



**Supplementary information, Figure S3** Comparison of our BRI1-KD-BIM complex with the recently reported BRI1-KD structures. Related to Figure 6.

(A) Sequence alignment of BRI1 and BAK1 fragments containing helices  $\alpha$ F,  $\alpha$ G and  $\alpha$ H with conserved residues highlighted in yellow. Secondary structural elements of the BRI1-KD-BIM complex (green and marine blue) and the reported BRI1-KD structure (4OAC, pink and red) are shown above the alignment, and that of two reported BAK1-KD structures (PDB IDs: 3UIM and 3TL8) are shown in light blue and light yellow below the alignment, respectively.

(B) Electron density map for the ATP binding site in our BRI1-KD-BIM complex. The  $2F_o-F_c$  electron density map (contoured at  $1.5 \sigma$ ) is shown in blue mesh. The AMP-PNP molecule and residues interacting with the  $\gamma$ -phosphate are highlighted in sticks, while water molecules are shown as red spheres.

(C) Comparison of the  $\gamma$ -phosphate orientation in three nucleotide-bound BRI1-KD structures. The reported structures of BRI1-KD/ATP complex (4OAB) and BRI1-KD/AMP-PNP/Mn<sup>2+</sup> complex (4OA9) are colored in salmon and violet, respectively, and our BRI1-KD-BIM/AMP-PNP complex follows the color scheme in Figure 2B except that the bound AMP-PNP molecule is shown in pale green. Distance between the  $\gamma$ -phosphate and catalytic Asp1009 in respective structure is indicated.

(D) Crystal structure of SeMet-labelled BRI1-KD-BIM complex. The anomalous difference Fourier map of the SeMet derivative (contoured at  $3.0 \sigma$ ) is shown in red mesh. Notably, residue Met1117 is indubitably located on helix  $\alpha G''$ .

(E) Model for BR signaling with preformed BRI1-BAK1 heterooligomers. In the absence of ligand, approximate 7% of BRI1 on the plasma membrane of live Arabidopsis root epidermal cells constitutively heterodimerizes with BAK1 (Bucherl et al., 2013). Upon ligand binding, the formation of BR-BRI1-BAK1 ternary complex results in transphosphorylation of BRI1 and BAK1, enabling downstream signaling. When  $K_1 > K_2$ , the total concentration of complexed BRI1 in the presence of BR ( $[BRI1-BAK1]$  plus  $[BR-BRI1-BAK1]$ ) is greater than that of  $[BRI1-BAK1]$  in the absence of BR, and when  $K_1 = K_2$ , the total concentrations of the BR-dependent hetero-oligomers will be similar to that of the preformed BRI1-BAK1 dimer in the absence of BR.