Australian National University

Combining whole genome resequencing and database-driven metabolic phenocopy analysis accelerates discovery of novel *Arabidopsis* photorespiratory mutants

Wednesday 18 December 2013 1 – 2pm

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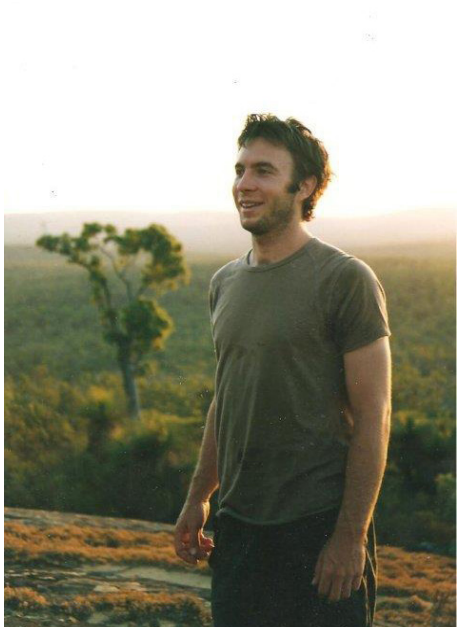
Slatyer seminar room R.N. Robertson Building (Bldg. 46), Linnaeus Way, ANU



Forward genetic screening remains promising for the isolation of novel photorespiratory mutants. However, general photorespiratory screens frequently isolate mutants affected in well-known photorespiratory genes and cost-effective methods are therefore required to discriminate these from novel mutants. We demonstrate how metabolic phenotype recognition and whole genome resequencing may be combined to accelerate the discrimination of novel photorespiratory mutants. For metabolic phenotype recognition, we present the *MetabolomeExpress PhenoMeter* - a public database-driven web tool enabling researchers to statistically match the metabolic phenotypes of mutants to a reference database containing metabolic phenotypes for a variety of known photorespiratory mutants. As a demonstration, we used the *PhenoMeter* to classify twenty new putative photorespiratory mutants isolated by chlorophyll fluorescence-based screening. Whole genome resequencing confirmed that PhenoMeter results were highly predictive of mutations affecting photorespiratory enzymes. The sequenced mutants displayed phenotypes consistent with photorespiratory impairment such as elevated CO_2 compensation points and photoinhibition and visible leaf chlorosis upon transfer from high- to low-CO_2. However, the phenotypes of two mutants were not explained by mutations in any known photorespiratory genes. One (*18-44H2*) appears to represent a novel class of photorespiratory mutant with an unusual metabolic phenotype. The other (*17-6E4*) displayed a phenotype highly similar to *glu1* mutants with impaired ferredoxin-dependent glutamine oxoglutarate aminotransferase (Fd-GOGAT). Notably, this phenotype was explained by near absence of GLU1 protein despite no mutation in the *GLU1* locus and normal expression of *GLU1* mRNA. The *glu1*-like phenotype of *17-6E4* therefore appears to be caused by decreased translation and/or stability of GLU1 protein.

Presented by

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