A distinctive voice for ethylene signaling in hypoxia

Alcohol dehydrogenase (ADH) is a key enzyme in fermentation and anaerobic metabolism where it contributes to ethanol production from acetaldehyde derived from pyruvate. During the anaerobic response in plants, transcription of the ADH gene is induced, transcripts are more stable and ADH protein levels increase. Indeed, ADH was identified as an anaerobic-induced polypeptide in several screenings. Furthermore, it has been known for some time that ethylene is an important mediator of the response to hypoxia in many plant systems.

‘…ethylene is involved in ‘late’ induction of ADH, but something else is responsible for the earlier induction’

Hsiao-Ping Peng et al.1 have now reached a crossroads in the investigation of the role of ethylene in the induction of ADH. Treatment with amino-oxacetic acid (AOA), an inhibitor of ethylene synthesis, resulted in a notable decrease in the hypoxic induction of an ADH::GUS transgene after 28 hours of hypoxia treatment; this effect could be reversed by the addition of ACC (ethylene precursor). Similar results supporting ethylene induction where observed when ADH mRNA was analysed in northern blots. Interestingly, these effects of AOA and ACC need several hours of the hypoxia treatment to occur. For example, after four hours in hypoxia, AOA treatment does not decrease the amount of mRNA for ADH; on the contrary, at these shorter incubation times, AOA seems to increase the amount of mRNA for ADH and ACC decreases it, which is the opposite effect of what happens after 28 hours.

Results with ethylene mutants come to the same conclusions: ethylene is involved in ‘late’ induction of ADH, but something else is responsible for the earlier induction. The case is similar to recently reported examples of gene induction during germination, where ethylene is known to regulate the expression of several genes, such as genes encoding cysteine proteinase or β-glucanase enzymes. However, ethylene is produced at the time of radicle emergence or thereafter, when these genes are already induced. Now the question remains: what other signals contribute to the induction of gene transcription during the hypoxic response?


Emilio Cervantes
ecervant@gugu.usal.es

Studying the historic migrations of the Irish potato famine pathogen using ancient DNA

Phytophthora infestans, the causal agent of potato and tomato late blight, is one of the most devastating crop pathogens known because it spreads quickly and the infection cannot be controlled effectively using genetic resistance or fungicides. Although its center of origin is believed to be in the Americas, a series of migrations has led to a worldwide distribution. The first documented migration occurred in the 1840s, and led to the Irish potato famine. Disease reports suggest that the pathogen reached the eastern USA in the early 1840s, from where it migrated to Western Europe. Previous work concerning this migration has involved drawing inferences based on genotyping isolates collected primarily from the late twentieth century. Now, J. P. Ristaino and colleagues1 have used genotyping of P. infestans from infected potato leaves collected as early as 1847 to define the history of the pathogen better.

Outside of its center of diversity, central Mexico, P. infestans is primarily asexually reproductive, owing to the requirement of two mating types for the formation of sexual spores. As a result, in most locales, one or a few clonal lineages exist. Genotyping of extant isolates collected worldwide has revealed that before the second known round of migration, which occurred in the 1970s–1990s, one clonal lineage, US-1, predominated outside of Mexico. Based on this distribution and the absence of other documented migrations since the 1840s, it was suggested that US-1 was probably responsible for the Irish potato famine. To test this hypothesis, Ristaino et al. isolated DNA from P. infestans-infected herbarium samples and sequenced a PCR-amplified 167 bp variable mitochondrial DNA (mtDNA) fragment to genotype the ancient isolates. Four major mtDNA haplotypes are known, type Ib being unique to US-1. Surprisingly, none of the ten herbarium samples successfully analyzed (collected in Europe and in the USA from 1847–1928) displayed the Ib haplotype, suggesting that US-1 was not solely responsible for the early migration. Additional analysis is necessary to identify more fully the mtDNA haplotype(s) present in these samples.

This is the first time that herbarium specimens have been successfully used to track the migrations of a plant pathogen, and the results reveal that the past of P. infestans is more sordid than previously believed.

‘…the past of P. infestans is more sordid than previously believed’

The real significance of this work is that it implies that additional evolutionary events must have occurred between this early migration and that in the 1970s–1990s. Although disease reports do not suggest as much, it is possible that US-1 gained its worldwide distribution by the 1970s via an intermediate migration event and successful outcompetition of the previously distributed isolate(s). Alternatively, US-1 might have been distributed with several other lineages at the time of the famine, and over time it outcompeted the others. Further work with these and additional herbarium samples should shed more light on these migrations.


Matthew R. Willmann
willmann@fas.harvard.edu