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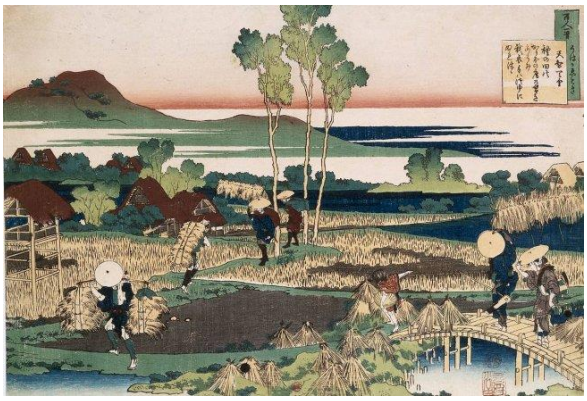
Vivienne Baillie Gerritsen

The moment life emerged on earth, the fight – or indeed the right – to multiply began. The notion of battle is particularly true for microbes such as bacteria, fungi and viruses, that may frequently depend on hosts to replicate. Over the aeons, the art of infection and its twin image immunity have both had plenty of time to devise intricate strategies, either to attack the enemy or to fend it off, respectively. If on the offensive, one way of diminishing an opponent's strength would be to confiscate an element of their protective gear. Let's say a soldier's helmet, or their walkie-talkie, or their bullet-proof jacket, or their gun. This is precisely one of the schemes pathogens have thought up to weaken their host's immune response – and there are many different ways of achieving it. As an illustration, the rice pathogen *Magnaporthe oryzae* is a filamentous fungus that secretes proteins, known as effectors, into the plant cell, whose role is to weaken the plant's immune response one way or another. Recently, researchers characterized two *M.oryzae* effectors that go straight into the nucleus of host rice cells. What do they do there? Tamper with the expression of genes involved in the plant's immune response. Their name: HTR1 and HTR2, for Host Transcription Reprogramming 1 and 2.



Rice Farmers

by Katsushika Hokusai (1760-1849)

Rice is the staple diet of 3 billion people around the globe. That is a lot of stomachs and the reason this particular cereal grain has become the focus of attention of botanists, farmers and agricultural engineers worldwide. Roughly 10 to 30% of rice crops are lost every year to disease – the most devastating being what is commonly known as rice blast and caused by the fungus *Magnoaporthe oryza*. 10% of the world's rice crops is enough grain to feed 60 million people for one year, and it is a lot to lose when 900 million people are already undernourished. Moreover, when crops are wiped out by disease, the price of rice increases making it all the more difficult for underdeveloped countries to cope. It is hardly surprising, then, that in the past century, scientists have been seeking ways to grow rice in a sustainable fashion.

How did this seed become such an important part of the global human diet? There are two main types of rice – one of African origin and one of Asian origin. When we talk about rice what we are really talking about is a seed that belongs to the grass species *Oryza sativa*, which is the rice of Asian origin. The African rice, *Oryza glaberrima*, was domesticated later and independently from its Asian counterpart, about 3,000 years ago, and is produced and consumed mainly on the African continent. *Oryza sativa*, however, is thought to have originated in China (although some argue India was the first country to cultivate rice) roughly between 13,000 and 8,000 years ago from where it spread westwards to the rest of Asia, reaching Europe in the 10th century, and then across to the Americas with the first European explorations. Today, there are about 40,000 different varieties of *O.sativa*, many of which are regional such as India's Basmati rice or Vietnam's and Thailand's Jasmine rice.

Rice blast – also known as rice seedling blight, neck blast or rice rotten neck – is caused by the filamentous fungus *Magnaporthe oryzae*. *M.oryzae* is not specific to rice but can also cause great damage – commonly called blight – to other agriculturally significant cereals such as wheat, barley, rye and maize. *M.oryzae*

attacks both the aerial part of its host plants and their roots, gradually killing off the cells as the fungus grows. Rice responds to infection in two ways. An initial immune reaction is triggered off following recognition of specific microbial patterns. That is to say, with time, rice plants have evolved sensors that are able to recognise molecular patterns that are specific to *M.oryzae*. A second immune reaction involves the recognition of specific fungal molecules, otherwise known as effectors, which *M.oryzae* secretes into plant cells as infection progresses.

M.oryzae has developed a rather sophisticated way of infecting its hosts. It begins by a non-invasive step where a spore, or conidium, adheres tightly to the leaf of a plant by releasing a sort of biological glue. Once adhesion has taken place, the conidium germinates to form a germ tube which transforms into a dome-shaped structure called an appressorium. Follows the invasive step: a second tubular structure, termed the penetration peg, grows from the appressorium and gradually eases its way into a leaf cell. At this point, the peg differentiates into invasive hyphae which grow and infect neighbouring plant cells by progressing through small existing intercellular canals, or plasmodesmata. During the whole process of infection, and from the very moment a conidium adheres to a leaf, the rice plant will be using all it has to ward it off. Concomitantly, *M.oryzae* will be secreting effectors to weaken the plant's immune response.

A special structure, known as the biotrophic interfacial complex (BIC), forms on *M.oryzae*'s invasive hyphae. It is from this structure that a variety of effector proteins are secreted into the live plant cell with one sole aim: dampen the host's surveillance system thereby weakening its global immune response. The nuclear effector proteins HTR1 and HTR2 are particularly intriguing in that, as they are secreted into the cell's nucleus, there is a fair chance that their role is to interfere with gene expression one way or another. Both effectors do indeed have a DNA-binding domain and could bind to the promoters of genes involved in the plant's immune response, thus modifying transcription. Studies have shown that their action may well repress the transcription of a large variety of genes – although it is not sure yet whether HTR1 and HTR2 repress transcription directly or whether they beat the plant cell's own transcription activators to their target promoters thus inhibiting their activity. Whichever way, the nuclear effectors HTR1&2 seem to be able to reprogram cell transcription so as to modulate the plant's immune response.

There is reason to believe that *M.oryzae* nuclear effectors probably restructure the immune response of other host plants in the same manner, such as the all-important crops of wheat, rye, barley or maize. This would offer the perspective of engineering plants that are susceptible to *M.oryzae* to make them more resistant to infection. However, by heightening a plant's resistance to one pathogen, you may be making it more susceptible to another. It is a tricky affair, demonstrating how important it is to understand in great detail how plant immunity and pathogen infection coincide. With the world's population rising as it is, solutions need to be found to supply enough cereal – at least by saving crops from being wiped out by disease. Especially as, despite the use of pesticides and rice cultivars that already carry resistance genes, like all pathogens, over the years, *M.oryzae* always finds ways to buck them.

Cross-references to UniProt

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HTR factor 2, *Magnaporthe oryzae (strain 70-15 / ATCC MYA-4617 / FGSC 8958)* (Rice blast fungus): G5EHQ6

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