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BRSSINAZOIE-RESISTANT *GULLIVER* MUTANTS OF *ARABIDOPSIS* DEFINE NOVEL PATHWAYS IN BRASSINOSTEROID SIGNALING CASCADES

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To better understand the brassinosteroid signaling pathways, we used a brassinosteroid biosynthetic inhibitor brassinazole (Brz) to isolate mutants that are resistant in the light. We have identified four loci from either EMS-mutagenized or T-DNA activation-tagged populations, and named *gulliver (gul)* since they all display the characteristic phenotypes, such as elongatedand long petioles. Double mutant analysis revealed that both *gul1-D* and *gul2* suppress BR insensitive *bri1* and *bin2/dwf12-D* dwarf phenotype, but have slightly reduced level of endogenous BRlevels. *gul1-D*seedlings exhibit reduced sensitivity to both BL and Brz while *gul2* responds to BL with reduced sensitivity, but hypocotyls of Brz-treated *gul2* are much longer than those of wild type. Microarray experiment revealed that the expression of many BR signaling components was increased in *gul2* background. We have identified *GUL1*and *GUL2* via a map-based cloning approach and *GUL3* by TAIL-PCR. *GUL1* encodes a receptor kinase and *GUL2* is a pivotal component in the light signaling pathway. Functional analysis of GUL1 and GUL2 provide new insights into not only the BL signaling pathway but also the cross talk between brassinolide and light signaling cascades.