by eukaryotic filamentous microorganisms belonging to the fungi and oomycete phyla (more than forty, Wilcox, Gubler, & Uyemoto, 2015). The pathogenic species responsible for these diseases present a high diversity in term of taxonomy, infection strategy, symptoms on cultivated grapevine and host range. The main economically important diseases are listed in Table 1 and shortly described below. They can be initiated on all the organs of grapevine either the young rapidly growing green tissues, the mature berries or the trunk.

The green tissues such as the leaves, shoots, inflorescences and young berries are susceptible to several pathogens that develop, at least in the initial stage of the infection, a biotrophic interaction *e.g.* an intimate interface with the host cells that allow them to feed on alive tissues. Most of these pathogens have host range limited to the *Vitis* species or species of related genus. One of the most important worldwide is *Plasmopara viticola,* the oomycete responsible for Downy mildew (DM) which is endemic to North America and was introduced in Europe in the late 1870s (Gessler, Pertot, & Perazzoli, 2011; Dussert et al., 2019; Fontaine et al., 2021; Koledenkova et al., 2022). *P. viticola* is an obligate pathogen which means that it relies fully on host cells in photosynthesis-active tissues to complete its life cycle. Under appropriate climate conditions *i.e.* high humidity and moderate temperature, zoospores of *P. viticola* settle on the abaxial surface of leaves, germinate and penetrate the stomatal cavities. Once *in planta*, hyphae grow through intercellular spaces and enter the cells of the mesophyll to form haustoria *e.g.* typical intracellular feeding structures made by biotrophic pathogens. The formation of haustoria involves the perforation of the plant cell wall but the plasma membrane of the host remains intact as an invagination surrounding the *P. viticola* cell. The symptoms on leaves are oil-spot lesions on the adaxial surface. Finally, sporangiophores emerge from the infected tissues and release sporangia, the source of zoospores for a new infection cycle. Powdery mildew (PM) is another major worldwide disease of the *Vitis* species that is due to an obligate pathogen originated from North America: the ascomycete *Erysiphe necator* (previously *Uncinula necator*). This fungus infects photosynthesis-active tissues

Table 1 Economically important grapevine diseases due to ascomycete (A), basidiomycete (B) and oomycete (O) pathogens.

	Pathogen species	Infected tissues	Infection strategy	Known host plants
Downy mildew (DM)	*Plasmopara viticola* (O)	leaves, berries and shoots	Obligate biotroph, endoparasite, grows between and inside (haustoria) host cells	*Vitaceae* species
Powdery mildew (PM)	*Erysiphe necator* formerly *Uncinula necator* (A)	leaves, berries and shoots	Obligate biotroph, ectoparasite, feeds (haustoria) on host cells and grows on the surface of leaves.	*Vitis* species
Black rot (BR)	*Phyllosticta ampelicida* also named *Guignardia bidwellii* (A)	leaves, berries and shoots	Hemibiotroph, grows in the subcuticullar area	*Vitis* species
Anthracnose (AN)[a]	*Elsinoe ampelina* (A)	leaves, berries and shoots	Hemibiotroph, grows first between epidermal host cells then inside host cells (necrotrophic stage)	Several *Rubus* and *Vitis* species
Grey mould (GM)[a]	*Botrytis cinerea* (A)	Mainly mature berries, flowers	Necrotroph, kills host cells before invading them	More than 1400 genus
Grapevine trunk diseases (GTDs):		Trunk vascular tissues	Grow in xylem vessels	Generally, several woody species

(*continued*)

Table 1 Economically important grapevine diseases due to ascomycete (A), basidiomycete (B) and oomycete (O) pathogens. (*cont'd*)

	Pathogen species	Infected tissues	Infection strategy	Known host plants
Eutypa dieback[a]	*Eutypa lata* (A)			
Esca[a]	*Phaeomoniella chlamydospora* (A)			
	Phaeoacremonium minimum (A)			
	Fomitiporia mediterranea (B)			
Botryosphaeria dieback[a]	*Botryosphaeria dothidea* (A)			
	Diplodia seriata, *D. mutila* (A)			
	Neofusicoccum parvum (A)			
	Lasiodiplodia theobromae, *L. viticola* (A)			
Phomopsis dieback[a]	*Diaporthe ampelina formerly Phomopsis viticola* (A)			
Black foot disease[a]	*Dactylonectria macrodidyma*, *D. novozelandica* (A)			

[a]indicates that other fungal species could be responsible for the disease. Only the major ones are indicated.

especially in dry and warm weather which results in reduced yields and fruit and wine quality (Darriet et al., 2002; Calonnec, Cartolaro, Poupot, Dubourdieu, & Darriet, 2004; Gadoury et al., 2012). Infection begins by the attachment and germination of a conidium on the epidermis. The germ tube differentiates into an appressorium, a specialized infection structure that allows the penetration of the host epidermal cell wall and the formation of an haustorium. If the establishment of this feeding structure is successful, the fungus continues to colonize the surface *via* hyphae producing additional haustoria (Qiu, Feechan, & Dry, 2015). Finally, conidiophores emerge carrying conidia from infected tissues resulting in a greyish-white powder.

In addition to these two major biotrophic pathogens causing DM and PM, grapevine is also confronted to hemibiotrophic fungi that first develop a biotrophic symptomless phase and then switch to a necrotrophic and damaging phase where they kill host cells and feed on them. This infection strategy occurs in the cases of the Black rot (BR) and Anthracnose (AN), re-emerging diseases (Pirrello et al., 2019). The ascomycete responsible for BR is *Phyllosticta ampelicida* more generally called *Guignardia bidwellii,* a species native from North America, present in South America and Europe and extending to China (Crandall et al., 2022; Szabó et al., 2023). This fungus infects young rapidly growing green tissues. On young leaves, pycnidiospores or ascospores germinate in favour of long wetting period and form appressorium. Once *in planta*, the biotrophic mycelium develop specifically in the space between the cuticle and the anticlinal epidermal cell walls. Finally, after a couple of weeks, necrotic spots are formed on leaves and berries and the two-dimensions hyphal nets start to generate pycnidia (generating asexual pycniospores) (Ullrich, Kleespies, Enders, & Koch, 2009). Infected berries then turn into black mummies and produce perithecia, the source of ascospores. For *Elsinoe ampelina*, the main agent of AN occurring in humid and warm areas, the localisation of the biotrophic mycelium is different: after the germination of ascospores, formation of appressoria and penetration of epidermal cells, the infection hyphae develop in the intercellular spaces all around the epidermal cells. After a couple of days, the fungus switches to intracellular development causing cellular damages and macroscopic symptoms such as reddish circular spots (Li, Zhang et al., 2019).

Grapevine berries are confronted to another major disease, Grey Mould (GM) which is mainly caused by *Botrytis cinerea*. This polyphagous and necrotrophic ascomycete has a large host range (more than 1400 host plant species) causing significant damages in most cultivated fruits (Elad, Pertot, Cotes Prado, & Stewart, 2016). Nevertheless, recent genomic studies revealed

distinct co-existing populations that show a certain level of host specialization on their host of origin notably grapevine or tomato (Mercier et al., 2019; Mercier et al., 2021; Simon et al., 2022). On grapevine, *B. cinerea* may infect different organs but mature berries are the most susceptible ones (Elmer & Michailides, 2007; Deytieux-Belleau et al., 2009; Kelloniemi et al., 2015), thus causing substantial damages to the phenolic and organoleptic properties of wines (Ky et al., 2012). The fungal infection is facilitated by skin pores and injuries present on berries. Conidia germination led to formation of unicellular appressoria and multicellular appressoria, also called "infection cushions". From one appressorium, a penetration peg is formed through the host cuticle and fungal colonization occurs between the cuticle and the epidermal outer wall, and then between the deeper cell layers. Host cell death is rapidly observed in the surrounding tissues (1–2 days post inoculation (dpi)) and the fungus develops both between and inside the damaged cells. This necrotrophic development is based on the production of a range of cell wall degrading enzymes, reactive oxygen species and toxic proteins and metabolites such as botrydial and botcinic acid (Choquer et al., 2007; Kelloniemi et al., 2015; Bi, Liang, Mengiste, & Sharon, 2023). An alternative infection strategy where *B. cinerea* infects grapevine inflorescences and stays latent until the maturation of the berries, was also reported (Viret, Keller, Jaudzems, & Cole, 2004). The infection cycle is completed with the formation of conidiophores on the infected tissues and the release of conidia. In addition, this pathogen has also developed a relationship of mutual benefit with the insect pest *Lobesia botrana*, favouring the development of the insect on the berries and spread of *B. cinerea* (Fermaud & Lemenn, 1992; Mondy, Charrier, Fermaud, Pracros, & Corio-Costet, 1998). In addition to *B. cinerea*, two other *Botrytis* species namely *B. pseudocinerea* and *B. sinoviticola* can cause GM on grapevine but they make a negligible contribution to epidemics in vineyards (Walker et al., 2011; Zhou et al., 2014). Although GM is a major concern in post-harvest and wine making industry, mature berries are the target of many other problematic late-season rots (Crandall et al., 2022).

Regarding the woody tissues of grapevine, they are confronted to the increased occurrence of three main Grapevine Trunk Diseases (GTDs) namely Eutypa dieback, Esca, Botryosphaeria dieback, but also Phomopsis dieback and Black foot disease (Mondello et al., 2018; Azevedo-Nogueira et al., 2022). Each of these slow perennial diseases is caused by one or several fungal species (Table 1), some of them known to infect other trees (*e.g.* almond, apricot, mango). These pathogenic fungi infect the perennial organs of the grapevine mainly through pruning wounds, develop into the xylem, lead to leaf and

berry symptoms and finally to the plant death. As the fungal pathogens were only isolated from the woody parts of the plant, the symptoms observed on the leaves and berries are expected to be due to the translocation of fungal phytotoxic metabolites through the transpiration stream (Bertsch et al., 2013; Fontaine et al., 2016). More than eighty fungal species have been isolated from wood showing symptoms of GTDs (*i.e.* discoloration, necrosis or decays) and a majority of them were additionally proved pathogenic by artificial inoculation. In the case of Esca which is actually a complex of diseases, different sets of ascomycete and basidiomycete species are successively responsible for different syndromes (Dark wood streaking, Petri disease, Leaf stripe, white rot and Esca proper) according to the growth stage of the vine (Mondello et al., 2018).

As exemplify above, grapevine pathogens show very distinct strategies to develop in tissues, especially during the early stages of penetration and biotrophic interaction. Although some fungal virulence factors are shared to develop infection structures (appressoria, haustoria…) or to degrade plant cell wall (lytic enzymes), the molecular host-pathogen dialogue can't be predicted from one disease to another.

2. Grapevine defences

Like most plants, grapevine is able to respond to pathogen attack by developing defence mechanisms, or even developing systemic acquired resistance (SAR) (Gao, Zhu, Kachroo, & Kachroo, 2015; Liu, Wu et al., 2016). The grapevine's immune system is based on recognizing molecules which trigger transduction signals and ultimately lead to developing defences. This innate immunity can be either basal and constitutive (based on genetic resistance, ontogenic resistance) or the result of an exogenous induction. *Vitis* species uses several mechanisms to defend itself up to the point of partial or total resistance.

Grapevine defences have been studied either in a context of interactions with microorganisms, or in response to exogenous elicitors. Experiments were performed at different scales: cell suspensions, *in vitro* plantlets, plants grown in controlled conditions, and vines in the vineyard (Table 2). As described above for the model plant *A. thaliana*, elicitor perception is the first crucial step for defence activation in *V. vinifera* as it triggers (ROS) and nitric oxide production, the plasmalemma depolarization, ion fluxes, increase of cytoplasmic Ca^{2+} concentration, and the activation of mitogen-activated

Table 2 Methods to study grapevine defence responses and visualize interaction with pathogens.

	Defence response	Methodology	Plant material	References
ROS production, signalling events	ROS production	Luminol chemiluminescence/ luminometer	Cell culture	Aziz et al. (2003); Poinssot et al. (2003); Aziz, Heyraud, and Lambert (2004); Vandelle, Poinssot, Wendehenne, Bentejac, and Pugin (2006); Aziz et al. (2007); Varnier et al. (2009); Dubreuil-Maurizi et al. (2010); Caillot et al. (2012); Gauthier et al. (2014); Lachhab et al. (2014); Claverie et al. (2018); Krzyzaniak, Trouvelot et al. (2018); Paris et al. (2019); Heyman, Ferrarini et al. (2021)
		L-012-based chemiluminescence/ luminometer	Roots	Heyman, Ferrarini et al. (2021)
		FOX1 method (peroxide-mediated oxidation of Fe^{2+}, followed by reaction of Fe^{3+} with xylenol orange)/spectrometry	Cell culture	Boubakri et al. (2012); Belchí-Navarro, Almagro et al. (2013); Boubakri, Chong et al. (2013); Boubakri, Wahab et al. (2013); Belchí-Navarro, Rubio, Pedreño, and Almagro (2019)
		Titanium peroxide complex/spectrometry	Berries collected in the field	Wang et al. (2015)

	Reaction with cerium chloride ($CeCl_3$), producing electron-dense deposits of cerium perhydroxides/ transmission electron microscopy	Cell culture	Belchí-Navarro et al. (2019)
	2′,7′-dichlorofluorescein diacetate (DCF-DA)/ Confocal laser scanning microscopy	Cell culture	Belchí-Navarro et al. (2019)
	Diaminobenzidine (DAB)/ light microscopy	Leaves from foliar cuttings	Trouvelot et al. (2008); Dubreuil-Maurizi et al. (2010); Boubakri et al. (2012); Boubakri, Chong et al. (2013); Kim Khiook et al. (2013); Gauthier et al. (2014); Paris et al. (2019)
	Nitroblue tetrazolium staining/light microscopy	Leaves from *in vitro* plantlets	Kortekamp and Zyprian (2003)
	Pharmacological approach (DETC)	Cell culture	Faurie, Cluzet, and Mérillon (2009)
Culture medium alcalanization	pH measurement	Cell culture	Aziz et al. (2003); Qiao et al. (2010); Belchí-Navarro, Almagro et al. (2013); Wang, Duan et al. (2022); Sofi et al. (2023)

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Table 2 Methods to study grapevine defence responses and visualize interaction with pathogens. (*cont'd*)

Defence response	Methodology	Plant material	References
Plasma membrane depolarization	DiBAC4 (Bis (1,3-dibarbituric acid)-trimethine)/fluorimetry	Cell culture	Vandelle et al. (2006); Gauthier et al. (2014)
Free cytosolic calcium variation	Aequorin bioluminescence/ luminometer	Apoaequorin-expressing cell culture	Aziz et al. (2003); Vandelle et al. (2006); Aziz et al. (2007); Varnier et al. (2009); Dubreuil-Maurizi et al. (2010); Gauthier et al. (2014); Lachhab et al. (2014)
Calcium influx	$^{45}Ca^{2+}$/scintillation counter	Cell culture	Aziz et al. (2003); Poinssot et al. (2003); Vandelle et al. (2006)
	Pharmacological approach (LaCl3, verapamil, EGTA), caffeine and ruthenium red	Cell culture	Faurie, Cluzet et al. (2009)
NO accumulation	4,5-diaminofluorescein diacetate (DAF-DA)/ fluorimetry or confocal laser scanning microscopy	Cell culture	Vandelle et al. (2006); Dubreuil-Maurizi et al. (2010); Belchí-Navarro et al. (2019)
	Griess reagent method	Cell culture	Belchí-Navarro et al. (2019)
Phosphorylation	Pharmacological approach (cantharidin and endothall)	Cell culture	Faurie, Cluzet et al. (2009)

Lipid peroxidation	Reaction product malondialdehyde/ spectrophotometry	Leaves	Wang, Duan et al., 2022
MAP Kinase activation	Western blot and in-gel kinase assays	Cell culture	Aziz et al. (2003); Poinssot et al. (2003); Vandelle et al. (2006); Varnier et al. (2009); Dubreuil-Maurizi et al. (2010); Gauthier et al. (2014); Lachhab et al. (2014); Krzyzaniak et al. (2018); Brulé et al. (2019); Paris et al. (2019)
Signalling hormones	HPLC/Fluorimetry (SA)	Leaves from wood cuttings	Bodin et al. (2020)
	LC-MS (SA and JA)	Leaves from foliar cuttings	Gauthier et al. (2014)
	HPLC MS/MS (JA and ABA)	Leaves of one-year-old potted grapevines grown outside	Pagliarani et al. (2020)
	UPLC MS/MS (SA, JA, and other phytohormones)	Leaves from wood cuttings	Zarraonaindia et al. (2023)
Gene expression	Southern blot	Leaves from cuttings	Busam et al. (1997)

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Table 2 Methods to study grapevine defence responses and visualize interaction with pathogens. (*cont'd*)

Defence response	Methodology	Plant material	References
	Northern blot	Cell culture	Busam et al. (1997); Aziz et al. (2003); Poinssot et al. (2003); Vandelle et al. (2006)
	Northern blot	Leaves from seedlings	Hamiduzzaman, Jakab, Barnavon, Neuhaus, and Mauch-Mani (2005)
	Northern blot	*In vitro* plantlets	Laquitaine et al. (2006)
	RT-qPCR	Calli	Rühmann et al. (2013)
	RT-qPCR	Cell culture	Aziz et al. (2003); Aziz et al. (2004); Aziz et al. (2007); Belhadj et al. (2008); Belhadj, Telef et al. (2008); Lijavetzky et al. (2008); Faurie, Cluzet, Corio-Costet, and Mérillon (2009); Faurie et al. (2009); Varnier et al. (2009); Dubreuil-Maurizi et al. (2010); Qiao et al. (2010); Caillot et al. (2012); Almagro et al. (2014); Lachhab et al. (2014); Pietrowska-Borek, Czekała, Belchí-Navarro, Pedreño, and Guranowski (2014); Almagro, Belchí-Navarro, Martínez-Márquez, Bru, and Pedreño (2015); Claverie et al. (2018); Krzyzaniak et al. (2018); Wang et al. (2022); Sofi et al. (2023)

RT-qPCR	Leaves from foliar cuttings	Belhadj et al. (2008); Trouvelot et al. (2008); Harm, Kassemeyer, Seibicke, and Regner (2011); Boubakri et al. (2012); Boubakri et al. (2013); Boubakri et al. (2013); Saigne-Soulard et al. (2015); Krzyzaniak et al. (2018); Dufour et al. (2013)
RT-qPCR	Leaves from wood cuttings	Belhadj et al. (2006); Slaughter, Hamiduzzaman, Gindro, Neuhaus, and Mauch-Mani (2008); Dufour et al. (2013); Banani et al. (2014); Mijailovic et al. (2022); Zarraonaindia et al. (2023)
RT-qPCR	Leaves collected in the vineyard	Nesler et al. (2015); Singh et al. (2019); Bellée et al. (2018)
RT-qPCR	Leaves of one-year-old potted grapevines grown outside	Pagliarani et al. (2020)
RT-qPCR	Leaves of two-year-old potted grapevines grown in greenhouses	Perazzolli et al. (2011)

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Table 2 Methods to study grapevine defence responses and visualize interaction with pathogens. (*cont'd*)

Defence response	Methodology	Plant material	References
	RT-qPCR	Berries collected in the vineyard	Wang et al. (2015)
	Microarray analysis (GrapeGen Affymetrix GeneChip)	Cell culture	Almagro et al. (2014)
	Microarray analysis (6 different GeneChip® Vitis vinifera Genome Arrays) (Affymetrix)	Cell culture	Zamboni et al. (2009)
	Microarray analysis (Combimatrix Grape Array)	Leaves from foliar cuttings	Gauthier et al. (2014)
	Microarray analysis (GrapeGen Affymetrix GeneChip)	Berries collected in the vineyard	Kelloniemi et al. (2015)
	Microarray analysis (NeoViGen96" and Biostim 96 chips)	Leaves from wood cuttings	Bodin et al. (2020); Burdziej et al. (2021)
	Microarray analysis (NeoViGen96" chip)	Leaves collected in the vineyard	Dufour et al. (2016); Bellée et al. (2018)

		Microarray analysis (NeoViGen96" chip)	Seedling leaves	Demanèche, Mirabel, Abbe, Eberst, and Souche (2020)
		RNA seq	Leaves of one-year-old potted grapevines grown outside	Pagliarani et al. (2020)
		RNA seq	Leaves of two-year old potted grapevines grown in greenhouses	Perazzolli et al. (2012)
		RNA seq	Berries	Li et al. (2021)
	mRNA localization	*in situ* hybridization/light microscopy	Woody tissues	Trouvelot et al. (2023)
Phytoalexins and other antimicrobials	Stilbene phytoalexins and other polyphenols	Autofluorescence/ Fluorescence microscopy	Leaves from foliar cuttings	Slaughter et al. (2008); Trouvelot et al. (2008); Bellow et al. (2012); Boubakri et al. (2012)
		Folin-Ciocalteu method/ Spectrophotometry, UPLC-DAD	Cell culture	Andi, Gholami, Ford, and Maskani (2019)
		Folin-Ciocalteu method/ UHPLC-UV-Vis-DAD coupled to ESI-MS/MS	Leaves collected in the vineyard	Bellée et al. (2018)

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Table 2 Methods to study grapevine defence responses and visualize interaction with pathogens. (*cont'd*)

Defence response	Methodology	Plant material	References
	UPLC/DAD	Cell culture	Lachhab et al. (2014); Krzyzaniak et al. (2018)
	UPLC-fluorimetry	Leaves from wood cuttings	Mijailovic et al. (2022)
	UPLC-MS	Leaves from foliar cuttings	Saigne-Soulard et al. (2015); Krzyzaniak, Trouvelot et al. (2018)
	HPLC—UV/DAD/ fluorimetry detection	Calli	Rühmann et al. (2013)
	HPLC—UV/DAD/ fluorimetry detection	Cell culture	Aziz et al. (2003); Poinssot et al. (2003); Bru, Sellés, Casado-Vela, Belchí-Navarro, and Pedreño (2006); Belhadj et al. (2008); Faurie et al. (2009); Faurie et al. (2009); Belchí-Navarro et al. (2013); Almagro et al. (2014); Pietrowska-Borek et al. (2014); Almagro et al. (2015); Claverie et al. (2018); Krzyzaniak, Negrel et al. (2018); Andi et al. (2019); Sák et al. (2021)
	HPLC—UV/DAD/ fluorimetry detection	Leaves from *in vitro* plantlets	Aziz et al. (2006); Laquitaine et al. (2006); Rühmann et al. (2013)

HPLC—UV/DAD/ fluorimetry detection	Leaves from foliar cuttings	Boubakri et al. (2013); Dufour et al. (2013); Saigne-Soulard et al. (2015); Paris et al. (2019); Burdziej et al. (2021); Zarraonaindia et al. (2023)
HPLC—UV/DAD/ fluorimetry detection	leaves from wood cuttings	Aziz et al. (2006); Belhadj et al. (2006); Belhadj et al. (2008); Dufour et al. (2013); Burdziej et al. (2021)
HPLC—UV/DAD/ fluorimetry detection	leaves of one-year-old grapevines grown in an open-air system	Pagliarani et al. (2020)
HPLC- UV/DAD/ fluorimetry detection	leaves collected in the vineyard	Garde-Cerdán et al. (2016); Gil-Muñoz et al. (2017)
HPLC- UV/DAD/ fluorimetry detection	berries	Wang et al. (2015); Li et al. (2021)
HPLC-UV-DAD/ESI-MS	cell culture	Lijavetzky et al. (2008); Martinez-Esteso, Sellés-Marchart, Vera-Urbina, Pedreño, and Bru-Martinez (2009); Santamaria, Mulinacci, Valletta, Innocenti, and Pasqua (2011)
LC-MS	leaves collected in the vineyard	Belchí-Navarro et al. (2013); Bellée et al. (2018)

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Table 2 Methods to study grapevine defence responses and visualize interaction with pathogens. (*cont'd*)

	Defence response	Methodology	Plant material	References
		HPLC-DAD/ESI-MS	berries	Ruiz-García et al. (2012); Miliordos et al. (2022)
		^{13}C NMR and LC-MS	hairy root culture	Tisserant et al. (2016)
		MALDI-TOF Mass Spectrometry Imaging and, LC-ESI/MS and MS/MS (for control with UV-C treated leaves)	leaves from foliar cuttings	Becker, Carré, Poutaraud, Merdinoglu, and Chaimbault, (2014)
Other defence-related compounds	Volatile Organic compounds (VOC)	GC MS, Enantio-MDGC-MS	foliar cuttings	Hampel et al. (2005); Chalal et al. (2015); Lemaitre-Guillier et al. (2021)
			vines in the field	Lemaitre-Guillier et al. (2022)

Metabolomic	Metabolites	GC-MS	cell culture	Krzyzaniak, Negrel et al. (2018)
		FT-ICR-MS	leaves from foliar cuttings	Adrian et al. (2017)
		^{1}H NMR	leaves from wood cuttings	Burdziej et al. (2019)
PR proteins	Chitinase activity	Carboxymethyl-chitin-remazol-brilliant violet as substrate/spectrophotomery	cell culture	Aziz et al. (2003); Aziz et al. (2004); Aziz et al. (2007)
		Carboxymethyl-chitin-remazol-brilliant violet as substrate/spectrophotomery	leaves from *in vitro* plantlets	Aziz et al. (2006)
		Carboxymethyl-chitin-remazol-brilliant violet as substrate/spectrophotomery	leaves collected in the field	Magnin-Robert, Trotel-Aziz, Quantinet, Biagianti, and Aziz (2007); Magnin-Robert, Quantinet, Couderchet, Aziz, and Trotel-Aziz (2013)
		Carboxymethyl-chitin-remazol-brilliant violet as substrate/spectrophotomery	berries collected in the field	Magnin-Robert et al. (2007); Magnin-Robert et al. (2013)

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Table 2 Methods to study grapevine defence responses and visualize interaction with pathogens. (*cont'd*)

Defence response	Methodology	Plant material	References
Peroxidase activity	4-methoxy- R-naphthol (4MN) and H_2O_2 as substrates/ spectrophotometry	cell culture	Bru et al. (2006)
	ABTS (2,2'-azino-bis-(3-ethylbenzthiazoline)-6-sulfonic acid/ spectrophotometer	leaves from foliar cuttings	Harm et al. (2011)
	pyrogallol/ spectrophotometry)	leaves collected in the field	Farouk, Belal, and El-Sharkawy (2017)
Catalase activity	H_2O_2/spectrophotometry	leaves collected in the field	Farouk et al. (2017)
Peroxidase localization	Benzidineor TMB as substrate/light microscopy	leaves from *in vitro* plants or cuttings	Kortekamp, Wind, and Zyprian (1998); Kortekamp and Zyprian (2003); Toffolatti, Venturini, Maffi, and Vercesi (2012)
Beta 1,3 glucanase activity	Laminarin as substrate/ spectrophotometrty	cell culture	Aziz et al. (2003); Aziz et al. (2004); Aziz et al. (2007)
	Laminarin as substrate/ spectrophotometrty	leaves from wood cuttings	Harm et al. (2011)

		Carboxymethyl-curdlan-remazol brilliant blue as substrate/spectrophotomery	leaves from *in vitro* plantlets	Aziz et al. (2006); Aziz et al. (2007)
		Carboxymethyl-curdlan-remazol brilliant blue as substrate/spectrophotomery	leaves collected in the field	Magnin-Robert et al. (2007); Magnin-Robert et al. (2013)
		Carboxymethyl-curdlan-remazol brilliant blue as substrate/spectrophotomery	berries collected in the field	Magnin-Robert et al. (2007); Magnin-Robert et al. (2013)
	Osmotin and thaumatin detection	Western-blot	Leaves from wood cuttings	Monteiro et al. (2003)
		Western-blot	Berries collected in the field	Monteiro et al. (2003)
Other proteins	Activity of enzymes of phenylpropanoid pathway	Phenylalanine-UL-14C, TLC and autoradiography (PAL)	Calli	Rühmann et al. (2013)
		l 2–14C-malonyl-CoA, TLC and autoradiography (CHS and STS)	Calli	Rühmann et al. (2013)

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Table 2 Methods to study grapevine defence responses and visualize interaction with pathogens. (*cont'd*)

	Defence response	Methodology	Plant material	References
	polyphenol oxidase	Catechol/ spectrophotometry	Leaves from wood cuttings	Harm et al. (2011)
		Spectrophotometry	Leaves collected in the field	Farouk et al. (2017)
	superoxide dismutase, ascorbate peroxidase, catalase, glutathione reductase	Spectrophotometry	Leaves from wood cuttings	Zarraonaindia et al. (2023)
Proteomic	proteins	2DE/identification by PMF MALDI-TOF analysis and MS/MS	cell culture	Martinez-Esteso et al. (2009)
		2DE/identification by Nano LC-MS/MS	leaves from foliar cuttings	Lemaître-Guillier et al. (2017)
Cell wall strengthening/ callose deposits	Cytoskeleton reorganization	Immunofluorescence (microtubules)	Cell culture	Qiao et al. (2010)
		FITC–phalloidin and confocal laser scanning microscopy (actin)	Cell culture	Qiao et al. (2010)

	FABD2-GFP marker (Actin)/spinning disk confocal microscopy	FABD2-GFP expressing cell culture	Sofi et al. (2023)
	GFP-AtTUB6 marker (tubulin)/spinning disk confocal microscopy	GFP-AtTUB6 expressing cell culture	Guan et al. (2015)
	Western blot (alpha-tubulin)	Cell culture	Qiao et al. (2010)
	GFP-tagged actin marker spinning disc confocal microscopy	GFP-AtFABD2 expressing cell culture	Wang et al. (2022)
Topography and elasticity	Atomic force microscopy	Cell culture	Lesniewska, Adrian, Klinguer, and Pugin (2004)
Cell wall impregnations (silica, lignin, …)	Methyl red/cristal violet staining/phloroglucinol light microscopy	Leaves	Blaich and Grundhöfer (1998); Hamiduzzaman et al. (2005)
Callose deposition	Aniline blue staining/ epifluorescence microscopy	Leaves	Gindro et al. (2003); Hamiduzzaman et al. (2005); Trouvelot et al. (2008); Boubakri et al. (2012); Boubakri et al. (2013); Gauthier et al. (2014); Claverie et al. (2018); Paris et al. (2019)

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Table 2 Methods to study grapevine defence responses and visualize interaction with pathogens. (*cont'd*)

	Defence response	Methodology	Plant material	References
		Scanning electron microscopy	Leaves of susceptible *vs* resistant grapevine	Gindro et al. (2003); Trouvelot et al. (2008); Alonso-Villaverde et al. (2011)
Organ surface	Epicuticular waxes	Environmental and scanning electron microscopy	Leaves of susceptible *vs* resistant grapevine	Viret et al. (2018)
	Leaf hairs	Environmental and scanning electron microscopy	Leaves of susceptible *vs* resistant grapevine	Kortekamp and Zyprian (1999)
	Stomata size and density	Light microscopy	Leaves of susceptible *vs* resistant grapevine	Mouafo-Tchinda, Fall, Beaulieu, and Carisse (2022)

Pathogen interactions with the host plant (at the cellular level)	Fungal restriction/ alteration during colonization or sporulation	Staining (aniline blue, trypan blue, toluidine blue)/light or epifluorescence microscopy	Leaves	Cohen, Reuveni, and Baider (1999); Hamiduzzaman et al. (2005); Hoffmann et al. (2008); Slaughter et al. (2008); Trouvelot et al. (2008); Dubreuil-Maurizi et al. (2010); Boubakri et al. (2012); Toffolatti et al. (2012); Boubakri et al. (2013); Martinez-Prada and Kortekamp (2015); Krzyzaniak et al. (2018); Hu et al. (2019)
			Berries	Kelloniemi et al. (2015)
		Scanning electron microscopy	Leaves	Crisp, Wicks, Troup, and Scott (2006); Ma et al. (2018)
			Berries	Kelloniemi et al. (2015)
		Transmission electron microscopy	Leaves	Musetti et al. (2006); Hu et al. (2019)
	Occlusion papillae at the infection site	Staining (toluidine blue)/ light microscopy	Berries	Kelloniemi et al. (2015)
		Scanning electron microscopy	Leaves of susceptible *vs* resistant grapevine	Alonso-Villaverde et al. (2011)

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Table 2 Methods to study grapevine defence responses and visualize interaction with pathogens. (*cont'd*)

	Defence response	Methodology	Plant material	References
	Localized cell death/ hypersensitive response	Staining (trypan blue)/light microscopy	Leaves	Langcake and Lovell (1980)
		Scanning electron microscopy	Leaves of susceptible *vs* resistant grapevine	Alonso-Villaverde et al. (2011)
		Transmission electron microscopy	Leaves	Langcake and Lovell (1980); Trouvelot et al. (2008); Steimetz et al. (2012)
			Berries	Kelloniemi et al. (2015)
	Tylosis formation in the xylem lumen	Scanning electron microscopy	Woody tissues	del Río, Gutiérrez, and Martínez (2004)

protein kinases (MAPKs). Finally, expressions of defence-related genes are thus activated, leading to defence responses *i.e.* the synthesis of phytoalexins, other phenolic compounds and pathogenesis-related (PR) proteins, cell wall reinforcement and, in some cases, the hypersensitive response (HR; for review, see Adrian et al., 2012). Additionally, evolution of the cell cytoskeleton during signalling (Qiao, Chang, & Nick, 2010; Guan, Buchholz, & Nick, 2015; Wang, Duan et al., 2022; Sofi, Metzger, Riemann, & Nick, 2023) and emission of volatile organic compounds (Hampel, Mosandl, & Wüst, 2005; Chalal et al., 2015; Lemaitre-Guillier et al., 2021; Lemaitre-Guillier et al., 2022) were also reported.

As highlighted by Table 2, various methodologies allow the study of grapevine defences. They include biochemistry methods (such as luminol chemiluminescence for H_2O_2, western blot for MAPK or spectrophotometry for PR proteins activities), biotechnologies (such as cells expressing aequorin for free cytosolic calcium detection), molecular biology (gene expression), chromatography (for phytoalexins and other phenolic compounds analysis), or microscopy (such as fluorescence microscopy for visualization of callose). Omics approaches (transcriptomic, proteomic, metabolomic) are also used to have an overview of the impact of defence activation on the global plant metabolism (Gauthier et al., 2014; Adrian et al., 2017; Lemaître-Guillier et al., 2017; Burdziej et al., 2019; Bodin, Bellée, Dufour, André, & Corio-Costet, 2020). The diversity of these methods makes it possible to select those best suited to the objectives of the experiments to be carried out, the plant material available, the nature and quantity of the elicitors to be tested or the type of the microbial interaction studied.

2.1 Perception of the plant pathogens

As described above for *A. thaliana*, grapevine (*V. vinifera*) has a repertoire of pattern recognition receptors (PRRs) mostly with an LRR domain *i.e.* 240 LRR-RLKs and 115 LRR-RLPs (Ngou et al., 2022). Up to now, very few of these PRRs have been characterized. The first one identified was the Flagellin Sensing 2 ortholog VvFLS2 which differentially perceives the flagellin epitopes flg22 coming from pathogenic or plant growth-promoting bacteria (Trdá et al., 2014). Grapevine also shows 16 LysM-RLKs including VvLYK1–1, VvLYK1–2 and VvLYK1–3 that are the putative orthologues of the *A. thaliana* chitin receptor AtCERK1/LYK1 (Brulé et al., 2019). The functional complementation of the *A. thaliana atcerk1* mutant was achieved by the constitutive expression of *VvLYK1–1* or the inducible expression of *VvLYK1–2* to restore chitooligosaccharide-induced

immune responses (Brulé et al., 2019). Moreover, *VvLYK1–1* expression in *atcerk1* also restored non-host resistance against *E. necator* (PM). These results demonstrated that VvLYK1–1 and VvLYK1–2 are involved in chitooligosaccharide-triggered immunity in grapevine (Brulé et al., 2019). More recently, it has been shown that the grapevine receptor VvLYK5–1 can complement the *A. thaliana* double mutant *atlyk4/5* and interact with VvLYK1–1, only after chitin perception (Roudaire et al., 2023). Altogether, these results indicate that two independent receptors in grapevine (VvLYK1–1 and VvLYK5–1) form a functional heterodimeric receptor complex for chitin perception, and play a role in the resistance to cell penetration by *E. necator* (Brulé et al., 2019; Roudaire et al., 2023).

2.2 Defences in susceptible varieties

The following paragraphs illustrate, in a non-exhaustive way, some defence responses observed during the interaction between susceptible *V. vinifera* varieties (cultivars) and their main fungal aggressors.

In *P. viticola* infected leaves of Riesling expressing oil spot symptoms, genes encoding cytoskeletal components, enzymes of the phenylpropanoid and *beta*-oxidation pathways, and PR proteins were upregulated, although weakly, compared to mock-inoculated ones (Polesani et al., 2008). Conversely, Dufour, Lambert, Bouscaut, Mérillon, and Corio-Costet (2013) reported mostly a down-regulation of defence genes in *P. viticola* and *E. necator* infected leaves of Cabernet Sauvignon at 24 h post-inoculation (hpi) and suggested a manipulation of the host response by the pathogens. Similarly as Dufour et al. (2013), Burdziej et al. (2021) noted that in *P. viticola* inoculated leaves, the majority of the studied defence-related genes were down-regulated at 48 hpi, particularly those coding for PR proteins and enzymes involved in phenylpropanoid biosynthesis. The differences in the results obtained by Polesani et al. (2008) and Dufour et al. (2013) may be explained by the different infection time points that were investigated (at the end and at the beginning, respectively). In this respect, Milli et al. (2012) conducted a proteomic analysis of Pinot noir *in vitro* plantlets infected by *P. viticola* and reported a time dependent modulation of proteins during the three dpi. They suggested a transient suppression of the host defence response at two dpi by the pathogen to facilitate mycelial development. Owing to their fluorescence, stilbenes (as *trans*-resveratrol, a grapevine phytoalexin) can be quite easily localized. They were mainly observed in cell walls of stomata guard cells, periclinal cell walls and vacuoles of Cabernet Sauvignon leaves infected by DM (Bellow, Latouche, Brown, Poutaraud, & Cerovic, 2012).

For PM, transcriptome profiling of Cabernet Sauvignon leaves infected by *E. necator* revealed the up-regulation of defences genes, among which some encoding PR-proteins (PR-1, PR-10), ROS metabolism, MAPK signalling, transcriptional regulation, stilbene synthesis and cell wall modifications (Fung et al., 2008). Quantitative proteomic analysis confirmed a higher abundance of PR-proteins (PR-10 and others) (Marsh et al., 2010). More specifically, the expressions of PR-protein genes *PR3* (chitinase) and *PR10* (ribonuclease) were up-regulated in Cabernet Sauvignon 48 hpi (Dufour et al., 2013). Likewise, the expression of the PR-proteins *PR-1* and *PR-5* (thaumatin/osmotin like protein) was induced in Moscatel leaves infected by *E. necator*, and also in that infected by *Diaporthe ampelina* (formerly *Phomopsis viticola;* Monteiro, Barakat, Piçarra-Pereira, Teixeira, & Ferreira, 2003). In berries, it was only induced in response to *E. necator* infection (Monteiro et al., 2003). These defence PR proteins undoubtedly play a part in the plant's basic defence and ontogenic resistance, but they are either insufficient or their genes induced too late to enable a Cabernet Sauvignon, for example, to resist infection by PM.

Renault, Deloire, and Berne (1996) reported the induction of PR-proteins (PR-2) in Chardonnay leaves infected by *B. cinerea*. Later, proteomic and transcriptomic analyses of Gamay cell cultures subjected to *B. cinerea* highlighted an induction of the synthesis of PR-proteins, and also of proteins involved in cell wall modifications (Dadakova et al., 2015). An upregulation of defence related gene expression (genes coding for phenylalanine ammonia lyase, stilbene synthase, an acidic and a basic chitinases, and a polygalacturonase inhibiting protein) was observed in leaves of *in vitro* Chardonnay plantlets (Bezier, Lambert, & Baillieul, 2002). Transcriptomic and metabolomic analyses of Trincadeira berries infected by *B. cinerea* performed at two developmental stages (peppercorn-sized berries and veraison) showed activation of defences, among which stilbenes and PR proteins. They concluded about a reprogramming of primary metabolism (carbohydrates and lipids) towards plant defence and pathogen nutrition (Agudelo-Romero et al., 2015).

Defence responses were also studied in different organs of grapevines infected by GTDs. Stilbenes (including *trans*-resveratrol) and other phenolics indeed accumulate in the symptomatic area of the wood of the trunk of esca-infected grapevines (Amalfitano et al., 2000). Modulation of the abundance of defence proteins was observed in the black streaked wood of the trunk of apoplectic and esca proper affected Chardonnay vines (Magnin-Robert et al., 2014), and also in their green stems (Spagnolo et al., 2012).

In leaves of esca-infected Chardonnay vines, the induction of the expression of several defence-related genes (associated to stilbenes and PR-proteins synthesis) occurs seven days before and at time of the occurrence of the apoplexy (Letousey et al., 2010). Pierron et al. (2016) observed the induction of defence genes in woody cuttings of Cabernet Sauvignon inoculated with *Phaeoacremonium minimum* and *Phaeomoniella chlamydospora* (fungi associated to Esca disease), close to the wound site of inoculation. Among these genes were those coding for PAL and STS, and also PR-5 and PR10.3. Proteomic and metabolomic analyses revealed a higher abundance of stilbenes and defence proteins (including PR-2, PR-5, PR-17 proteins) in the symptomatic area (brown stripe) of the trunk of vines expressing Botryosphaeria dieback, than in the asymptomic area (Spagnolo et al., 2014; Lemaitre-Guillier et al., 2020). Stilbenes were found in the wood of Cabernet Sauvignon cuttings infected by *Neofusicoccum parvum* (Massonnet et al., 2017) and in the green stems of Tempranillo cuttings infected by *N. parvum* and *Diplodia seriata* (Reis et al., 2016). After inoculation of different species of Botryosphaeriaceae (*N. parvum, D. seriata, Diplodia mutila, Lasiodiplodia viticola*) in the wood of different varieties (Merlot, Cabernet Sauvignon, Ugni Blanc), a large number of PR genes (*PR1*, *PR2*, *PR6*, *PR5*, *PR10*) and *STS* gene were over-expressed, with a variability in the responses linked to the genetic background of the different tested varieties and to the aggressiveness of the pathogens (Bellée et al., 2017). It was further reported that metabolites secreted by the responsible fungi (*N. parvum* and *D. seriata*) can induce phytoalexin synthesis and other defence responses (Abou-Mansour et al., 2015; Stempien et al., 2018; Trotel-Aziz et al., 2019).

These examples illustrate the capacity of grapevine to activate defences in response to microbial attacks. However, these defences are not always effective in preventing the pathogen survival, progression and subsequent disease expression. There are several reasons for this. Firstly, the microorganism may have developed strategies, mainly the production of effectors, to stop plant defence responses (Fig. 1, part 1.1). Secondly, the microorganism can metabolize and detoxify active defence metabolites (*e.g. B. cinerea* metabolize some stilbenes *via* laccase-type polyphenol oxidases). Finally, not all vines have the same genetic capacity to defend themselves. This point will be developed further below (part 2.4).

2.3 Elicitor-induced resistance

The term "elicitor" was originally used to define a compound that induces accumulation of phytoalexins in plants (Ebel & Cosio, 1994). It was next

applied to any agent capable of stimulating any type of plant defence response (Nürnberger, 1999). As elicitors have been used as a strategy of crop protection, they were also defined as plant resistance inducers (Walters, Walsh, Newton, & Lyon, 2005). Elicitors are able to induce plant defence which may or may not result in resistance against pathogens. The so-called "Induced resistance" (Fig. 1) is actually a state of enhanced defensive capacity that can mimic genetic resistance (ETI, Fig. 2, Parts 2.4 and 2.6). Nevertheless, this induction is not always sufficient to control the development of the pathogen.

Various types of defence elicitors were reported as active in grapevine (Adrian et al., 2012; Delaunois et al., 2014), either endogenous (mainly DAMPs such as oligogalacturonides released from plant-cell wall by microbe hydrolytic enzymes), or exogenous (such as chemicals). As shown in Table 3,

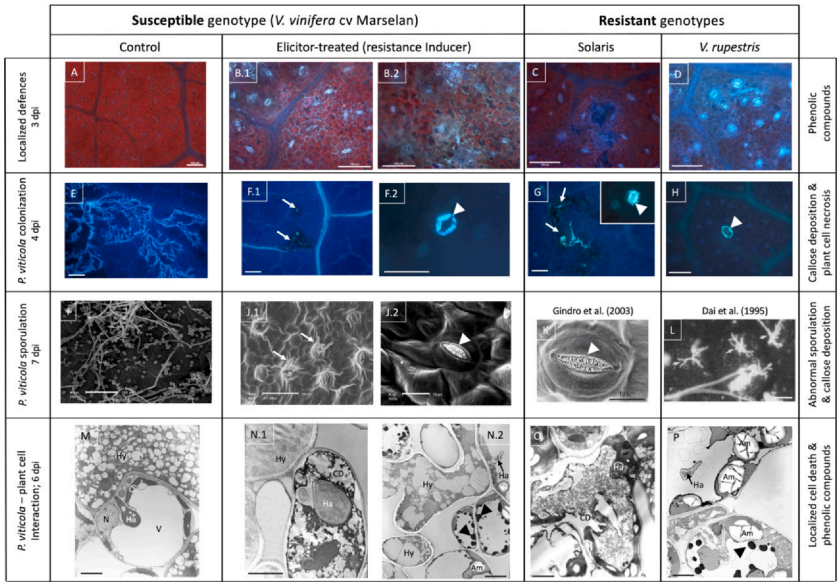

Fig. 2 Illustration of phenotypes and defence reactions expressed in a similar way for resistance to grapevine downy mildew in the case of genetically resistant (C, D, G, H K, L, O and P), or susceptible and elicitor-treated (B, F, J and N) genotypes, respectively. Pathogen colonization and/or plant defences were observed by autofluorescence under UV light (A-D), after aniline blue staining (E-H and L), by scanning electron microscopy (I-K) or by transmission electron microscopy (M-P). Bars represent 100µm (A-F.1 and G-I) 50 µm (F.2, J.1 and L), 10 mm (J.2 and K) or 5 µm(M-P). Arrows represent plant cell necrosis (F.1, and G) or aborted sporangiophores (J.1 and L); white arrow heads represent callose deposition in stomata (F.2, G-encarta, H, J.2 and K) and black arrow heads point out phenolic compounds in plant cells (N.2 and P). Am: amyloplast, CD: cell death, Ch: chloroplast, Ha: haustorium, Hy: hyphae, N: nucleus and V: vacuole.

Table 3 Elicitor-induced resistance against grapevine pathogens.

Category	Nature	Pathogens affected by elicitor-induced resistance	References
Amino acids	DL-β-aminobutyric acid (BABA)	*P. viticola, B. cinerea*	Cohen et al. (1999); Reuveni, Zahavi, and Cohen (2001); Hamiduzzaman et al. (2005); Slaughter et al. (2008); Dubreuil-Maurizi et al. (2010); Harm et al. (2011); Tamm et al. (2011); Li et al. (2021)
	Methionine	*P. viticola*	Boubakri et al. (2013)
Carbohydrates	Beta-1,4 cellodextrins	*B. cinerea*	Aziz et al. (2007)
	Chitin	*B. cinerea, P. viticola*	Brulé et al. (2019)
	Chitosan	*B. cinerea, E. necator, P. viticola*	Aziz et al. (2006); Trotel-Aziz, Couderchet, Vernet, and Aziz (2006); Iriti et al. (2011); Farouk et al. (2017); Brulé et al. (2019)
	COS-OGA (complex of oligochitosans and oligopectates)	*E. necator*	van Aubel et al. (2014)
	Cyclodextrins, modified cyclodextrins	*B. cinerea*	Bru et al. (2006)
	Laminarin	*B. cinerea, P. viticola, E. necator*	Aziz et al. (2003); Pagliarani et al. (2020)
	Laminaran derivatives (acylation, lauroylation, sulfation)	*P. viticola*	Paris et al. (2019)

	Phycarine (sulfated laminarin) or PS3	*P. viticola*	Trouvelot et al. (2008); Gauthier et al. (2014)
	Oligogalacturonides	*B. cinerea, P. viticola*	Aziz et al. (2004); Aziz et al. (2007); Allègre et al. (2009)
	Tagatose	*P. viticola*	Mijailovic et al. (2022)
	Xyloglucan	*B. cinerea*	Claverie et al. (2018)
Lipids/lipo-peptides	Ergosterol	*B. cinerea*	Laquitaine et al. (2006)
	Linoleic acid	*P. viticola*	Harm et al. (2011)
	Rhamnolipids	*B. cinerea*	Varnier et al. (2009)
Vitamins	Riboflavin (B2 vitamin)	*P. viticola*	Boubakri et al. (2013)
	Thiamine (B1 vitamin)	*P. viticola*	Boubakri et al. (2012)
Phosphonates	Phosphonates and fosetyl Al	*P. viticola, E. necator, Esca expression*	Dercks and Creasy (1989); Di Marco et al. (2011); Pinto, do Nascimento, de Souza Gomes, da Silva, and dos Reis Miranda (2012); Dufour et al. (2016); Pagliarani et al. (2020); Burdziej et al. (2021); Burdziej et al. (2019)
Hormones/ hormone-like	Acibenzolar-S-methyl/ benzothiadiazole (BTH)	*P. viticola, B. cinerea, E. necator*	Campbell and Latorre (2004); Iriti et al. (2004); Laquitaine et al. (2006); Perazzolli et al. (2008); Harm et al. (2011); Dufour et al. (2013); Banani et al. (2014); Farouk et al. (2017); Bellée et al. (2018); Bodin et al. (2020); Pagliarani et al. (2020); Burdziej et al. (2021)

(*continued*)

Table 3 Elicitor-induced resistance against grapevine pathogens. (*cont'd*)

Category	Nature	Pathogens affected by elicitor-induced resistance	References
	Methyl jasmonate	*E. necator, P. viticola, B. cinerea*	Belhadj et al. (2006); Faurie et al. (2009); Wang et al. (2015); Belchí-Navarro et al. (2019); Burdziej et al. (2021)
	Ethephon	*E. necator*	Belhadj et al. (2008); Faurie et al. (2009)
Specific chemical	Silicon	*P. viticola*	Farouk et al. (2017)
Culture filtrates	*Aureobasidium pullulans*	*B. cinerea*	Rühmann et al. (2013)
	Bacillus subtilis GLB191-S	*P. viticola*	Li et al. (2019)
	Botrytis cinerea	*P. viticola, E. necator*	Saigne-Soulard et al. (2015)
Algae/plant extracts	Soybean protein hydrolysate	*P. viticola*	Lachhab et al. (2014)
	Grapevine extracts	*P. viticola, B. cinerea*	Gabaston et al. (2017); De Bona et al. (2019)
	Various plant extracts (*i.e. Solidago canadensis*)	*P. viticola*	Harm et al. (2011)
	A monocotyledon plant extract	*P. viticola*	Krzyzaniak et al. (2018)
	Ulvan extracts	*P. viticola*	Jaulneau et al. (2011)

Other biological extracts/ lysate	Casein hydrolysate	*P. viticola*	Lachhab et al. (2014)
	Cell wall fragments of yeast cerevisiane)	*P. viticola*	De Miccolis Angelini et al. (2019)
	Lysate of amoeba *Willaertia magna* C2c Maky	*P. viticola*	Demanèche et al. (2020)
	Mycelium extract of *Penicillium chrysogenum*	*P. viticola*	Harm et al. (2011); Tamm et al. (2011)
	Extract of *Penicillium chrysogenum*	*P. viticola*	Harm et al. (2011)
	Protein-based composition, nutrient broth (meat and yeast extract)	*E. necator*	Nesler et al. (2015)
	Unsaturated oligoglucuronans (obtained from glucuronans secreted by *Rhizobium meliloti*, M5N1CS strain)	*B. cinerea*	Caillot et al. (2012)

they are of very different origins (plant or microbial extracts, synthetic chemicals, *etc.*), nature (carbohydrates, lipids, peptides, etc), and complexity (purified molecules or extracts). Despite this wide diversity, a rather limited number of them induces efficient resistance against pathogens, especially in field conditions (Table 3). As examples, the sulfated laminarin induces resistance against *P. viticola* (Gauthier et al., 2014), and COS-OGA (oligochitosans/oligopectates complex) induces resistance against *E. necator* (van Aubel, Buonatesta, & Van Cutsem, 2014). Moreover, few of them induce resistance against different pathogens, as BTH do (Busam, Kassemeyer, & Matern, 1997; Iriti, Rossoni, Borgo, & Faoro, 2004; Perazzolli, Roatti, Bozza, & Pertot, 2011; Dufour et al., 2013; Bellée, Cluzet, Dufour, Mérillon, & Corio-Costet, 2018; Bodin et al., 2020; Burdziej et al., 2021; Jiang et al., 2022). Conversely, they often induce resistance to one pathogen but not to others. This lack of "genericity" may be due to the different lifestyles of pathogens (external *versus* internal, biotrophic *versus* necrotrophic, *etc*), and also to the fact that some of them also have direct antimicrobial effect, as reported for a plant extract (Krzyzaniak, Trouvelot et al., 2018). Interestingly, elicitors that directly interfere with the hormonal pathways involved in defence *e.g.* the salicylic acid (SA) and jasmonate (JA) pathways appear usually to be more effective than other ones. This is the case of acibenzolar-S-methyl or BTH (a SA analogue) and methyl jasmonate (a JA derivative), which are effective in vineyards against a range of pathogens (Campbell & Latorre, 2004; Iriti et al., 2004; Belhadj et al., 2006; Dufour, Magnin, Dumas, Vergnes, & Corio-Costet, 2016; Bellée et al., 2018; Burdziej et al., 2021). Several studies have also reported defence activation by physical elicitors. Irradiation by UV-C indeed stimulates the expression of defence genes and phytoalexin synthesis (Douillet-Breuil, Jeandet, Adrian, & Bessis, 1999; Bonomelli et al., 2004). Moreover, application of UV-C flashes to grapevines by specific devices can limit PM in vineyards (Ledermann, Daouda, Gouttesoulard, Aarrouf, & Urban, 2021), although the absence of effects on grapevine microbiote has to be checked.

Elicitor-induced resistance is currently developed as a strategy to reduce the use of fungicides for vineyard protection, especially against DM and PM. However, the effectiveness of this strategy remains variable. Research is still ongoing to identify highly effective elicitors. It is well known that defence activation doesn't systematically lead to induced resistance against pathogens. Conversely to fungicides that directly target the pathogen, elicitors act *via* activation of grapevine defence responses, the level of which depends on the genetic background and the physiological status of

the plant. In connection, we have observed that the use of biostimulants could be a lever to increase elicitor-induced resistance *via* their effects on plant physiology (Bodin et al., 2020; Jacquens et al., 2022; Jindo et al., 2022). Numerous factors are likely to impact elicitor efficiency, especially abiotic stresses (as water stress or heat stress), nutrition, cultural practices, *etc.* In this context, identification of markers of elicitor-induced resistance against diseases would be helpful to identify the optimal periods for elicitor application and to secure the use of elicitors in protection strategies. Studies were conducted to identify such markers (Adrian et al., 2017; Lemaître-Guillier et al., 2017). Erythritol phosphate was identified as a candidate for sulfated laminarin-induced resistance to DM (Adrian et al., 2017). Its putative role of induced-resistance marker needs to be confirmed with other elicitors and for other grapevine/pathogen interactions. Multidisciplinary approaches are now making it possible to gain a better understanding of the role of elicitors on gene expressions or metabolomes and to find specific markers of efficient elicitors (Dufour et al., 2016; Burdziej et al., 2019; Bodin et al., 2020).

2.4 Genetic resistance

The wild *V. vinifera* subsp. *sylvestris* is considered to be the progenitor of *V. vinifera* subsp. *vinifera*. The grapevine domestication process took place in a large region between the Black See and Central Asia during the Holocene period (*ca* 11,000 BC). The spread of domesticated *V. vinifera* subsp. *vinifera* into new European environments is associated with cross fertilization with local Western European's wild populations due to human selection or accidental crosses. Genes flows between the two populations occurred allowing local adaptation to water stress or diseases (Grassi & De Lorenzis, 2021; Magris et al., 2021; Dong et al., 2023). Today, they are 8000–10,000 domesticated varieties of vine, table grapes and raisin, with a range of colours from black to white (McGovern et al., 2017). They are well adapted to a wide range of environments of temperate regions.

Taxonomically, the genus *Vitis* can be divided into two subgenera based in chromosome number, *Muscadinia* ($2n = 40$) and *Vitis* ($2n = 38$). The genus *Vitis* has about 70 species spread across the Northern Hemisphere. Two major centres of origin generated about 30 species in North America (Clade I) and about 40 species in Eurasia (including *V. vinifera*, Clade II). The two clades diverged between the middle-late Miocene and the late Pliocene (Liu, Ickert-Bond et al., 2016; Zecca et al., 2020). *Vitis* species are interfertile but most of them remain distinct due to various flowering dates or habitats and to the presence of geographical barriers. However, where species range overlap,

natural hybridizations and introgressions have been a central evolutionary process (Zecca, Labra, & Grassi, 2019). In North America, *Vitis* species are present in South-West, Central and East as well as in Mexico and South Canada (Klein et al., 2018). They are organized around two clades or groups (Klein et al., 2018; Nie et al., 2023; Péros et al., 2023). Clade or group I includes species from the Eastern region as *V. aestivalis; V. cinerea*; *V. cordifolia*, *V. labrusca* and *V. candicans* whereas clade or group II gathered *V. arizonica*, *V. rupestris*, and *V. riparia*. A large diversity has been observed inside *Vitis* species *ca. V. aestivalis* , *V. cinerea* and *V. riparia* (Péros et al., 2021).

Many wild grapes endemic to North America and China display significant levels of resistance to the pathogens. This offers the opportunity to generate by crossings, numerous hybrids and by a process of backcrosses with *V. vinifera* varieties, to produce new resistant grape varieties. Around 1800, in the North America, the European settlers started to plant *V. vinifera* varieties without success because local diseases devasted them. They also cultivated natural hybrids between *Vitis* species with a very low success due to the bad taste of the vines. Then, they started hybridization between native wild resistant species with *V. vinifera*. In a context of breeding, the significant exploitation of *Vitis* species began in the 1850s when the phylloxera louse devastated European vineyards. Hybridization has been perpetuated throughout Europe ever since (Alleweldt & Possingham, 1988; Migicovsky et al., 2016; Merdinoglu, Schneider, Prado, Wiedemann-Merdinoglu, & Mestre, 2018; Yobrégat, 2018; Töpfer & Trapp, 2022; Vezzulli et al., 2022). During this long process, breeders began to introduce resistance against mildew diseases into *V. vinifera* varieties from North American species and more later, from Asian species. It took more than 100 years to develop resistant varieties combining quality of European wine grape and resistance conferred by *Vitis* species. The evaluation of *Vitis* species collections was mainly carried out in the vineyard, under natural infection conditions, and rarely under controlled laboratory conditions, using pathogen strains characterized for their virulence. The number of accessions evaluated and the method of evaluation also fluctuated, according to the studies. For DM and PM, whatever the geographical origin (North America, Asia), some *Vitis* species are more resistant than others and all the *Vitis* species have both resistant and susceptible accessions (Dai, Andary, Mondolot-Cosson, & Boubals, 1995; Denzer, Staudt, & Schlosser, 1995; Staudt & Kassemeyer, 1995; Staudt, 1997; Dalbó et al., 2001; Kortekamp & Zyprian, 2003; Unger, Büche, Boso, & Kassemeyer, 2007; Cadle-Davidson, 2008; Cadle-Davidson, Mahanil, Gadoury, Kozma, & Reisch, 2011; Gao et al., 2016; Fayyaz, Tenscher, Viet Nguyen, Qazi, & Walker, 2021; Péros et al., 2021).

As for DM and PM, *V. vinifera* varieties are highly susceptible to Grey mould (GM) and to Black rot (BR). Nevertheless, very few studies focus on GM or BR resistance. Resistance to GM has been identified in some accessions of North American and Chinese *Vitis* species such as *V. aestivalis*, *V. amurensis*, *V. labrusca*, *V. lincecumii* and *Muscadinia rotundifolia* (Gabler, Smilanick, Mansour, Ramming, & Mackey, 2003; Sapkota, Chen, Schreiner, Ge, & Hwang, 2015; Wan et al., 2015; Naegele, 2018). Concerning the BR resistance, surveys of *Vitis* species native of North America have shown a great deal of variability between and inside species. Therefore, *V. cinerea*, *V. rupestris* and *V. riparia* are considered as the most resistant species (Jabco, Nesbitt, & Werner, 1985; Hausmann, Rex, & Töpfer, 2017; Szabó et al., 2023).

Natural sources of disease resistance are normally found in geographic regions where populations of pathogens and host plants co-evolved. However, few resistant *V. vinifera* varieties have been identified in germplams originated from Georgia, Caucasus and Central Asia, such as Mgaloblishvili for DM (Toffolatti et al., 2016) and Dzhandzhal kara, Kishmish vatkana and Shavtsitska for PM (Hoffmann et al., 2008; Riaz et al., 2013; Possamai et al., 2021). Resistance has been also identified in two wild *V. vinifera* subsp. *sylvestris* accessions in Armenia and Iran (Riaz et al., 2013) and in German wild *sylvestris* (Schröder et al., 2015). One of the German wild *sylvestris* showed also resistance to BR (Schröder et al., 2015).

2.4.1 Resistance loci identification

Some resistance sources have been investigated for the genetic determinism study of pathogen resistance through QTL (Quantitative Trait Loci) analysis. During the last two decades, thirty three *Rpv* loci (Resistance to *P. viticola*) and fifteen *Ren/Run* loci (Resistance to *E. necator,* previously *U. necator*) were identified whereas only three resistance loci to BR, named *Rgb* (Resistance to *G. bidwellii*) and one potential (unnamed) to GM were described (Maul, 2021). These QTL were identified either in vineyard conditions, in greenhouse or in controlled laboratory conditions where the pathogen strains and the plant age are defined (Possamai & Wiedemann-Merdinoglu, 2022). As presented in Tables 4 and 5, the most studied *Vitis* species are North American species such as *V. aestivalis*, *V. cinerea*, *V. rupestris, V. riparia* and *M. rotundifolia.* The most Asian studied species are *V. amurensis* and *V. piazeskii*. The resistance QTL were identified on quite all the chromosomes, but an important concentration is observed on chromosomes 9, 12, 14 and 18. The QTL confer different degrees of resistance range defined as weak, partial, high and total. For DM and PM, the resistance is defined according to

Table 4 Loci of resistance to *P. viticola* (*Rpv*) identified in grapevine. Resistance loci are listed with information about the grapevine accession/species of origin, the linkage group and the putative level of resistance associated with the locus.

Locus	Linkage group	Original species	Resistance type	References
Rpv1	12	*M. rotundifolia*	High	Merdinoglu et al. (2003)
Rpv2	18	*M. rotundifolia*	Total	Wiedemann-Merdinoglu, Prado, Coste, Dumas, and Butterlin (2006)
Rpv3.1	18	*V. rupestris*	Partial	Fischer et al. (2004); Welter et al. (2007); Bellin et al. (2009); van Heerden, Burger, Vermeulen, and Prins (2014); Zyprian et al. (2016); Röckel et al. (2021)
Rpv3.2	18	*V. rupestris or V. lincecumii*	Weak	Zyprian et al. (2016)
Rpv3.3	18	*V. riparia or V. labrusca*	Weak	Vezzulli et al. (2019)
Rpv4	4	MD	Weak	Welter et al. (2007)
Rpv5	9	*V. riparia*	Weak	Marguerit et al. (2009)
Rpv6	12			
Rpv7	7	MD	Weak	Bellin et al. (2009)
Rpv8	14	*V. amurensis*	High	Blasi et al. (2011)

Rpv9	7	*V. riparia*	Weak	Moreira et al. (2011)
Rpv10	9	*V. amurensis*	High	Schwander et al. (2012)
Rpv11	5	MD	Weak	Fischer et al. (2004); Bellin et al. (2009); Schwander et al. (2012)
Rpv12	14	*V. amurensis*	High	Venuti et al. (2013); Frommer et al. (2023)
Rpv13	12	*V. riparia*	Weak	Moreira et al. (2011)
Rpv14	5	*V. cinerea*	Weak	Ochssner et al. (2016)
Rpv17	8	MD	Weak	Divilov, Wiesner-Hanks, Barba, Cadle-Davidson, and Reisch (2017)
Rpv18	11			
Rpv19	14	*V. rupestris*		
Rpv20	6	MD		
Rpv21	7			
Rpv22	2	*V. amurensis*	Weak	Fu et al. (2020)
Rpv23	15			
Rpv24	18			

(*continued*)

Table 4 Loci of resistance to *P. viticola* (*Rpv*) identified in grapevine. Resistance loci are listed with information about the grapevine accession/species of origin, the linkage group and the putative level of resistance associated with the locus. (*cont'd*)

Locus	Linkage group	Original species	Resistance type	References
Rpv25	15	*V. amurensis*	Weak	Lin et al. (2019)
Rpv26	15		Partial	
Rpv27	18	*V. aestivalis*	Partial	Sapkota et al. (2019)
Rpv28	10	*V. rupestris*	Partial	Bhattarai, Fennell, Londo, Coleman, and Kovacs (2021)
Rpv29	14	*V. vinifera*	Weak	Sargolzaei et al. (2020)
Rpv30	3			
Rpv31	16			

Table 5 Loci of resistance to *E. necator* (*Ren* and *Run*), *G. bidwellii* (*Rgb*) and *B. cinerea* (*unnamed*) identified in grapevine. Resistance loci are listed with information about the grapevine accession/species of origin, the linkage group and the putative level of resistance associated with the locus.

Locus	Linkage group	Origin species	Resistance type	References
Ren1	13	*V. vinifera*	Partial	Hoffmann et al. (2008)
Ren1.2	13	*V. vinifera*	Partial	Possamai et al. (2021)
Ren2	14	*V. cinerea*	Partial	Dalbó, Ye, Weeden, Wilcox, and Reisch (2001)
Ren3	15	MD	Partial	Welter et al. (2007); van Heerden et al. (2014); Zyprian et al. (2016); Teh et al. (2017); Zendler, Schneider, Töpfer, and Zyprian (2017)
Ren4	18	*V. romanetii*	Partial	Ramming et al. (2011); Mahanil et al. (2012)
Ren5	14	*V. rotundifolia*	Total	Blanc, Wiedemann-Merdinoglu, Dumas, Mestre, and Merdinoglu (2012)
Ren6	9	*V. piazeskii*	Total	Pap et al. (2016)
Ren7	19	*V. piazeskii*	Partial	Pap et al. (2016)
Ren8	18	MD	Partial	Zyprian et al. (2016)
Ren9	15	MD	Partial	Zendler et al. (2017)
Ren10	2	MD	Partial	Teh et al. (2017); Zendler et al. (2021)

(*continued*)

Table 5 Loci of resistance to *E. necator* (*Ren* and *Run*), *G. bidwellii* (*Rgb*) and *B. cinerea* (*unnamed*) identified in grapevine. Resistance loci are listed with information about the grapevine accession/species of origin, the linkage group and the putative level of resistance associated with the locus. (*cont'd*)

Locus	Linkage group	Origin species	Resistance type	References
Ren11	15	*V. aestivalis*	Partial	Karn et al. (2021)
Ren12	13	*V. amurensis*	Total	Sapkota et al. (2023)
Run1	12	*M. rotundifolia*	Total	Pauquet, Bouquet, This, and Adam-Blondon (2001); Feechan et al. (2013)
Run2	18	*M. rotundifolia*	Partial	Riaz, Tenscher, Ramming, and Walker (2011)
Rgb1	14	*V. cinerea*	Partial	Rex et al. (2014)
Rgb2	14	*V. cinerea*	Weak	Rex et al. (2014)
Rgb3	14	Var. Merzling	Partial	Bettinelli et al. (2023)
Unnamed	2	*V. aestivalis*	MD	Sapkota et al. (2019)

two variables: sporulation and necrosis. For these two pathogens, total resistance is observed when no sporulation occurs and HR reaction is present, it is the case for *Rpv2* (*M. rotundifolia*), *Ren5* (*M. rotundifolia*), *Ren6* (*V. piazeskii*), *Ren12* (*V. amurensis*) and *Run1* (*M. rotundifolia*). Weak, partial and high resistance levels are associated to various levels of sporulation with or without HR reaction. The *Rpv1*, *Run1* and *Rpv3* loci were cloned and defined as TIR-NB-LRR (Feechan et al., 2013; Foria, Magris, Morgante, & Di Gaspero, 2018). Moreover, some *V. vinifera* varieties of Georgia and of the Caucacus region, contain resistance loci such as *Rpv29, Rpv31,* and *Ren1* or *Ren1.2* which have weak or partial effect (Hoffmann et al., 2008; Toffolatti et al., 2018; Possamai et al., 2021).

It should be pointed out that for the last decades, only few *Rpv* (*Rpv1*, *Rpv3*, *Rpv10* and *Rpv12*) and *Ren/Run* loci (*Run1*, *Ren3/Ren9*) were used in breeding programmes and are now, found in new resistant varieties (Zini et al., 2019; Töpfer & Trapp, 2022; Vezzulli et al., 2022).

Some resistant varieties for which the content of resistance loci is known have been analysed and compared for the symptomatology and defences mechanisms with strains collected on *V. vinifera* varieties (naïve strains) to define the effect of the major resistant loci.

Comparison between the effect of some *Rpv* loci showed that the effect on DM is partial, sporulation is always present. The effect of *Rpv3.1*, *Rpv3.2* and *Rpv3.3* are very different between them. The *Rpv3.1* locus mediated a reduction of the sporulation rate in association with necrosis (Bellin et al., 2009; Özer et al., 2017; Foria et al., 2018; Eisenmann et al., 2019; Possamai, Migliaro, Gardiman, Velasco, & De Nardi, 2020; Heyman, Ferrarini, Sanchez, Barka, & Höfte, 2021; Wingerter, Eisenmann, Weber, Dry, & Bogs, 2021; Juraschek, Matera, Steiner, & Oerke, 2022) but this effect varied according to the genetic background (Foria et al., 2018). In comparison, the *Rpv3.2* and *Rpv3.3* loci displayed a weak effect on sporulation with a delayed necrosis (Vezzulli et al., 2019; Possamai et al., 2020; Heyman, Ferrarini et al., 2021). The locus *Rpv1* is described to induce a strong effect (Feechan et al., 2013), but in another study, its effect was less important compared to the effect of *Rpv3.1* (Heyman, Ferrarini et al., 2021). The *Rpv* loci originated from Asian origin such as *Rpv8*, *Rpv10* and *Rpv12,* reveal a strong effect on sporulation, combined also with necrosis (Gindro, Pezet, & Viret, 2003; Alonso-Villaverde, Voinesco, Viret, Spring, & Gindro, 2011; Blasi et al., 2011; Possamai et al., 2020; Heyman, Ferrarini et al., 2021; Wingerter et al., 2021; Juraschek et al., 2022; Frommer et al., 2023). The level of resistance conferred by a resistance gene may also depend on the organ. Indeed, DM assessments

on a segregating population for *Rpv1* showed a high level of leaf resistance with low variability in the field (<10% severity) whereas the disease levels on bunches were much higher with a high variability between *Rpv1* genotypes (1–48%) (Calonnec, Wiedemann et al., 2013).

For PM, among the dozen *Run/Ren* loci described, only very few are present in resistant varieties. The *Run1* locus conferred a total resistance characterized by the presence of dark brown spots due to programmed cell death (PCD; Feechan et al., 2013; Agurto et al., 2017). The *Ren3* and *Ren9* loci mediated partial resistance explained by a reduced growth of the mycelium and sporulation associated to few necrosis and to the establishment of papillae (Zendler, Töpfer, & Zyprian, 2021).

Resistance and locus identification for BR are scarce (Dalbó et al., 2001; Rex, Fechter, Hausmann, & Töpfer, 2014; Bettinelli et al., 2023). Several studies have searched for sources of resistance (Szabó et al., 2023), but BR QTLs appear to have organ-specificity, making resistance more complex. Recent analyses performed on the progeny of a cross of the varieties Merzling (resistant) and Teroldego (susceptible) have identified two QTLs *i.e. Rgb1* and *Rgb3* on chromosome 14 (Bettinelli et al., 2023). Some *V. vinifera* varieties seem to be less susceptible to BR (Tomoiaga & Chedea, 2020).

Regarding resistance to GM, only one to two potential QTL have been identified in grapevine (Sapkota, Chen et al., 2019). However, studies have assessed the susceptibility of different *Vitis* genotypes (Gabler et al., 2003; Wan et al., 2015; Coelho et al., 2019). Resistance has been found in various species, particularly in accessions of *V. amurensis, V. quinquangularis, V. adstrictia, V. davidii, V. pseudoreticulata*, *V. romananeti, V. yenshanensis* and even *V. vinifera* (Rizamat genotype, Wan et al., 2015). The most resistant accession found in the study of Wan et al. (2015) (Chinese wild *Vitis*, (Qinling grape) Pingli-5), showed faster and more intense antioxidant activities than in susceptible genotypes which may be related to resistance.

No complete resistance to the pathogens responsible for GTDs is known, but the susceptibility of *Vitis* species is highly variable, particularly that of *V. vinifera* (Csótó et al., 2023). The evaluation of 305 varieties of *V. vinifera* or interspecific hybrids (crosses with *V. labrusca, V rupestris* and *V. amurensis*) concludes that ancestral diversity shows the existence of greater resilience to GTDs. Domestication of the grapevine is thought to have created a bottleneck effect with low genetic diversity in *V. vinifera* (Csótó et al., 2023), favouring the absence of resistance.

2.4.2 Functions of proteins involved in genetic resistance toward oomycetes and fungi

The defence mechanisms set up in resistant varieties combine different processus: parietal reinforcement, production of phytoalexins or PR proteins, such as hydrolytic enzymes. Involvement of HR is also recurrent mechanism in genotypes resistant to DM *e.g.* Bianca, Regent, Solaris (Bellin et al., 2009; Gong et al., 2022; Figueiredo et al., 2023).

Regarding genetic resistance to DM, callose synthesis is probably one of the first plant defences described. Callose is a ß-(1,3)-D-glucan and is deposited between the cell wall and the plasma membrane at the sites of the pathogen attack. The major roles of callose are to reinforce plant cell wall by increasing the stiffness at the site of infection to limit the ingression of pathogens and to reduce the nutrient uptake by compart the pathogen (Wang, Wang et al., 2022). For the *Rpv3.3, Rpv10* Solaris variety, callose deposits were observed on stomata, around germinated zoospores already, at 7 hpi and on the adjacent non-infected stomata, after 48 hpi (Gindro et al., 2003) (Fig. 2, Part 2.6). Again, for Solaris variety, a deposit was observed on the whole haustoria surface whereas for the *Rpv3.1* Regent variety, callose was noticed only on the neck of these structures (Juraschek et al., 2022).

Most resistant genotypes or varieties that are derived from breeding by introgressing resistance genes from *V. amurensis, M. rotundifolia* and even *V. riparia*, *V. amurensis,* have been described to activate defensive pathways involving phytohormones such as SA, JA, ET and ABA, and to rapidly induce overexpression of PR and phenylpropanoid biosynthetic pathway genes during early stages of infection. All these factors vary as a function of time after infection (Li et al., 2015). Several resistant varieties with different *Rpv* resulting from a cross between *Rpv3.1* and a susceptible variety have been compared and the obtained results seem to indicate that phytohormones may be involved in resistance to *P. viticola* (Heyman, Ferrarini et al., 2021). Resistant genotypes specifically modulate genes involved in signalling, stilbene biosynthesis and defence hormone biosynthesis at an early stage of infection (Polesani et al., 2010; Malacarne et al., 2011; Figueiredo et al., 2017). Finally, the Georgian *Rpv* loci seem to be based on the overexpression of genes encoding ethylene signalling (Sargolzaei et al., 2020; Sargolzaei et al., 2021).

The effect of the DM resistance loci in the variety Regent (*Rpv3.1)* (Eisenmann et al., 2019) triggers strong overexpression of stilbene synthase genes, leading to the production of zoospore-toxic stilbenes (trans-pterostilbene,

epsilon-viniferin) with a PCD. The genes induced by the presence of *P. viticola* include also the overexpression of PR and stilbene pathway genes (*VvPR10–1, VvPR5, VvROMT, VvSTS1*). The presence of the *Rpv3.3* locus was correlated with stilbenoid concentrations and the efficacy against *P. viticola* (Malacarne et al., 2011). In addition, several genes are modulated exclusively in resistant genotypes (compared to susceptible varieties), including ethylene biosynthesis genes. Similar effect is found for *M. rotundifolia* and in the variety Solaris (Alonso-Villaverde et al., 2011; Wang, Wu, Zhang, & Lu, 2018). There is a closed correlation between resistance to *P. viticola* and epsilon-viniferin and the level of trans-pterostilbene at the site of infection. Confirming this fact, a cluster of phenylpropanoid pathway genes (2 *VvPal* and 34 *VvSTS*) has been described in *Rpv10* locus in Solaris (homozygous) or Sibera (heterozygous) varieties, with maximum overexpression in the *Rpv10* homozygote. Peroxidase genes (involved in the dimerisation of epsilon-viniferin), the transcription factor WRKY75, the ethylene-related factor ERF5 and PR genes (PR1, PR1.3) were more highly expressed in the Solaris variety (Fröbel, Dudenhöffer, Töpfer, & Zyprian, 2019). In *M. rotundifolia* Noble, both the stilbene synthase gene and the *ROMT* gene coding for an enzyme catalysing the pterostilbene biosynthesis were recorded as strongly overexpressed (Wang, Wu et al., 2018). In the presence of *P. viticola*, resistant genotypes overexpressed the stilbene pathway genes more intensely and more rapidly than susceptible varieties. In addition, classically, the levels of SA, MeJA and ABA are increased in *M. rotundifolia*, associated with the production of pterostilbene and epsilon-viniferin. Regarding the ethylene pathway, it is thought to be involved in the resistance of the Mgaloblishivilii grape variety associated with the overexpression of a GDSL lipase-like gene involved in local and systemic resistance, a glucanase and parietal reinforcing genes (Toffolatti et al., 2018). Depending on the locus or QTL present, resistance to the pathogen is more or less total (see above) and cell death plays an important role. Two gene clusters associated with *Rpv12* were found by Frommer et al. (2023). One of them is related to cell death (ECD6-like gene) and the second one contains genes that are involved in DM recognition and are thought to contribute to the quantitative resistance. Genes involved in ethylene regulation (ethylene-insensitive EIN4) and regulatory genes that activate ROS production and lead to cell death were identified. The mechanisms underlying the resistance of the three loci *Rpv3*, *Rpv10* and *Rpv12* revealed an early cell death response between 8 and 12 hpi for *Rpv12* and *Rpv10*, coupled with the formation of ROS and an increase in resveratrol levels preceding cell death. The rapid onset could explain the differences observed in DM growth and sporulation. *Rpv12* and *Rpv10* were more efficient than *Rpv3.1*. However, the

Rpv12Rpv3.1 combination was able to withstand the high incidence of DM (Wingerter et al., 2021). There are subtle interactions between cell death, ROS and stilbene production depending on QTLs present in the resistant varieties. However, the extent of the response is closely related to the QTLs present and the aggressiveness of the pathogen.

The speed with which cells respond to attack and the rate of accumulation of toxic phytoalexins (*e.g.* viniferins) are certainly a key of the efficiency of resistance (Bavaresco, Mattivi, De Rosso, & Flamini, 2012).

In addition to stilbenes, more molecules have been described to accumulate in mildew-resistant *Vitis* varieties (*e.g.* quercetin-3-O-glucosides, linoleic acid, alanine, isoprenoids, malic acid, tartaric acid) (Batovska et al., 2008; Ali et al., 2012). Research on biomarkers associated with DM resistance in the Bianca variety, between 24 and 96 hpi with *P. viticola*, confirms the accumulation of phenylpropanoids, including phytoalexins, VOCs (benzaldehyde) and other molecules whose nature and content vary over time (Chitarrini et al., 2017). It has also been described that ceramides involved in PCD accumulate. Metabolic biomarkers have been identified by characterizing metabolic profiles associated with disease resistance (Viret, Jean-Laurent, & Katia, 2018; Maia et al., 2020). Histochemical, transcriptomic, and metabolomic analyses of DM response in susceptible and resistant varieties showed strong correlation between stilbenoid biosysnthesis related genes, stilbene accumulation, and pathogen growth inhibition (Eisenmann et al., 2019). These types of studies can be complemented by targeted metabolomic measurements and quantification of compounds involved in defence.

Targeted metabolomic analysis showed a significant induction of stilbenoids in a population segregating for DM resistance (Vezzulli et al., 2019). At the proteomic level, resistant genotypes (*e.g.* Regent) accumulated the PR10 protein and proteins associated with cell death, reactive oxygen species and JA processes (Figueiredo et al., 2017). Cell death in plant has been shown to be one of the keys to differentiate resistant genotypes. Liu et al. (2021) showed that the resistant genotype *V. davidii Liuba-8* after infection with *P. viticola* favoured photosynthetic activity, but also PR proteins such as PR4, PR5, PR10.2 and PR10.3 and proteins involved in ROS metabolism. They suggest that PR4 has a basal function in resistance. In addition, genotypes with the *Rpv1* and *Rpv3 loci* pyramided QTLs accumulate large quantities of proteins associated with redox systems, energy metabolism and stress responses (Nascimento-Gavioli et al., 2017).

Development of defence molecules also requires mobilisation of nitrogen and carbon resources. Upon *P. viticola* infection, the primary

metabolism of resistant varieties may be affected at the level of sugars, organic acids and amino acids, which can be found in greater quantities (Chitarrini et al., 2017), linked to modulations of genes involved in carbon metabolism, including photosynthetic activity (Malacarne et al., 2011). In the Regent variety, several proteins specific to the resistant varieties have been described to be involved in primary and general carbon and energy metabolism processes on a global scale (Figueiredo et al., 2017).

For PM resistance, callose was described at two dpi around the first haustoria of genotypes carrying *Run1* (Agurto et al., 2017). A recent review of genetic resistance to PM (Sosa-Zuniga et al., 2022) reports some effective defence mechanisms for the *Ren1* QTL with overexpression of the *VvCAD* gene, which encodes cinnamoyl-CoA reductase, a key enzyme in lignin biosynthesis, and a GTPase involved in membrane trafficking in the secretory pathway. The *Run1 Ren1* QTLs are thought to lead to more intense ROS production, callose accumulation, activation of stilbene synthase, a penetration gene, and cell death, all associated with improved inhibition of *E. necator* development (Agurto et al., 2017). The cuticle plays an essential role in inhibiting or inducing *E. necator* penetration into leaves and berries of resistant Italian varieties (Özer et al., 2017). The link between the resistance gene and the triggered defence can however be more complex to understand, especially for PM resistance. Comparing two resistant varieties Artaban [*Run1 Ren3 Ren9*] and Prior [*Ren 3 Ren 9*] for defence genes expression and susceptibility to PM, two PR genes coding for chitinases were constitutively overexpressed (*VvPR3, VvPR4*) in Artaban, whereas in Prior *VvPR4bis*, and *VvAlli2* (an allinase) were overexpressed but only after induction with PM (Calonnec, Jolivet, Ramaroson, Dufour, & Corio-Costet, 2021). The parent of Artaban, the 3082–1-42 genotype carrying also the *Run1* locus is however totally resistant to PM without overexpression of PR3. Other genes from the Artaban or the Prior varieties were found to be potentially involved in PM resistance: genes encoding *PR10.1,* transcription factors, defence regulators or genes that activate SA signalling (Qiu et al., 2015).

Functional genomics studies to validate the candidate genes involved in defence are difficult to perform in *V. vinifera*. However, several genes from *V. amurensis* (*VaPRR* gene, *VaPR15*-TLp), *V. pseudoreticulata* (*VpPR4, VpPR 10.1, VpRpW8, VpCDPK9-13, VpRH2,* F-box protein; *VpSTS29/STS2*), *V. quinquangularis* (*VqSTS6*) or *V. riparia* (*VrRH2*) have been characterised in susceptible *Vitis* by functional genomics. The genes involved have conferred either resistance or tolerance, mainly to *E. necator* or *P. viticola* (Vezzulli et al., 2022).

For BR resistance, two out of three loci are described including *Rgb1* that is thought to be enriched in genes involved in phloem dynamics, mitochondrial respiration and ATP formation. This may be related to cell death and ROS reduction. The *Rgb3* locus, is described to contain a cluster of genes involved in lipid transport, phosphatidic acid dephosphorylation, regulation of apoptosis, germin-like proteins (GLP3) and cell death promoters (Bettinelli et al., 2023). These GLPs have diverse functions including oxalate oxidase, superoxide dismutase and polyphenol oxidase, involved also in cell death.

Resistance to *B. cinerea* appears to be more closely linked to morphological or anatomical barriers (film and cuticle thickness, number of pores) and antifungal compounds (tannins, phenylpropanoids, PR proteins) (Gabler et al., 2003). A marker based on the physical impedance of the berries, in addition to cluster architecture, has been described to facilitate the selection of resistant grape varieties, as well as potential QTLs that could be related to cuticle regulation and transcript factors (Herzog et al., 2021). Low infection to GM linked to smaller pores on the berry surface, greater skin thickness and a thicker cuticle rich in compounds has been confirmed in a number of resistant grape genotypes (Gabler et al., 2003). Contrary to what has been described for DM, resveratrol levels are not correlated with resistance, to GM and phytoalexins do not reach sufficient concentrations to inhibit its growth. Another study indicated that the antioxidant activity is temporally higher in a resistant genotype infected by *B. cinerea* than in a susceptible one (Wan et al., 2015). More recently, *B. cinerea* attack on flowers was shown to induce the expression of many genes involved in the accumulation of stilbenoids, lignin and ROS. These responses are likely to have an impact on the quiescent *Botrytis* (Haile et al., 2020). These pathways are dependent on the SA pathway and are able to interrupt the progression of the pathogen at the veraison stage (Kelloniemi et al., 2015). More recently, a possible QTL has been identified as the EDR2 factor, which is negatively regulated in resistant varieties in association with the expression of several transcription factors (ERF, NAC and WRKY; Su et al., 2023).

Information on GTDs resistance in vine is severely lacking. However, *V. vinifera* subs *sylvestris* show resistance to *Neofusicoccum parvum* that are not related to vessel size, but to the accumulation of stilbenes, particularly viniferin trimers, and to the overexpression of *PAL* and *STS* genes (Khattab et al., 2021). Stilbenes could have an effect on GTD pathogens, but depending on the pathogen, the quality and quantity of the molecules, the efficacy would vary (Lambert et al., 2012; Stempien et al., 2017).

With regard to vine resistance to various pathogens, while we currently have a growing amount of information on the mechanisms involved in resistance to DM and PM, there is a serious lack of data or even sources of resistance for other pathogens.

2.5 Ontogenic resistance

Depending of its developmental stage, a susceptible host can acquire a form of resistance to a pathogen (Develey-Rivière & Galiana, 2007). This age-related resistance/tolerance, commonly called ontogenic resistance, reflects the dynamic modification of tissue receptivity concomitant to organ development. This may not lead to immunity *per se*, but the levels of resistance that are achieved during the aging of plant tissues/organs can greatly affect the severity of the disease by reducing the different traits of the pathogen development. When the cycle of the pathogen and that of the plant are sufficiently delayed, the plant may even escape infection, especially when it concerns fruits.

In grapevine, plant tissues have been reported to develop age-related resistance to several fungal pathogens. It has been highlighted and assessed on leaves and/or bunches for *P. viticola* (Kennelly, Gadoury, Wilcox, Magarey, & Seem, 2005), *E. necator* (Gadoury, Seem, Ficke, & Wilcox, 2003; Merry, Evans, Corkrey, & Wilson, 2013; Calonnec, Jolivet, Vivin, & Schnee, 2018), *G. bidwellii* (Hoffman, Wilcox, Gadoury, & Seem, 2002; Molitor & Berkelmann-Loehnertz, 2011), and *B. cinerea* (Deytieux-Belleau et al., 2009). For PM, the three traits of pathogenicity (infection efficiency, sporulation and mycelium growth) were affected by leaf age (Calonnec et al., 2018). Indeed, depending on the age of the organ, the germination of conidia decreases and the sporulation time increases. In a similar manner, for DM, Steimetz et al. (2012) reported that the first fully developed leaf beneath the apex of plants grown in greenhouses had a disease severity of 90% after inoculation with *P. viticola*, whereas the next two leaves showed only 80% and 50%, respectively. Concerning clusters, their sensitivity could be lower from 20 days after bloom (Kast & Stark-Urnau, 2000) and fewer symptoms would be then observed on the oldest berries. Indeed, it is also often reported that, after veraison, grape berries become less susceptible to mildews (Ficke, Gadoury, & Seem, 2002; Kennelly et al., 2005). In the case of PM, three to four weeks after bloom, the latter are almost totally resistant to *E. necator* (Kast & Stark-Urnau, 2000; Gadoury et al., 2003), however the variability of flowering time has to be considered to predict the susceptibility of the bunch population at the plot scale. The level of

radiance received before inoculation can also strengthens ontogenic resistance through modifications to berry physiology, such as a decrease in pH and K concentration and an increase in polyphenol and anthocyanin concentration (Zahavi & Reuveni, 2012). Regarding DM, grape clusters are susceptible as soon as inflorescences become visible (BBCH 53) but their susceptibility decreases rapidly few weeks after bloom (Kennelly et al., 2005). Conversely, in the case of the GM agent *B. cinerea*, green berries are more resistant than older berries and it is only after veraison that they become increasingly sensitive to this necrotrophic pathogen (Kelloniemi et al., 2015).

Although the mechanisms involved in ontogenic resistance remain speculative, they might differ from those involved in response to infection in the classical defence system (Develey-Rivière & Galiana, 2007; Gee, Gadoury, & Cadle-Davidson, 2008). Regarding PM, the process has been first described for berries, for which the infection is stopped when the sugar content of the berries reaches 8% (Delp, 1954). At this stage, Ficke et al. (2002) report that the low penetration rate of the pathogen is correlated with a change in both the structure and the chemical composition of the berry cuticle, suggesting that cuticle thickness and/or sugars and polyphenols in berries are involved in ontogenic resistance. In leaves, a clear correlation exists between the ontogenic resistance and the leaf transition state from sink to source (Calonnec et al., 2018). In this case, soluble and complex sugars could constitute indicators of a change in the metabolic processes and could be considered as indirect markers of tissue age-related resistance. For the young expanding leaves, a high rate of cellular reactions of plant defence might be energetic and the consumption of carbohydrates too costly (Huot, Yao, Montgomery, & He, 2014). Mildew Locus O (MLO) genes (PM susceptibility genes) might also be involved in the regulation of plant defence. One hypothesis is that in young leaves, PM effectors may combine with specific MLO proteins, preventing the fungal chitin from being recognized as a pathogen-associated molecular pattern (PAMP) by the plant's membrane receptors and thus suppressing the PAMP-triggered immunity (PTI) (Dry et al., 2010). In the old leaves, changes in the expression or activity of the MLO genes may restore the PTI (Qiu et al., 2015) with consequence of increase ontogenic resistance.

PR proteins could also be components of ontogenic resistance. Indeed, after inoculation with PM, thaumatin like (PR5), germin like protein (PR15), polygalacturonase inhibitor protein (PGIP) and chitinase (CHIT3) were overexpressed in the oldest leaves of Cabernet Sauvignon (Calonnec et al., 2021). Phenylpropanoid and ethylene pathways were also 'leaf age'-dependent.

This overexpression was not recorded in the youngest, susceptible leaves as if their physiology dedicated to primary growth did not allow them for this basic defence. Therefore, the age of the leaf is of prime importance to consider in the literature related to gene expression and defence. In the case of *P. viticola* (DM), functional stomata are required to enter the plant. In this context, microscopic analysis of different grapevine varieties at two phenological stages, namely BBCH 69 (all caps fallen) and BBCH 75 (pea-sized berries), revealed that at both stages, stomata of the receptacle (lenticels), berries or rachis pedicels are closed or collapsed in increasing numbers compared to BBCH stage 53 (inflorescences clearly visible, flower buds not separated) (Gindro et al., 2012). Since the number of functional lenticels decreases during the aging of the berries, this considerably limits the possible points of entry for *P. viticola* and contributes to a natural increase in the resistance of the organ.

Regarding *B. cinerea*, Kelloniemi et al. (2015) reported that infected young berries (at veraison) activate efficient defence mechanisms involving localized H_2O_2 production, activation of SA-dependent defence genes, and genes involved in stilbene and lignin biosynthesis. Conversely, in inoculated mature (susceptible) berries, a very different transcriptome is activated in which part of the difference is linked to the activation of the JA-dependent defence pathway.

From a practical point of view, the additional effects of ontogenic resistance modification should be explored in order to reduce fungicide application (Calonnec, Wiedemann et al., 2013; Bleyer, Bleyer, & Schumacher, 2023) and obtain better control of fungal diseases in sustainable agriculture. Finally, note that this ontogenetic resistance also has significant consequences on elicitor-induced resistance. For example, Steimetz et al. (2012) showed that the younger, not fully expanded leaves were less protected after a treatment by sulfated laminarin (PS3) than older ones. Indeed, the adult leaf turns out to be more responsive to PS3 treatment than the young one and this is correlated with the fact that the guard cells of old (*i.e.* mature) leaves have a higher capacity to produce defence reactions, including H_2O_2, or to produce them earlier than younger leaves. Thus, whatever the pathosystem considered, it is fundamental to consider ontogenetic resistance as a factor impacting the level of resistance and therefore of immunity (basal as well as induced) of the host plant.

2.6 Comparison between genetic and induced resistances

The induction of defences by an exogenous agent (microorganisms, extracts, molecules) can lead to numerous modulations of the grapevine's defences, many of which are similar to those described for resistant *Vitis* species and

varieties, including signalling pathways (see above). Indeed, several defence processes specific to a resistant variety against DM attack can be initiated in susceptible varieties by elicitor induction of defences (Iriti et al., 2004; Aziz et al., 2006; Belhadj et al., 2006; Perazzolli et al., 2011; Boubakri et al., 2012; Dufour et al., 2013; Gauthier et al., 2014; Garde-Cerdán, Portu, López, & Santamaría, 2016; Gil-Muñoz, Fernández-Fernández, Crespo-Villegas, & Garde-Cerdán, 2017; Bellée et al., 2018; Krzyzaniak, Trouvelot et al., 2018; Burdziej et al., 2021). As an example, some PR genes and other genes involved in the phenylpropanoid pathway are potential markers of pathogen resistance both in resistant varieties (Eisenmann et al., 2019) and after elicitor induction (Fedorina, Tikhonova, Ukhatova, Ivanov, & Khlestkina, 2022; Jeandet et al., 2023).

Most of these pathways can also be activated in susceptible varieties during pathogen attack but the activation occurs at a lower intensity, resulting in different kinetics of gene expressions and no effective protection.

In the case of PR proteins, overexpression of the thaumatin-like gene in Chardonnay (*PR5/TL1*) (Dhekney, Li, & Gray, 2011) results in a delay of PM symptoms and a decrease of severity of BR symptoms. The *PR5* gene has also been described to be overexpressed after defence stimulation with efficacy against DM (Burdziej et al., 2021) and in resistant genotypes expressing the *Rpv3.1* locus (Eisenmann et al., 2019). In most cases, the efficacy of elicitors correlates with the overexpression of different *PR* genes (*PR1*, *PR2*, *PR4*, *PR5*) or with the accumulation of these proteins (Perazzolli et al., 2011; Banani et al., 2014; Dufour et al., 2016; Calonnec et al., 2018; Burdziej et al., 2021).

Besides PRs, genes and/or molecules from the phenylpropanoid pathway (Wang, Liao, Kan, Han, & Zheng, 2015; Burdziej et al., 2021; Jeandet et al., 2023) as well as those involved in parietal reinforcement are generally overexpressed or accumulated. Identical molecules (*e.g.* pterostilbene, viniferins) have been described to play a protective role in genetic and induced resistance (Schnee, Viret, & Gindro, 2008; Jeandet et al., 2023).

Concerning plant metabolism, as for resistant varieties, elicitors induce changes in primary metabolism (Lemaître-Guillier et al., 2017; Krzyzaniak, Trouvelot et al., 2018; Burdziej et al., 2019; Bodin et al., 2020) and modulate the levels of certain amino acids, organic acids and sugars. However, depending on the genetic background of the plant, the observation time after treatment in the case of elicitors, and the pathogen under consideration, the gene modulations and/or accumulation of products obtained may vary. Moreover, there

are similarities in the hormonal signalling pathways (SA, JA, ET) between induced and genetic resistance. For example, segregating populations with the *Rpv3.3* locus (Malacarne et al., 2011) strongly modulate the ethylene pathway, as do plants treated with elicitors (BTH, MeJA, ethephon) to control DM and PM (Belhadj et al., 2006; Belhadj, Telef et al., 2008; Perazzolli, Dagostin, Ferrari, Elad, & Pertot, 2008; Dufour et al., 2013; Dufour et al., 2016; Burdziej et al., 2021). Genotypes with the *Rpv1* and *Rpv3.1* pyramidal loci accumulate large amounts of proteins that are associated with redox systems, energy metabolism and stress responses (Nascimento-Gavioli et al., 2017). Similarly, plant protein content (associated with primary metabolism, defence pathways, stress responses and redox systems) is increased by the induction of defences by elicitors such as laminarin (Lemaître-Guillier et al., 2017), even JA or BTH (Burdziej et al., 2019; Burdziej et al., 2021).

A major difference between resistance induced by an elicitor (PTI) and genetic resistance (ETI) appears to be the phenomenon of apoptosis (HR). This is widespread in resistant varieties (Figueiredo et al., 2017; Possamai et al., 2020; Sosa-Zuniga et al., 2022) and less commonly described after elicitation. However, several studies have shown that elicitors (thiamine, sulfated laminarin, MeJA, harpin) induce cell death (Trouvelot et al., 2008; Boubakri et al., 2012; Gong et al., 2022; and Fig. 2 N.1-O), which is closely linked to the accumulation and regulation of ROS.

Finally, all these defence responses counteract the development of the pathogen and, when they are not sufficient to block it *in planta*, often contribute to limiting the asexual sporulation phase. In this context and in response to DM, it is not uncommon to observe aborted sporangiophores, incapable of producing sporangia (Fig. 2 J.1 and L).

Although gene expressions combined with metabolomics and molecular quantification are becoming commonplace to explain or better understand the effects of elicitor-induced resistance, genetic resistance studies still lack data using combined approaches as described for elicitors. In all studies of resistance, induced or genetic, most of the molecules or gene regulations identified as markers of resistance are correlated between the presence of molecules and/or gene expression and effectiveness against the disease.

Overall, the ability of resistant varieties to induce a HR in an almost generic manner is one of the major differences between genetic resistance and resistance induced by an elicitor. It seems that this cell death plays an important role in grape-biotrophs interactions such as *P. viticola* and *E. necator* by reducing their colonization.

3. Concluding remarks

As described above, grapevine has developed complex defence mechanisms to avoid or limit infection by fungi and oomycetes. However, these mechanisms are confronted to the evolution of pathogens and are also dependent on environmental factors.

Co-evolution of the host and its pathogens is a constant arm race, also known as the "Red Queen hypothesis" (Solé, 2022). Pathogens can counteract defence mechanisms by several strategies including the rapid evolution of their pathogeny and virulence factors. Notably, *P. viticola* has a great potential to fastly evolve thanks to a large population size, an obligatory sexual cycle (Gessler et al., 2011) and a highly repetitive genome (Dussert et al., 2019). In Europe, breakdown of *Rpv3.1*, *Rpv3.2*, *Rpv10* and *Rpv12* resistance loci has been reported for some adapted strains of *P. viticola* (Peressotti et al., 2010; Eisenmann et al., 2019; Heyman, Chrysargyris, Demeestere, Tzortzakis, & Höfte, 2021; Wingerter et al., 2021; Paineau, Mazet, Wiedemann-Merdinoglu, Fabre, & Delmotte, 2022). Breakdown of the *Run1* resistance locus has also been observed for an *E. necator* strain collected on *M. rotundifolia* in North America (Feechan et al., 2015). A genome-wide association study (GWAS) identified mutations associated with the lack of effector recognition and the resulting overcoming of the resistance locus *Rpv3.1* (Paineau et al., 2023) and *Rpv10*. The presence of adapted strains to some resistance loci raises the question of the resistance durability. As grapevine is a perennial crop, the most convenient strategy to strengthen the resistances is to stack several resistance loci in a same variety, this is also called "pyramiding resistances". Defence elicitors or biological control agents, besides their use to protect susceptible grape varieties against DM and PM, can also be applied to resistant genotypes to limit the risk of resistance evasion by pathogens (Corio-Costet, Dufour, Cluzet, Lambert, & Merdinoglu, 2013) and to induce resistance against other diseases namely BR.

Environmental factors play a crucial role in plant pathogen interactions, *via* effects on plant and/or pathogens. On the pathogen side they condition the development and the risk of infection. For example, it is well known that the number and duration of asexual cycles of *P. viticola* and *E. necator* are highly dependent on climatic parameters such as temperature, rainfall and hygrometry. On the plant side, environmental factors impact the plant development and physiology. Optimal photosynthetic activity is essential for plant growth and development, and also for fuelling the energy costs necessary for defence responses (Bolton, 2009). In addition to its role on

plant development, nutrition can also promote diseases by direct effect on the pathogen nutrition. High vigour makes vines more susceptible to DM, PM and GM (Valdés-Gómez, Fermaud, Roudet, Calonnec, & Gary, 2008; Valdés-Gómez, Gary, Cartolaro, Lolas-Caneo, & Calonnec, 2011; Calonnec, Burie et al., 2013; Marcianò et al., 2023). Environmental factors can also be sources of stress for the plants. Depending on their nature, intensity and frequency, abiotic stresses (alone or in combination) can cause morphological and physiological alterations in grapevine and impact interaction with pathogens (Heyman, Chrysargyris et al., 2021). In relation to their effects on grapevine and pathogens, environmental factors thus surely condition the efficiency of elicitor-induced resistance. This may explain, at least in part, the variability of the efficiency of elicitors to protect grapevine against pathogens in field conditions. In addition to abiotic factors, biotic factors can also have an impact on the interaction between grapevine and its pathogens. In vineyard, grapevine lives in close association with large numbers of microorganisms, structured into communities and actors of the plant holobiont (Vandenkoornhuyse, Quaiser, Duhamel, Le Van, & Dufresne, 2015; Hassani, Durán, & Hacquard, 2018). In this context, the overall health of the plant may be conditioned by diversity, disparity and structure of its microbial communities (Bettenfeld et al., 2020; Bettenfeld et al., 2022). A better understanding of the most important drivers in the composition of plant microbiomes is clearly important to (i) better characterize plant immunity in an agricultural context and (ii) imagine new ecofriendly phytosanitary protection strategies taking into consideration the management of the vine holobiont.

GTDs, especially Esca, are undoubtedly among the most complex grapevine diseases to understand and control. As mentioned above, they are caused by several fungi and the etiology and epidemiology are highly complex (Claverie, Notaro, Fontaine, & Wery, 2020). Sodium arsenite has long been used to control Esca before it was banned due to its high toxicity. Its mode of action was investigated (Bruez et al. 2021, Trouvelot et al. 2023) but it didn't allow to design alternative solution. Other physical, chemical, and biological solutions have been assessed but their effectiveness is often limited and/or their deployment at the vineyard scale turns out to be not possible (Gramaje, Urbeez-torres, & Sosnowski, 2018, Mondello et al. 2018, Kenfaoui et al. 2022). Though no variety or species was found to be resistant to GTD pathogens yet (Bertsch et al., 2013), it is worth continuing the search for resistance genes, as these genes may be recessive. Notably, a first resistant gene for the grapevine fanleaf virus

(GFLV) was recently identified as a single recessive locus (*rgflv1*) in the Riesling susceptible variety (Djennane, Prado, & Dumas, 2021).

The increasing knowledge in the molecular mechanisms involved in the grapevine immunity, together with newly available genomic tools, offer new plant-protection approaches that can be used in combination with those described above. Notably, genome editing, which consists of modifying one or more sequences in a plant genome at a precise locus using nucleases (CRISPR/Cas9 technology), will undoubtedly be exploited in the future for disease resistance. It has recently been developed in grapevine to improve berry quality (Ren et al., 2016) and resistance to different diseases (Malnoy et al., 2016; Wang, Tu et al., 2018; Giacomelli et al., 2023). After mutation of the grapevine *VvWRKY52* transcription factor gene, the edited plants showed the *Botrytis*-resistant expected phenotype (Wang, Tu et al., 2018). For resistance to PM or DM, the strategy was to target the susceptibility genes *VvMLO* or *VvDMR6*, respectively. The *DMR6* gene encodes an important enzyme of the plant immunity involved in the SA catabolism as demonstrated in tomato (Thomazella et al., 2021). Targeted mutations of *VvDMR6* and some of the *VvMLO* genes in grapevine allowed to significantly reduce PM or DM severity (Pessina et al., 2016; Wan et al., 2020; Giacomelli et al., 2023). If these proofs of concept are confirmed in vineyard conditions, this technology should open new perspectives to promote a more sustainable viticulture.

Recently, another innovative approach based on small RNAs was tested to protect grapevine against its pathogens. As first demonstrated with the model plant *A. thaliana* and *B. cinerea*, small interfering RNAs (siRNA) are exchanged between the pathogen and the host during the early stage of infection. Fungal siRNA act as effectors by hijacking the silencing machinery of the plant cell to reduce the expression of genes involved in the defence process such as those encoding the conserved MAP-kinases MAPK1 and MAPK2 (Weiberg et al., 2013). On the other way, plant siRNA can target the expression of fungal genes resulting in a defect in pathogeny (Cai et al., 2018). Based on these mechanisms, an approach called Spray-induced gene silencing (SIGS) is currently developed to protect crops from pathogens. Studies conducted on grapevine berries and leaves in laboratory conditions suggest that SIGS could reduce the infection by *B. cinerea* (Nerva, Sandrini, Gambino, & Chitarra, 2020; Qiao et al., 2021) and *P. viticola* (Haile et al., 2021) but this strategy of protection also needs to be tested in vineyards.

Regardless of the grapevine variety used, the geographical location and the environment, wine growers are trying to ensure the quality of their

grapes while producing the expected yield. Whether the varieties used are susceptible or partially resistant, the aim is to achieve tolerance to both biotic and abiotic stresses. Grapevine resilience can be partially achieved through varietal improvement programmes run in a context of changing environment (global change). In addition to the need to adapt to climate, vines also need to acquire resistance to the current and emerging pathogens using more eco-friendly practices. From the knowledge recently acquired on vine immunity and interactions with pathogens, the challenge is now to design new sustainable production systems including the combination of several levers (genetic resistance, induced resistance, ontogenic resistance management, holobiont and microbiota management, nutrition, biostimulants, new technologies) to preserve grapevine health by increasing its defensive level.

Acknowledgement

The authors acknowledge the support of the French National Research Agency (ANR) under the grant 20-PCPA-0010 (PPR VITAE). (Growing grapevine without pesticides: towards agroecological wine producing socio-ecosystems).

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