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Characterization and Purification of Protein Factor that Binds to Protein-binding Region on rbcL Promoter in Maize(*Zea mays*)

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In maize, the plastid proteins of light-grown seedlings bound to R3 DNA region(-230 to -418 from ATG) of the *rbc*L promoter(Lee and Sim, 1996). In order to identify the binding proteins to the R3 fragments, we carried out gel retardation assay. From these results, we observed that R3 region of the *rbc*L promoter binds to high salt extracts from chloroplast of maize to form multiple complexes. The major complexes are designanted A to F. Binding protiens were fractionationed by ammonium sulfate precipitation. The proteins forming the complexes A, B and C were precipitated between 0 - 35 % saturation, and the proteins for D, E and F were preciptated between 35 - 73 % saturation. To further purify the proteins forming the complexes D, E and F, we carried out heparin-agarose column chromatography. Fractions eluted between 0.3 and 0.4 M KCl were enriched in proteins that form complexes D, E and F. The abundances of complexes D, E and F decreased in the competition experiment with the unlabeled R3′ sequences(-324 to -343 from ATG) of the *rbc*L promoter, suggesting that these complexes are formed by interaction between the plastid proteins and the R3′ sequences.

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The FIN2 Gene of *Arabidopsis thaliana* Controls a Subset of the Responses in the Phytochrome A-Specific Signalling Pathway

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The photoreceptor phytochrome A (PHYA) mediates various plant responses to far-red (FR) light. While the molecular and biochemical nature of PHYA have been extensively characterized, the downstream signaling pathway remain largely unknown. Here, we report isolation and characterization of a genetic mutation that defines a branch of the PHYA-specific signaling pathway in *Arabidopsis thaliana*. The mutation was isolated in a screening for mutants that show seedling phenotypes insensitive to FR, but not to red (R) light. Genetic analyses revealed that the mutation named *fin2-1* is a single recessive nuclear mutation and is not allelic to the *phyA* mutation. Phenotypic analyses of the *fin2* mutation revealed that the mutant seedlings showed defects in hypocotyl growth inhibition, apical hook opening, cotyledon expansion, and blocking of greening in FR light but not in R light. However, other PHYA responses such as seed germination, agravitropic hypocotyl growth, and anthocyanin accumulation were not or only slightly affected. Furthermore, RNA gel blot analysis revealed that the *fin2* mutation greatly impaired expression of the *CAB* gene but slightly that of the *CHS* gene in FR light. Immunoblot analysis showed that PHYA is expressed normally in the mutant. These results show that *FIN2* controls a branch of a downstream component in the PHYA-specific signal transduction pathway. In conjunction with the previous model on the PHYA-signalling, we propose that the *FIN2* gene is involved either in the Ca^{2+}/CaM mediated branch or upstream of both of the Ca^{2+}/CaM and cGMP braches of the PHYA signaling pathway.