

Prions can improve the health of fungal populations

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(Phys.org) -- Prions, infectious agents composed of proteins with a specific misfolded and transmissible 3D structure that causes diseases like BSE, can be present in fungal populations on a large scale, preventing the spread of fungal parasites and keeping the population relatively healthy. This remarkable discovery was published in this week's edition of the scientific magazine *PNAS* by scientists from Wageningen University, part of Wageningen UR. What makes the findings so extraordinary is that many scientists had previously assumed that prions had only a negative effect on the health of their 'host'.

"We have seen that prions can create a type of barrier between various fungal colonies, preventing them from making contact," says Wageningen University scientist Fons Debets. "In this way, the prions stop the fungi from passing on diseases among themselves."

Prions can lead to serious diseases in mammals, such as BSE and CJD. They are different versions of the normal proteins naturally prevalent in organisms due to the fact that their 3D structure is misfolded. Moreover, the altered 3D structure is infectious: When a deviating protein comes into contact with a normally folded [protein](#), the latter will take on the misfolded shape and become a [prion](#) too. Proteins that can form prions are found in people and other mammals as well as in fungi.

Many scientists had thus far assumed that prions only had negative effects on their 'host', explaining why they are rare in these organisms. The Wageningen research shows that this is not the case for a prion

found in a fungus.

Fungal hyphal threads have the tendency to come into contact with each other. This contact can be useful; for example, it can make the fungus more capable of extracting nutrition from the material in which it grows.

The contact also comes with risks, however. The contact between fungal threads can cause a [disease](#) from one thread to be transferred to another. Debets: “Fungi also have ‘diseases that are transferred via cellular contact, where one fungal thread infects the other.’”

Fungal threads cannot build up contact with all other fungal threads of the same fungal species. The contact is often blocked when the two fungal threads are overly different with regards to genetics and biochemistry. In such cases the cells that merge die off as they are mutually incompatible. This incompatibility is a rejection response that prevents the transfer of parasites.

The Wageningen scientists studied a local population of the fungus *Podospora anserina*. The ‘STD’ they studied was a certain parasite that infected no less than 40 per cent of the *podospora* fungal colonies in the Wageningen population.

Podospora has colonies that are able to produce prions (s) and those that cannot (S). The trait is genetically determined by a single gene. When an s-colony contains prions, it cannot make contact with S-colonies; they are incompatible.

The prions therefore create an extra ‘barrier’ between S and s; they reduce the chance of contact and, therefore, the likelihood of diseases being transferred between S and s-colonies.

This corresponds to observations in the field. Two-thirds of the fungal

colonies found are of the s-type and able to produce prions. Nearly all of these s-colonies were indeed shown to contain the prion.

Only one-third of the colonies are S-type. For an optimal functioning of the incompatibility, s and S should be present in almost equal amounts. This reflects the fact that when a type becomes more frequent, the benefits of the incompatibility for that type are reduced. In this case, the chance that a more prevalent type comes into contact with another colony of the same type is relatively large. The more prevalent s-type therefore meets few colonies that are incompatible, which means that the risk of infection is larger for the more common type. A rare type, on the other hand, increasingly comes into contact with a different, incompatible type and therefore has a lower risk of infection.

The s-type, which is most prevalent, is nonetheless most commonly infected with the fitness-reducing parasite. The fact that the prion is still so successful is illogical and the explanation can be found in another effect of the prion – the way it disrupts the sexual development of fungal spores in such a way that part of the S-spores die. As a result, the relative percentage of s-colonies in the population increases; and all those colonies have the genetic code to produce prions.

The scientists concluded that the prions, although selfish on the one hand, ensure that the s-majority does not infect the S-minority with parasites. This keeps the fungal populations relatively healthy.

More information: "High Natural Prevalence of a Fungal Prion", Alfons JM Debets, Henk JP Dalstra, Marijke Slakhorst, Bertha Koopmanschap, Rolf F Hoekstra & Sven J Saupe, www.pnas.org/content/early/2011/11/14/1205333109.abstract

Provided by Wageningen University

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