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Resistance management in oilseed rape pathogens in France

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Abstract

In France, a dozen of diseases can affect rapeseed but sclerotinia stem rot is the major disease for which chemical control remains the main way to prevent severe attacks. Five fungicide modes of action are effective against *Sclerotinia sclerotiorum* and some solo or mixed products are registered in France. History and current status of resistance to these different groups in French oilseed rape crops are presented. Strategies for preventing and managing fungicide resistance are mainly based on the alternation of products from different classes of biochemical modes of action.

1-Introduction

Oilseed rape is the major oilseed crop cultivated in France with 1.5 million ha per year. The increased production of oilseed rape has also increased the risk of diseases. Nowadays, a dozen of fungal diseases are known to infect French oilseed rape crops but two of them - blackleg caused by *Leptosphaeria maculans* and sclerotinia stem rot (SSR) caused by *Sclerotinia sclerotiorum* - can cause severe damage and important yield losses.
In France, blackleg is mainly controlled by using resistant cultivars, and resistance groups are alternated in order to prevent an overcoming of single resistance genes (Gladders et al. 2006). Chemical control is only performed with DMI fungicides on some susceptible varieties according to the epidemics provided by the

monitoring of the release of *L. maculans* ascospores and a forecasting model so that any shift in fungicide sensitivity has never been observed in practice.
After two severe epidemics in 1971 and 1979 in the middle of France, SSR has become the second more damaging disease on oilseed rape. Therefore in the 1980s, its control had been developed. It was usually associated with light leaf spot (*Pyrenopeziza brassicae)* then white leaf spot (*Mycosphaerella capsellae)* control involving up to three applications. Afterwards due to the cropping of more tolerant cultivars to light leaf spot, the chemical control was focused on sclerotinia stem rot. It was based on one or two applications of benzimidazoles used alone or associated with dicarboxamides or DMIs. As these fungicides provided more preventive effects than curative effects, the applications have become systematic even if a severe epidemic occurs once or twice per decade (Penaud et al. 2013a). In the 2000s, biological control using *Coniothyrium minitans* has provided a new tool for controlling SSR (Penaud and Michi 2009). Moreover tools for disease management such as risk indicator or forecasting models have been developed to optimize the timing of fungicide application and to reduce fungicides use.
Historical and current status of resistance to the different groups of fungicides used in French oilseed rape crops are presented in this chapter. Most data were produced from a network involving the French plant protection service DQSPV, INRA as scientific support, CETIOM as the technical institute for oilseed crops and recently most chemical companies. Resistance management options, based on the reduction of fungicides applications per season and the alternation of solo-products or the mixture of different modes of action, are discussed.

2- Anti-microtubule agents

Carbendazim and its precursor thiophanate-methyl belong to the benzimidazoles group and were introduced in the late 1960s in European crops. Their French registration on oilseed rape was achieved in the 1980s (Fig. 1). The emergence of resistance to carbendazim in oilseed rape was first reported in 1994 in Burgundy in two fields in which reduced efficacy was suspected (Souliac and Leroux 1995). Four years later in 1998, two new resistant strains were detected. In 1999, the presence of resistant strains was detected in 20% of the sampled plots with a proportion of resistant isolates to carbendazim ranging from 10% to 100%. The high level of resistance was associated with a reduction in the efficiency of the fungicidal control (Kaczmar et al. 2000). Since 2000, sclerotinia monitoring was yearly carried out. Depending on disease pressure, between 150 and 340 field locations were sampled for 10 sclerotia per site. The sclerotia were tested for cardendazim resistance using two discriminatory doses (Penaud et al. 2003 and 2013b). Most resistant strains showed normal growth on the two rates of

carbendazim and furthermore exhibited an increased sensitivity to the phenylcarbamate diethofencarb, suggesting a mutation similar to that of the BenR1 phenotype of *Botrytis cinerea* (Leroux et al. 2002). Few weakly carbendazim resistant strains were insensitive to diethofencarb, suggesting another mutation such as in *B. cinerea* as BenR2 phenotype. Benzimidazole fungicides prevent microtubule assembly by binding to β-tubulin (Davidse and Ishii 1995). In *B. cinerea*, resistance is conferred by allelic changes in the gene encoding β-tubulin leading to the change G198A in BenR1 strains and F200T in BenR2 strains (Leroux et al. 2002). Similarly, the G198A change in *S. sclerotiorum* has also been reported in China and PCR methods have been developed to detect both mutations (Li et al. 2002; Yin et al. 2010).

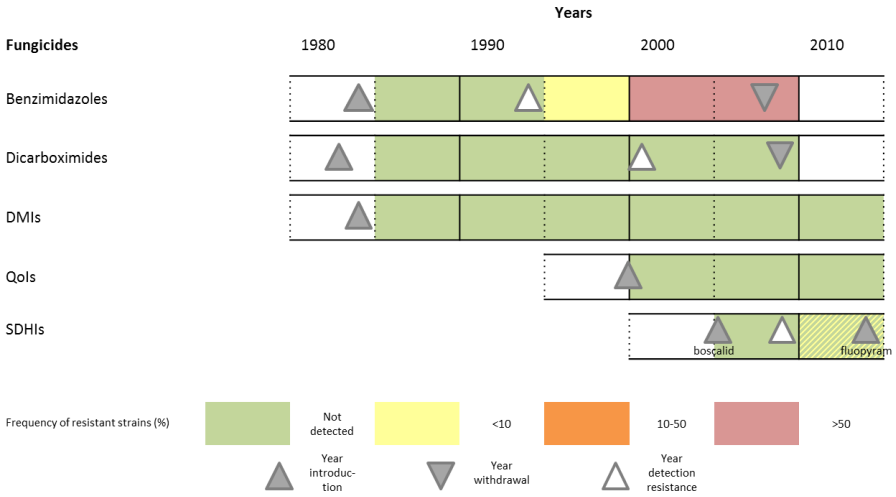

Fig. 1 Evolution of resistance in *S. sclerotiorum* populations in winter oilseed crops in France. (Data from monitoring conducted by ONPV, CETIOM, ANSES, INRA and BASF)

Over the country, the resistance to carbendazim occurred mainly in north-eastern and central regions where 60% to 75% of monitored fields have produced resistant isolates to carbendazim. In the Lorraine region, it was up to 90% field samples collected in 2005 which were resistant. Due to a general widespread of carbendazim resistance and a future withdrawal of this fungicide, there was no more interest for monitoring sclerotinia resistance to benzimidazole fungicides after the mid-2000s (Penaud et al. 2013b).

An analysis of cultural practices suggested that the development of carbendazim resistance was associated with short rotations of oilseed rape (every 2 or 3 years) and at least 5 sprays of benzimidazole fungicides during the last 10 years (Penaud

et al. 2001). Repeated applications of benzimidazole fungicides have also promoted the emergence of resistant strains of *S. sclerotiorum* in oilseed rape in China or Canadian prairies (Pan et al, 1997; Gossen and Rimmer 2001; Wang et al 2014a).
The occurrence of *S. sclerotiorum* resistant to carbendazim led us to develop resistance management strategies and several recommendations are made to farmers : i) give up the systematic treatment at the beginning of flowering and prefer the optimum timing to control the disease according to the climate conditions, ii) when a protection is needed, apply one single spray at the appearance of the first pods and iii) choose an efficient fungicide among different fungicide groups without cross-resistance to carbendazim. Until 2007, three groups of fungicides were registered for sclerotinia use in France: 1) dicarboximide fungicides (iprodione, procymidone or vinchlozolin) which have high intrinsic activity, 2) DMIs (tebuconazole, metconazole) which are of lower intrinsic activity than the previous but interesting against other rapeseed diseases such as light leaf spot or powdery mildew and 3) a strobilurin (azoxystrobin). Thus, using effective fungicides only if necessary and alternating them could make it possible to manage practical resistance and maintain an effective chemical control. At the same time, studies of biological control and of decision support system are investigated for a sustainable crop protection (Penaud and Michi 2009; Penaud et al, 2013a).

3- Dicarboximides

Dicarboximides (*i.e.* iprodione, procymidone, vinclozolin) were registered on oilseed rape in the early 1980s firstly for controlling black spot on pods (*Alternaria brassicae*) and afterwards for sclerotinia control.
Although the dicarboximide fungicides were the first authorized for sclerotinia control, the high price of these products has restricted their use, farmers preferring cheaper solutions mainly based on carbendazim alone or in mixture. However, their use has increased as soon as the resistance of *S. sclerotiorum* to carbendazim became widespread across the country. Dicarboximide fungicides used solo and in mixture represented 50% of fungicide treatments applied to prevent attacks of sclerotinia in the mid-2000s (Fig. 2).

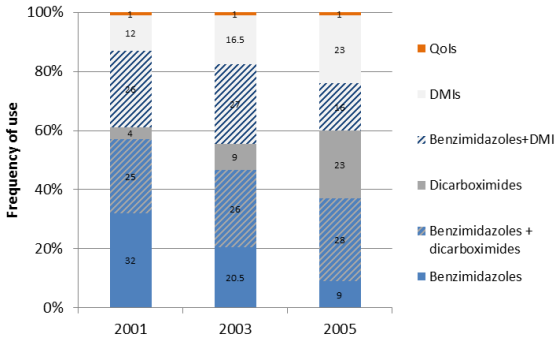

Fig. 2 Use of the main groups of fungicides for controlling SSR, between 2001 and 2005 (from CETIOM cultural practices surveys)

Despite the increase in use, there was no practical loss of efficacy in the field. For 6 years (2001 – 2006) of monitoring for dicarboximide fungicides, *S. sclerotiorum* isolates resistant to iprodione were only detected in 9 field locations (Fig. 3).

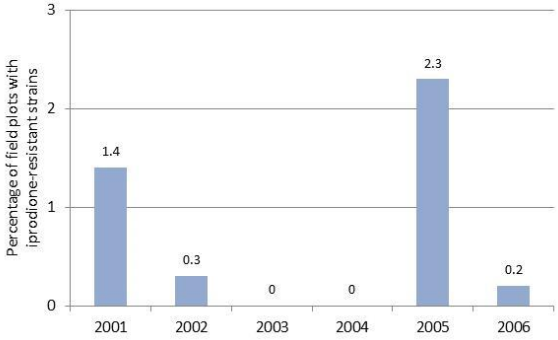

Fig. 3 Frequency of oilseed rape fields with at least one iprodione-resistant strain of *S. sclerotiorum*

In each case, the rare detections were unrelated to the use of this chemical group in the field, suggesting that they could have randomly emerged in local populations of *S. sclerotiorum*. Since 2007, procymidone, vinclozolin and iprodione were withdrawn for toxicological reasons and nowadays, no further fungicides from this group are registered for controlling SSR on oilseed rape. Therefore the monitoring was also given up.

The first field resistant isolate was introduced as a resistance check in monitoring tests. This strain had gradually lost its ability to produce sclerotia after successive subcultures, suggesting that resistance would have a negative impact on the fitness of this resistant strain which therefore could be less competitive compared to the population of susceptible strains as also suggested for some *B. cinerea* resistant phenotypes. Otherwise, resistant mutants of *S. sclerotiorum* can easily be induced in the laboratory (Gindrat 1993, Liu et al. 2009, Duan et al. 2014). In a recent

study, the laboratory induced iprodione-resistant mutants lost the ability of producing sclerotia after 10 generations on PDA medium. These mutants also showed an increase in sensitivity to osmotic stress. The putative target site of dicarboximides is supposed to be a two-component histidine kinase, encoded by the group III HisK gene (*Shk1*, syn. *os-1*). It contains an osmotic sensing domain, six 90-amino acid repeat motifs (also named HAMP domains), a kinase core and response regulator domains. Sequence analysis of this histidine kinase gene has shown either a deletion or single point mutations suggesting that these modifications may be associated with dicarboximide resistance and also involved in sclerotial development and virulence (Liu et al. 2009). In two field mutants of *S. sclerotiorum*, various mutations in the amino acid repeat region of the histidine kinase gene led to different degrees of sensitivity to dicarboximides and phenypyrroles (Alberoni et al. 2010). Further disruption of *Shk1* in laboratory mutants resulted in resistance to phenypyrrole and dicarboximide fungicides and increased sensitivity to osmotic and oxidative stresses. The *Shk1* mutant also showed a significant reduction in hyphal growth and was unable to produce sclerotia (Duan et al. 2013). In addition, the *S. sclerotiorum shk1* gene appears to be the orthologue of *B. cinerea Bos1* (Duan et al. 2014).

4- Demethylation-inhibitors fungicides (DMIs)

Among the inhibitors of sterol synthesis, only the class of inhibitors in 14α-demethylase (DMIs) is used in oilseed rape. Most are triazoles compounds (e.g. flutriafol, flusilazole, dinoconazole, metconazole, tebuconazole) but prochloraz (imidazole) and prothioconazole (triazolinethione) are also included in this class of fungicides. Their target site is the cytochrome P450 14α-demethylase (CYP51). Multiple mechanisms of resistance to DMI fungicides have been described involving i) mutations in the *Cyp51* gene, ii) *Cyp51* overexpression and iii) an upregulation of membrane transporters leading to an increase in the efflux of DMIs. A combination of these mechanisms has been reported in field isolates of *Mycosphaerella graminicola* in winter wheat displaying low to high resistance to DMIs (Leroux and Walker 2010) but not in *S. sclerotiorum*, according to current litterature.

In oilseed rape, some DMI fungicides are also very effective against secondary diseases. For example prochloraz for controlling light leaf spot (*Pyrenopeziza brassicae)* or flusilazole towards powdery mildew caused by *Erysiphe cruciferarum*. For over 20 years, DMI compounds were associated with carbendazim. Higher was the rate of carbendazim and more effective were the mixtures for controlling SSR (Kazmar et al. 2000). Unfortunately, owing to a restricted use of carbendazim before its withdrawal, all mixtures of carbendazim + DMI were no longer re-registered. However, tebuconazole and metconazole have remained on the market as solo products and prothioconazole has recently been

introduced in the panel of tools for diseases control in oilseed rape. Since 2007, DMIs tests were also added in the monitoring conducted every year. Up to now, no resistant strains of *S. sclerotiorum* have been detected. Sometimes, few isolates are detected as less susceptible at the discriminatory concentration used in the test, suggesting that a shift in sensitivity of *S. sclerotiorum* to DMI fungicides could occur. But under field conditions, no significant decline has ever been reported in SSR control.
Therefore, these fungicides are involved in resistance management strategies. In practice, tebuconazole or metconazole which have more irregular efficacy than boscalid or prothioconazole are recommended when sclerotina risk is low or moderate. This sclerotinia risk can be reduced by using biological control and a program combining biological control with *Coniothyrium minitans* in autumn and a complementary application of tebuconazole at the flowering stage has proved to be as effective as a preventive application of boscalid (Penaud and Michi 2009). DMIs are also good partners to associate with QoIs and SDHIs, for managing fungicide resistance.

5- Fungicides affecting specifically respiration

5.1- Inhibitors of mitochondrial complex III (QoIs)

QoI fungicides are able to inhibit mitochondrial electron transfer by binding to the Qo site (an outer ubiquinol oxidizing pocket) of cytochrome *bc1* complex or complex III. In most pathogens, resistance to QoIs (strobilurins) is conferred by a single point mutation in the mitochondrial cytochrome b gene *cyt b* resulting in peptide sequence changes that prevent fungicide binding (Gisi et al. 2002).
In French oilseed rape crops, the first QoI fungicide (azoxystrobin) was introduced in the early 2000s. Until now, no QoI resistance has yet been detected in field isolates of *S. sclerotiorum*, maybe due to a weak selection pressure or a negative fitness of resistant isolates. Azoxystrobin used to be applied in mixture with DMI fungicide rather than alone, which could have prevented resistance selection.
In most pathogens, the resistance to QoI fungicides is mainly due to the amino acid substitution from glycine to alanine at position 143 (G143A) in the *cyt b* which is also associated with a failure in disease control. In some fungi, QoIs resistance has never been recorded due to the presence of an intron inserted directly after codon 143. In those cases, G143A change prevents the splicing of the intron, leading to a deficient cytochrome b, which is lethal (Grasso et al. 2006). In a few cases of our monitoring, some sclerotia of *S. sclerotiorum* have failed to germinate and some have given colonies which have poorly grown on SHAM amended media with or without 1 mg/L of azoxystrobin. These field

isolates could be resistant and they need to be investigated for mutations and the intron structure of the *cyt b* gene that could explain why QoI resistant mutants of *S. sclerotiorum* are rare or unsuccessfully induced *in vitro* (Wang et al. 2014b). In some other fungi, other substitutions in the *cyt b* such as F129L and G137R have also been reported to confer a low to medium level of resistance, so that the efficacy of QoI fungicides is not affected in practice, but such substitutions have not yet been recorded in *S. sclerotiorum.*

5.2- Inhibitors of mitochondrial complex II (SDHIs)

The SDHI fungicides are able to inhibit the mitochondrial electron transfer from succinate to coenzyme Q by binding to the complex II or succinate dehydrogenase complex (SDH). The enzyme is a component of the inner mitochondrial membrane and consists of four nucleus-encoded proteins SDH-A, B, C and D. The binding site of ubiquinone is formed by residues of the subunits B, C and D and accounts for the target site of carboxamide SDHI compounds. Therefore, resistance to SDHIs is conferred by single point mutations in the *Sdh* genes encoding sub-units B, C or D. Many mutations have been reported in field populations of different pathogens (Sierotzki and Scalliet 2013). In France, the first strains of *S. sclerotiorum* resistant to boscalid were detected within two seasons after commercial use of boscalid in 2008. Although poor epidemics of SSR occurred, the number of detected strains resistant to boscalid has gradually increased since 2011 (Fig. 4).

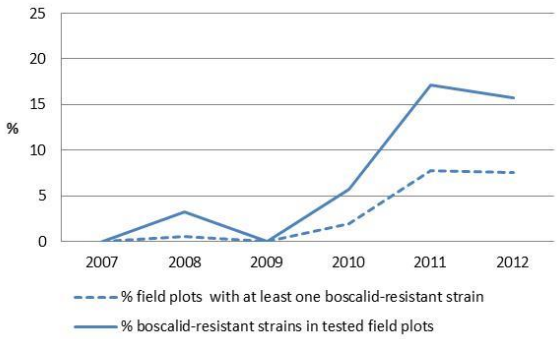

Fig. 4 Occurrence of boscalid - resistant strains of *S. sclerotiorum.* Results of monitoring conducted by ONPV – CETIOM – ANSES – INRA and chemical companies.

However no unusual control has yet been reported at the field level. Most field-resistant strains of *S. sclerotiorum* exhibit moderate resistance to boscalid and sequence analysis has revealed a point mutation in the *SdhD* gene, involving the replacement of histidine by arginine at position 132 (H132R) (Glättli et al. 2009). For fungi as *Alternaria alternata* or some strains of *B. cinerea*, the SdhD H132R

mutants have shown positive cross resistance between all novel SDHIs at low frequency which could be explained by a negative fitness cost (Leroux et al. 2010; Avenot et al. 2010). Other resistance mechanisms are suspected in resistant strains not bearing the SdhD H132R change (Walker unpublished). Recently, boscalid-resistant mutants of *S. sclerotiorum* have been induced *in vitro*, carrying a mutation in the *SdhB* gene (Wang et al. 2014c).

In the mids-2000s, boscalid was the first SDHI registered for the control of SSR on oilseed rape in France. Due to carbendazim resistance of *S. sclerotiorum* and the withdrawal of benzimidazole and dicarboximide fungicides, boscalid has quickly entered into widespread use. As it came on the market in 2007, the monitoring was then focused on this newly registered compound (Moinard et al. 2009). The method used for detecting boscalid resistant field isolates is based on classical mycelial growth on minimal agar medium with succinate as carbon source and amended with one single discriminatory dose of boscalid. This method is proved as reliable as Stammler's microtiter method (Stammler et al. 2007; Leroux unpublished; Wang et al. 2009).

Since its commercial use on French oilseed rape crops, boscalid was firstly applied alone once per year because only one application is effective to prevent oilseed rape infection if it is sprayed when the first petals are falling on leaves. Due to its efficacy, boscalid has become the most used active ingredient towards SSR which is applied alone and more and more in mixture with other fungicides. The most popular mixture is boscalid + metconazole and provides not only a high efficacy on SSR but also a better yield increase. When another application is sometimes required, alternation with other no cross resistant fungicides to limit the selection pressure is recommended. In order to develop anti-resistance strategies, new SDHIs are formulated with products carrying other modes of action. However mixtures with QoIs could present a risk to select double resistant strains as it has already been reported on other crops. Nevertheless this risk could be low if *S. sclerotiorum* is demonstrated as a pathogen with rare QoIs resistance. At last, the recent introduction of the mixture of fluopyram + prothioconazole provides another tool in resistance management. Though cross resistance between boscalid and fluopyram is suspected, prothioconazole offers a different mode of action with a high intrinsic activity against *S. sclerotiorum* strains populations so that SDHI resistant strains could be controlled.

6- Conclusions

Oilseed rape diseases are controlled as far as possible by host resistance. Due to a lack of cultivars resistant to *S. sclerotiorum*, chemical control remains the main way to control SSR in rapeseed. Usually, only one foliar spray is necessary for controlling the disease. As all the available fungicides are preventive with little or none curative effects on *S. sclerotiorum*, they need to be applied just before petals

fall on leaves, between 20 and 30 percent bloom. Sometimes the treatment can be repeated for controlling other diseases such as powdery mildew or black spot. Over 15 years, SSR was well controlled by benzimidazole fungicides used solo or in mixture with sterol demethylation inhibitors or dicarboximide fungicides. But because of intensive use, resistance to carbendazim emerged and generalized. Therefore, strategies for managing resistance were developed. They were firstly based on the use of the two remaining fungicides groups at full doses. It is also advised to choose the fungicide according to sclerotinia risk levels. When sclerotinia risk was high, the most effective dicarboximide fungicides were preferred to DMIs. Farming practices are also recommended such as to enhance crop rotation including less susceptible crops and to eliminate susceptible weeds. In addition, biological control using *Coniothyrium minitans* to reduce soil infestation can also help to limit the risk of sclerotinia and thus to avoid spraying or at least to allow the alternation of fungicides with different modes of action. After the withdrawal of dicarboximide fungicides, two new groups SDHIs and QoIs have fortunately been registered. But because they are single target-site fungicides a risk of developing resistance is to be feared. Therefore, strategies must be considered for preventing resistance. With one treatment per season, alternation strategy of different modes of action can only be promoted at the scale of the crop rotation. But when two treatments are required for controlling not only SSR but also other diseases, alternation of active ingredients with different modes of action can easily be implemented with the available three different modes of action. The strategy of fungicide mixtures with different modes of action is expanding in chemical companies in order to prevent or delay the emergence of resistance to their active ingredients prone to resistance such single target-site fungicides. But this strategy of mixtures requires suitable partners in appropriate rate to provide effective disease control without selecting for resistance. At last, the best strategy of resistance management of *S. sclerotiorum* should be to limit chemical control by improving sclerotinia prediction and to spray only when it is needed.

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