

Dear Faculty, Postdocs, Students, and Friends:

**You are cordially invited to attend a special seminar presented by the
Institute for Integrative Genome Biology**



**Dr. Jesse Woodson
School of Plant Sciences
University of Arizona**

Title:

**“Control of cellular degradation pathways by
chloroplast signaling”**

DATE: Friday, May 21, 2021

TIME: 12:00 pm PST

MEETING ID: 963 2886 4096

PASSCODE: 929813

Host: Dr. Katie Dehesh

Abstract: Chloroplasts constantly experience photo-oxidative stress while performing photosynthesis. This is particularly true under abiotic stresses that lead to the accumulation of reactive oxygen species (ROS). While ROS leads to the oxidation of DNA, proteins, and lipids, it can also act as a signal to induce chloroplast degradation, cell death, and nuclear gene expression. Although the mechanisms behind ROS signaling from chloroplasts remain mostly unknown, several genetic systems have been devised in the model plant *Arabidopsis* to understand their signaling properties. One system uses the plastid ferredoxin-NADP+ oxidoreductase two (*fc2*) mutant that conditionally accumulates the ROS singlet oxygen (1O_2) leading to chloroplast degradation and eventually cell death. Here we have mapped mutations that suppress chloroplast degradation in the *fc2* mutant (*fts* mutations) and demonstrate that they affect loci encoding chloroplast proteins predicted to be involved in post-transcriptional gene expression and nucleotide metabolism. These *fts* mutations were shown to lead to broadly reduced chloroplast gene expression, impaired chloroplast development, and reduced chloroplast stress signaling. 1O_2 levels were uncoupled to chloroplast degradation, however, suggesting that these mutations also affect ROS signaling pathways. Together these results suggest plastid gene expression (or the expression of specific plastid genes) is a necessary prerequisite for chloroplasts to activate 1O_2 signaling pathways to induce chloroplast degradation and/or cell death.