The rapid and highly sophisticated adaptation of asexual pathogens

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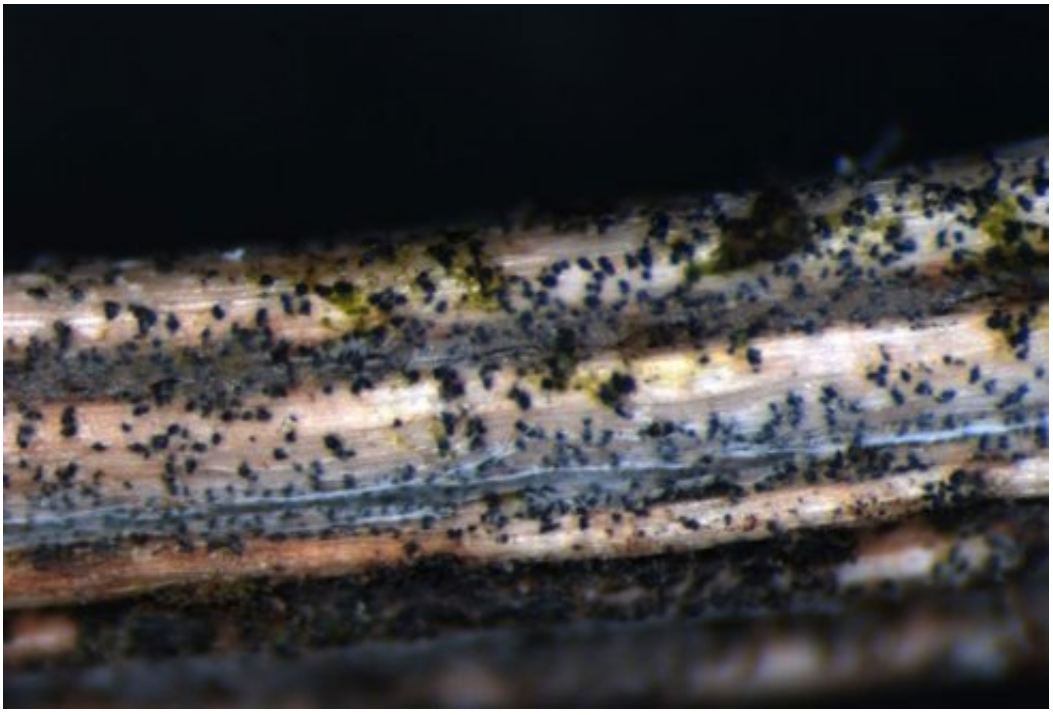

Credit: Wageningen University

How can fungi that only multiply vegetatively adapt so quickly to in the immune system of plants? The answer appears to lie in the way how the DNA of an asexual pathogen can rapidly adapt due to 'jumping genes' and also genetic modification that is prevalent in nature.

The battle between a pathogenic fungus and its host plant can be

compared to an infinite arm wrestling session that is ultimately won by the strongest genes. The fungus infects the plant, and the plant's immune system 'learns' how to recognise it. The pathogen then has to adapt to circumvent the immune system of its host so that it is no longer recognised… And the cycle continues again.

As asexual fungi have offspring that are identical copies of themselves, they cannot produce new combinations of properties in the same way that sexually reproducing organisms do. It has long been assumed that asexual pathogens are poorly able to evolve and fail to adapt well, which means that they are eventually intercepted by the hosts' immune system. This is not the case, however. Bart Thomma's research group at Wageningen UR has now discovered how the asexual pathogen Verticillium uses transposons, or 'jumping genes', to quickly and effectively adapt to new challenges.

Each genome consists partly of segments of foreign DNA (including viruses) that lead their own lives as 'intruders'. Some of these intruders can jump and these jumping genes can change the functioning of the gene in which they land.

In their research – published in *Genome Research* – Luigi Faino and Michael Seidl (led by Thomma) show that the pathogenic fungus Verticillium dahliae, which causes Verticillum wilt in plants such as the tomato, is able to activate its transposons. Doing so in a targeted way, namely only in specific parts of the genome which are important for the production of components that are required for plant infection, leads to accelerated evolution of genomic regions that are relevant to pathogenicity.

As a result, the fungus can adapt such that it is no longer recognised by the immune system of the host plant. Because Verticillium dahliae is able to pacify the transposons in other parts of the genome, the proteins

that create the most important basic cellular functions of the pathogen remain unchanged. The genome of Verticillium dahliae also shows that the pathogen sometimes kidnaps DNA from other organisms and inserts it into its own DNA to manipulate the host. Wherever transposons jump, there are fractures in the DNA which may allow foreign DNA to be inserted.

"You could call this natural genetic modification, and it is certainly a very clever move by the pathogen," says Bart Thomma. "Our research provides proof of accelerated evolution via transposons. It continues to be the same fungus with the same DNA, but by using the jumping genes and foreign DNA in a smart way it is able to adapt. As a result, the immune system of the host no longer recognises it and the pathogen can continue infecting the plant."

Another way that asexual pathogens are able to adapt is described in a recent publication in Current Opinion in Microbiology by Jasper Depotter and Bart Thomma, in which they describe the role of hybridisation in the evolution of pathogens. This involves the fusion of two varieties of fungi resulting in a new variety with twice as much DNA and a full set of new properties. The pathogen can therefore become more aggressive or may even result in an entirely new pathogen. This type of fusion generally occurs in micro-organisms, and is also possible for asexual pathogens.

DNA of the fungal pathogen Verticillium longisporum shows that it is a fusion of Verticillium dahliae with another unknown fungus. Thanks to this fusion Verticillium longisporum has more DNA, and thus a large number of extra properties, which allows it to infect Brassica varieties in a way that Verticillium dahliae is unable to do.

The DNA of Verticillium varieties uncovers the evolution of the fungus. For example, the genes show how Verticillium has re-adapted itself over

the years in order to survive in its host. Thomma: "The DNA clearly illustrates the importance and process of evolution."

More information: Luigi Faino et al. Transposons passively and actively contribute to evolution of the two-speed genome of a fungal pathogen, *Genome Research* (2016). DOI: 10.1101/gr.204974.116

Jasper RL Depotter et al. Interspecific hybridization impacts host range and pathogenicity of filamentous microbes, *Current Opinion in Microbiology* (2016). DOI: 10.1016/j.mib.2016.04.005

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