proteinspotlight
> ONE MONTH, ONE PROTEIN <

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# lure

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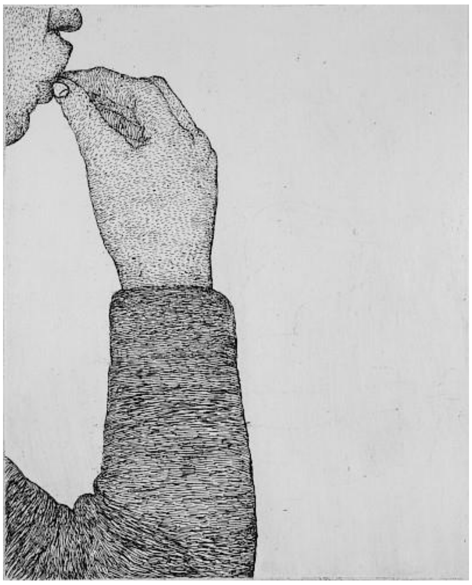
**Walking down a busy main street a few days ago, from the corner of my eye I saw a teenager rooting rapidly through a wallet he had just pulled out of a girl's backpack. Before I had registered what was going on, a young man approached me to ask where he could catch a bus. Flustered, I told him. In between times, the teenager and the stolen wallet had disappeared. Minutes later, I realised what had just occurred. The young man who had asked me about a bus had – successfully – diverted my attention from what his accomplice was doing. This is very similar to the kind of lure a plant pathogen known as *Phytophthora sojae* uses to confound soybean's immune response to infection. *P.sojae* secretes a protein known as XEG1 into the soybean plant where it can do significant harm. Soybean, however, reacts to the infection and muffles the effects of XEG1 thanks to a protein known as GIP1. To bypass this inconvenience, *P.sojae* promptly secretes a second protein – XLP1 – that soybean GIP1 mistakes for XEG1. XEG1 is then free to continue infection while the plant's immune system is tricked to attend to XLP1. This is a perfect example, in Nature, of two entities working together to confound a third.**



A Man Trying To Eat a Soybean by Art Hanson

Courtesy Estate of Art Hansen

When an infectious agent – such as the flu virus for example – invades our body, its goal is less to cause harm than to use our system to survive and proliferate. Over time, the human organism has elaborated a very complex and elaborate process, known as the immune response, to recognise what does not belong to it – like viruses, bacteria or parasitic worms for instance but also, unfortunately, organ transplants. When foreign bodies ("non-self") enter our body, our immune system fires off all sorts of molecules and pathways to ward them off. The immune reaction is not particular to humans: all living organisms have an immune system, however rudimentary, that will work around the clock, constituting a barrier that infectious agents usually give in to, or manage to overcome. How? One way is to shun the immune system by distracting it. *Phytophthora sojae* has achieved just this by producing a harmless protein that looks very similar to its infectious counterpart and tricks the immune system into believing it is. In this way, the infectious protein – also called the virulence factor – is not "caught" and can proceed to infect.

Soybeans have been part of human diets for ages. *Glycine max* is the domesticated variety of soybean grown today, and its closest living ancestor, *Glycine soja*, grows wild in China, Japan, Korea and Russia. Plants were grown by farmers in Eastern Asia at least 5,000 years ago it seems, perhaps even earlier. In Chinese mythology, Shennong or the "Divine Farmer", taught his people basic agricultural practises and was also known as the "Five Cereals God" – the five cereals being soybeans, rice, wheat, barley and millet. In the 17$^{th}$ Century, soybeans travelled westwards with the Dutch, Spanish and Portuguese traders who introduced the plants to India, Europe and the Americas and, in due time, to Africa. Boosted by WW2, by the 1960s the USA were exporting 90% of the world's soybeans. In the past decades, however, things have changed and today's top soybean exporters are Argentina (39%), the USA (37%), and Brazil (16%) while, ironically, the top importer is China (41%).

How does *P.sojae* infect soybean plants? *P.sojae* inhabits many types of soil. In wet conditions, it produces water-borne spores that are attracted to soybean roots. Once attached, they germinate and infect the plant tissues – first the roots and then the stem, eventually killing the entire plant. Plant cells are structurally similar to animal cells save for the existence of photosynthetic chloroplasts and large vacuoles of water in their cytoplasm, and a cell wall that surrounds the cell membrane. Cell walls are made out of polysaccharides (cellulose, hemicellulose and pectin) and constitute a first physical barrier to infectious agents. *P.sojae* begins by injecting virulence factors, notably XEG1, that degrade cell wall polysaccharides to ease infection. This action triggers off the plant's immune system, and the space between the plant's cell membrane and cell wall – known as the apoplast – turns into a field of vicious battle between soybean immune factors and *P.sojae* virulence factors.

More specifically, *P.sojae* secretes XEG1 into the soybean root. XEG1 is a xyloglucan-specific endoglucanase, or xylanase, that degrades the xylan backbone of hemicellulose in cell walls by hydrolysing xyloglucan and endo-beta-1,4-glucan into sugars. XEG1 presence ("non-self") in the soybean concomitantly triggers off the plant's immune system that reacts by dispatching GIP1 to the site of infection. GIP1 binds to XEG1; as a result, cell-wall degradation is checked and *P.sojae* virulence weakens. To divert the immune reaction and keep virulence high, *P.sojae* sends in XEG1 decoys: XLP1, an inactive glycoside hydrolase that is structurally similar to XEG1. *P.sojae* has evolved such that soybean GIP1 actually prefers XLP1 to XEG1. So, while GIP1 is busy courting XLP1, XEG1 is left free to degrade the plant's cell wall, and infection pursues.

Today, soybean plants constitute major crops worldwide, producing vegetable oil and protein used across the planet for human and animal consumption. Its cultivation has begun to pose serious environmental issues especially in Brazil where huge areas of the Amazon rainforest have been destroyed for soybean plantations, of which a whopping 80% is for feeding livestock. The more we know about how pathogens infect soybean plants the better we will be able to develop pesticides to save existing crops while causing the least harm possible to the environment. The earliest reference to an immune response dates back to 430 BC when the Greek historian and general Thucydides noted that people who had suffered from a disease could nurse the sick without falling ill a second time. It was not until the early 1900s, however, that microorganisms were stated to be the cause of infectious diseases, and it took a further 50 years before the theory of "self" and "non-self" emerged – the very key to the immune response.

## Cross-references to UniProt

Xyloglucan-specific endo-beta-1,4-glucanase 1, *P. sojae* (Soybean stem and root rot agent): G4ZHR2
Inactive glycoside hydrolase XLP1, *Phytophthora sojae* (Soybean stem and root rot agent): G4ZHR3
Probable aspartic proteinase GIP1, *Glycine max* (Soybean): I1JNS6

## References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