The mode of action and the genetic basis of resistance in the case of benzoxazinones

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Abstract

Several grass species release precursors of benzoxazinones, a class of potent allelochemicals. Although the synthesis pathways and degradation processes of these compounds have been well characterized, the molecular mode of action by which they inhibit plant growth has remained elusive. Here, we present a combination of biochemical, molecular, and genomic analyses, by which we show that 2-amino-*3H*-phenoxazin-3-one (APO) and its methoxylated analog 2-amino-7-methoxy-*3H*-phenoxazin-3-one (AMPO) act by binding to the catalytic unit of histone deacetylases (HDACs), inhibiting these enzymes. APO and AMPO activity leads to genome-wide changes in histone acetylation, altering chromatin configuration and transcriptional profiles of the target plant, which ultimately results in a slow-down of growth. The high evolutionary conservation of the targeted pathway - APO and AMPO inhibit even human histone deacetylases - raises two questions: Is there specificity at the enzymatic and/or at the histone level? And, how do some plant species manage to be tolerant or even resistant to these allelochemicals? To answer the first question, we use biochemical and mass spectrometry analyses of HDACs and acetylated histones, as well as chromatin immunoprecipitation targeting specific acetylated histone residues. To understand the evolution of resistance, we employ a genomics approach, making use of the natural variation spectrum and the genomic resources of the model plant *Arabidopsis thaliana*, to map resistance-promoting alleles. Altogether, I will present the latest results of our attempt to comprehensively understand benzoxazinone-mediated allelopathy between plants.

Keywords: Allelopathy, benzoxazinones, DIBOA, DIMBOA, chromatin, histone

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