When rain is pelting down and you have no protection, the umbrellas carried by other pedestrians suddenly become attractive. The thought of making one yours might even occur to you. In the same vein, a child who is being hit with a stick by another child might decide to grab the stick and hit their assailant with it in return. Taking possession of a means of defence that does not initially belong to you is not only a time-saver but also a sure way of dealing appropriately with the enemy, since it has been used just for that. The same kind of commerce exists between all sorts of organisms, though in a far more subtle manner. Many plants produce toxic compounds to fend off insects that feed off their sap. Recently, scientists discovered that a herbivorous species of whitefly – the sweet potato whitefly *Bemisia tabaci* – seems to have acquired an enzyme of plant origin. The enzyme in question modifies specific toxins – namely phenolic glycosides or PGs – so as to render them harmless. These particular toxins are secreted by plants precisely to ward off herbivorous insects. By acquiring the plant enzyme, *B. tabaci* has also acquired the means to detoxify PGs and therefore take advantage of plants that produce them. And the enzyme is? Phenolic glucoside malonyltransferase 1 (MAT1), that we shall call BtPGMT1.

Plants and insects have been living together on this planet long before humans were even a blip on evolution’s radar. For the best part of 400 million years, they have been fine-tuning their genomes to live side by side without either of them losing too much ground. In a way, it is a constant race to arms. One will find ways to bypass the enemy’s defence while the other sharpens its knives. This is what seems to have happened between the sweet potato whitefly *Bemisia tabaci* and the plants it feeds off. Based on phylogenetic analysis and comparing key domains of the enzyme BtPGMT1 in *B. tabaci* with that of hundreds of plant genomes, it is likely that *B. tabaci* stole the phenolic glucoside malonyltransferase gene, MAT1, from a plant that
Insects are harmful to plants in two ways. First, they feed on their sap – which is what carries water and nutrients to every part of the plant – usually leaving in their wake the all too familiar honeydew on the plants’ leaves. Second, they invariably inject pathogens such as viruses into the plants. In response, plants have designed strategies to keep insects off them, one of which is the synthesis of phenolic glycosides, or PGs. PGs are a combination of one sugar (glycone) group with one functional non-sugar (aglycone) group. As secondary metabolites, they are not involved in the plant’s normal growth and development but, depending on their functional group, are used to ward off noxious organisms or sometimes even to lure them – as plant pigment flavonoids cunningly evolved to attract insects for pollination for example.

Certain PGs are toxic to insects in that they can strongly affect their growth, development and behaviour – although it is not known how exactly. While feeding off plant sap, insects will inadvertently ingest PGs that end up in their gut cells where they will exert their toxicity. Surprisingly, PGs can also be toxic to plants! This is where BtPGMT1 comes in. This particular enzyme catalyzes the transfer of a malonyl group from malonyl coenzyme A to PGs. Such a transfer, or malonylation, confers various roles to PGs, one of which is detoxification. In plants, PGs detoxified in this way are stored in vacuoles until the toxic form is activated – presumably by demalonylation – and released upon insect invasion. When B.tabaci attacks a plant, the plant reacts by secreting toxic PGs into its sap, which end up in the insect’s gut cells. There, they are detoxified by the insect’s personal copy of BtPGMT1.

It is the detoxifying action of the plant’s BtPGMT1 that will have seduced – understandably so – B.tabaci so many millions of years ago. If it could acquire a copy, then it would be able to neutralise the PGs it ingests and feed off the host plant unperturbed. None of this happened with clear intention on the insect’s behalf naturally. As for all similar genetic events, chance played the biggest part and adaptation did the rest. Today, plant PGs are little threat to B.tabaci, tomorrow its host plants will have developed yet another means of defence. Such is the driving force of evolution. In the meantime, B.tabaci still causes damage to crops worldwide – by feeding off their phloem while injecting pathogens, all of which causes huge economical loss. Now that scientists know more about BtPGMT1 and where it originally came from, it should help to develop ways of fighting B.tabaci – perhaps by tampering with BtPGMT1’s gene to silence it. Thus, in a way, playing the same game and taking back what never really belonged to the whitefly in the first place.

Cross-references to UniProt

Phenolic glucoside malonyltransferase 1, Bemisia tabaci (Sweet potato whitefly): P0DUQ3

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