QoI Working Group

FRAC

FUNGICIDE RESISTANCE ACTION COMMITTEE

Impact of Position 143 Intron on Resistance Risk to QoI Fungicides in Some Pathogens

As described by FRAC and others, there are some pathogens in which resistance to the QoI fungicides has so far not been reported. There could be several reasons for this including effective anti-resistance strategies, lack of exposure of the pathogen to QoI fungicides or strong fitness penalties of mutant strains to survive or be competitive. However in the case of the rusts, belonging to the Basidiomycetes, treatment frequency with QoIs has been at least that experienced by the cereal powdery mildews where resistance rapidly arose. The strobilurin-producing Basidiomycete, *Strobilurus tenacellus* and *Mycena galopoda* exhibit 'natural resistance' to QoIs and the molecular mechanisms of this 'natural resistance' are known to be point mutations in the cyt b gene. This phenomenon was therefore investigated for *Puccinia* species (Grasso et al. 2006).

In different *Puccinia* species, the presence of an intron has been observed directly after the triplet GGT that encodes for glycine at position 143. In all rust species included in this study, as well as in *Alternaria solani* and *Pyrenophora teres*, the codon GGT at position 143 is located exactly at the exon/intron boundary and is likely part of the signal sequences essential for the recognition of the intronic RNA to be excised. The authors predict that a nucleotide substitution in codon 143 (GGT → GCT), which is two nucleotides upstream from the exon/intron junction, will strongly affect the splicing process, leading to a deficient cytochrome b. The substitution of guanine to cytosine obviously does not allow a proper pairing of the exonic nucleotides with the intronic IGS sequence in the pre-mRNA molecule. Therefore, this substitution will be lethal, and individuals carrying this mutation will not survive. As a consequence, it is concluded that resistance to QoI fungicides based on the G143A mutation is not likely to evolve in species such as rusts (*Puccinia* spp., *Uromyces appendiculatus*, *Phakopsora pachyrhizi*, and *Hemileia vastatrix*), *P. teres* and *A. solani*. The presence of such an intron has also been reported in *Monilinia laxa*, *Monilinia fructicola* (Miessner and Stammler 2010, Luo et al.,2010) and *Guignardia bidwellii* (Miessner et al. 2011). In the

fungal species investigated so far, the presence of an intron was conserved over all investigated isolates within a species, even after many years of high selection pressure by QoIs. There is only one exception, *Botrytis cinerea*, where two forms of the cytochrome b gene have been reported (Banno et al., 009). However, it cannot be excluded that mutations other than G143A conferring resistance may arise in upcoming populations selected by the use of QoI fungicides. For *A. solani* and *P. teres* the mutations F129L and/or G137R have been reported (Sierotzki et al. 2007, www.frac.info) as a mechanism for QoI tolerance. Both mutations are of minor importance, however, because they generally lead to lower resistance factors (www.frac.info) than the G143A mutation and it has been found that these two mutations have no, or only limited impact on the field efficacy of QoIs (Semar et al. 2007). The results give some confidence around the continued sustainability of disease control with QoI fungicides in pathogens containing an intron after codon 143 in the cytochrome b gene providing responsible resistance management practices are implemented.

Citations

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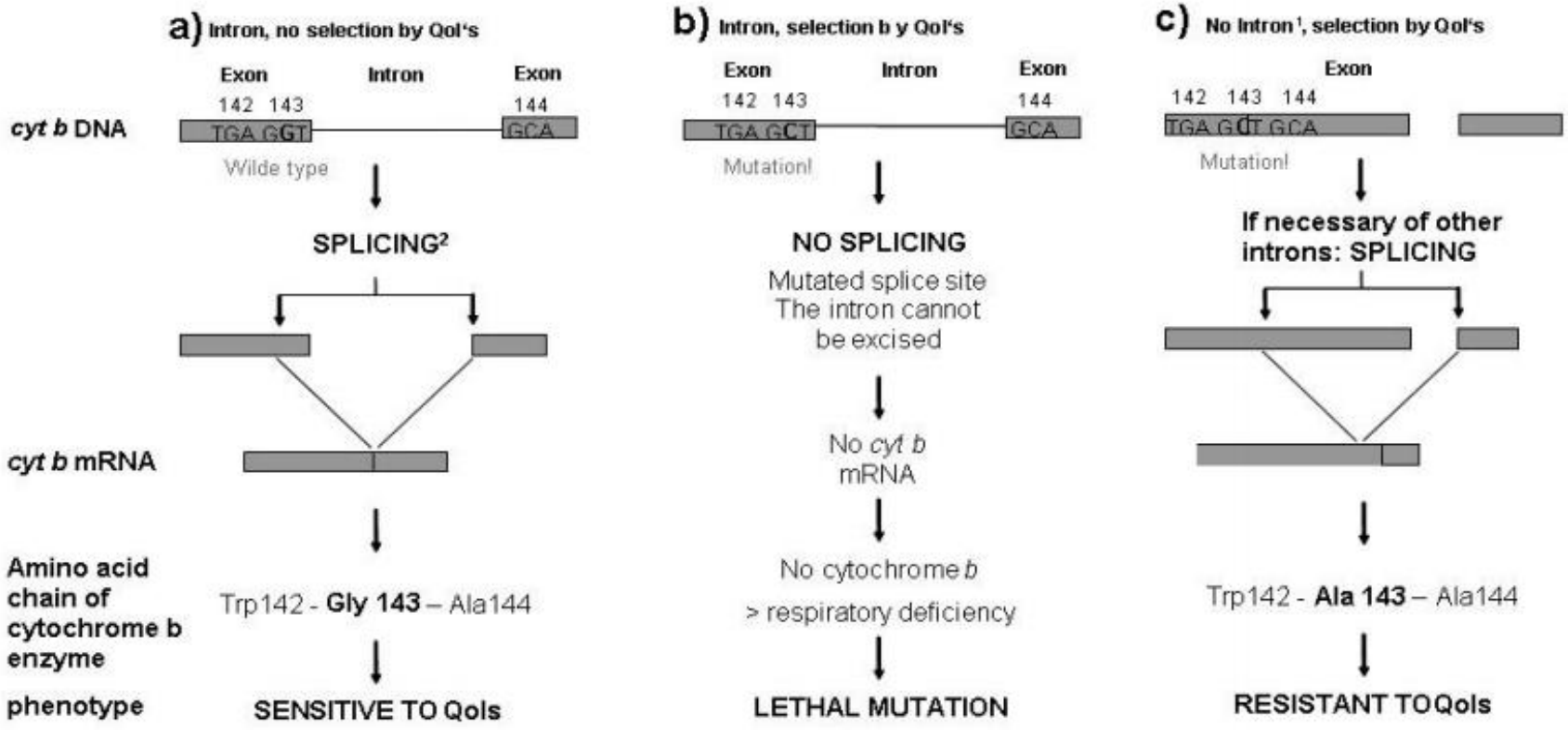
Effect of the mutation G143A on the processing of the *cyt b* gene pre-mRNA



¹or intron is located more than 6 bp after the aa position 143

²the splicing is a GTP mediated self-splicing process of the folded RNA (group II introns), which depend on the hairpin formation of the splice site

Source: www.frac.info

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