Acyl-CoA-binding proteins help plants “degrease” adversities

Serving as the building blocks for triacylglycerides and membrane phospholipids, fatty acids are essential in all forms of life. Given their chemical inertness, fatty acids need activation into a more water-soluble form (as acyl-CoA esters), prior to their utilization in complex lipid assembly, cellular energy generation, or conversion to other metabolites. To participate in such physiological processes, acyl-CoA esters must be delivered from the site of *de novo* biosynthesis, namely the plastids in plants, to various subcellular locations such as the cytoplasm, endoplasmic reticulum, plasma membrane and peroxisomes, where they bind to specific members of the acyl-CoA-binding protein (ACBP) family. By this reversible lipid-protein association, acyl-CoA esters are protected from hydrolytic degradation. In turn, the conformations of cellular membranes and their associated enzymes are preserved from the harmful detergent-like activities of acyl-CoA esters, which structurally resemble soaps (salts of fatty acids).

Apart from the well-recognized ubiquitous functions of ACBPs as lipid “chaperones”, evidence is accumulating in higher plants that ACBPs influence the survival of these sessile organisms upon biotic invasion or environmental stress. Terrestrial plants appear to have benefited from the evolution of diverse ACBP members (e.g. six each in Arabidopsis and rice as representative dicot and monocot model species, respectively) which may uniquely exhibit a broad selectivity for different ligands, some of which are important messengers in plant signaling. For instance, arachidonic acid is a pathogen-secreted lipid and is naturally absent from plants, but could the recognition of its derivative (arachidonyl-CoA) by Arabidopsis ACBP3 act to switch on defensive measures? It was shown that Arabidopsis mutant plants without ACBP3 were more tolerant to the fungal necrotrophic pathogen *Botrytis cinerea* but less tolerant to the bacterial biotrophic pathogen *Pseudomonas syringae*, and *vice versa* for transgenic Arabidopsis ACBP3-overexpressors. These observations suggested the involvement of ACBP3 in multiple defense-related signaling in response to distinct pathogens.

At sub-zero temperatures, freezing poses severe damages to plants, which have innate ability to generate phosphatidic acid (PA) as a warning signal arising from the breakdown of membrane phosphatidylcholine (PC). In Arabidopsis, ACBP6 binds PC and induces the expression of phospholipase D? which produces PA upon freezing stress. Hence, ACBP6-overexpressing Arabidopsis was more freezing-tolerant than the wild type. In another study, higher freezing tolerance of Arabidopsis occurred following the depletion of ACBP1, which binds both PC and PA. By protein-protein interaction with phospholipase D?1, ACBP1 may promote the generation of harmful PA that deteriorates membrane structures during freezing and post-freezing recovery. This deleterious effect could be alleviated in the *acbp1* mutant. Besides cold spells, plants are stressed by water shortage, during which they face the dilemma of breathing in carbon dioxide for photosynthesis while losing water with their stomata open. By overexpressing ACBP2, transgenic Arabidopsis plants were made more drought-tolerant resulting from the better regulation of

stomatal closure *via* a signaling pathway mediated by the hormone abscisic acid. Interestingly, Arabidopsis ACBP2 and its homolog ACBP1 bind heavy metal ions in addition to lipids. Accordingly, the overexpression of ACBP1 and ACBP2 in transgenic Arabidopsis promoted tolerance to lead (II) and cadmium (II), respectively.

As Arabidopsis ACBPs have been identified to be of great importance under plant stress, research is ongoing to characterize their homologs in commercial crops, which may become promising targets for genetic manipulation of agronomically important traits to boost crop productivity under adversity.

Shiu-Cheung Lung *and* ***Mee-Len Chye***
School of Biological Sciences
The University of Hong Kong
Pokfulam, Hong Kong, China

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