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.
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.

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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

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