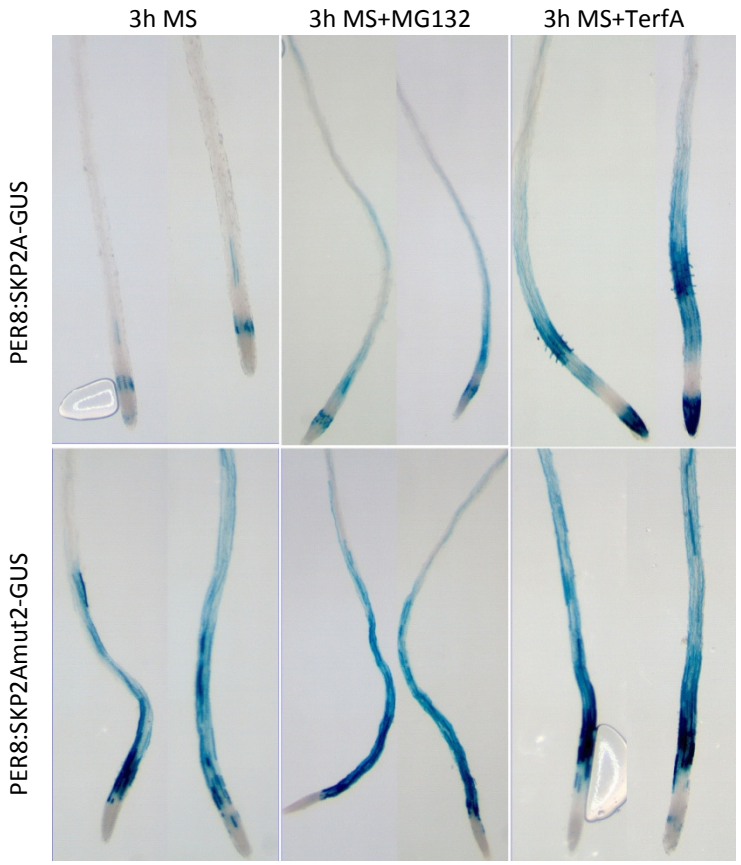
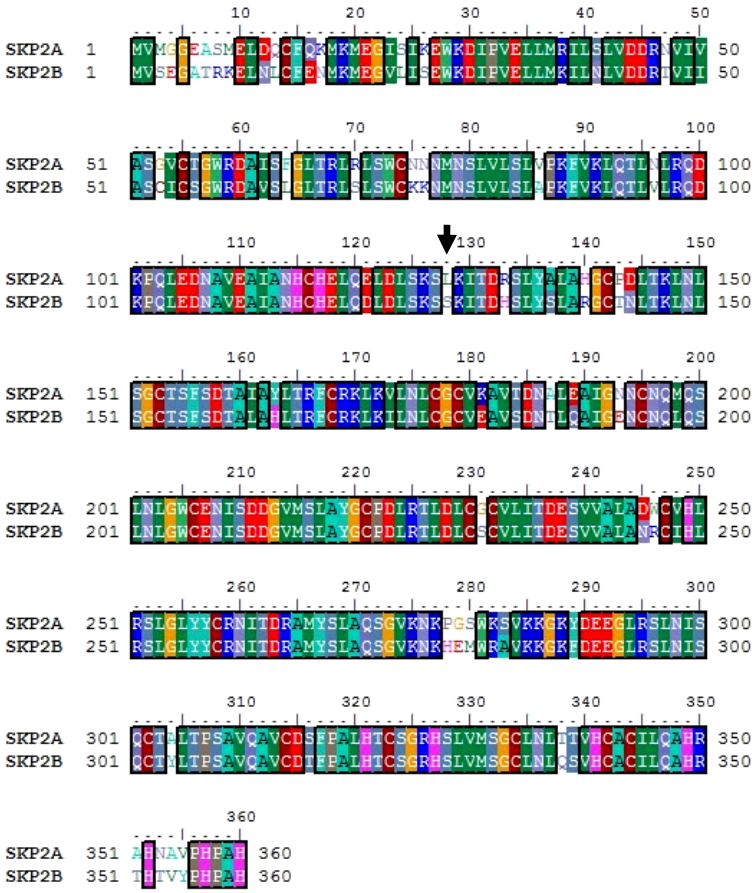
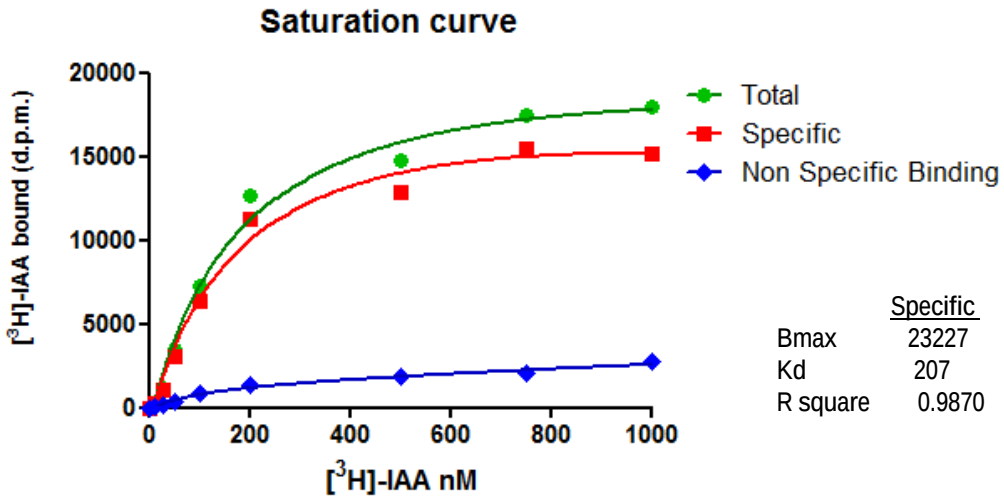


Supplemental Figure 1. SKP2A-GUS protein accumulated in response to the proteasome inhibitor MG132 or Terfestatin A (TerfA).
Five days old pER8:SKP2A-GUS and pER8:SKP2Amut2-GUS transgenic seedlings were transferred to MS plates containing 15 µM of estradiol to induce the expression of the chimeric proteins during 16 hours . After induction, these seedlings were incubated during 3 hours in a MS medium alone (3h MS) or containing 40 µM of MG132 (3h MG132) or 50 µM of Terfestatin A (3h MS+TerfA) and then stained for GUS activity. Bar scale corresponds to 1 mm.

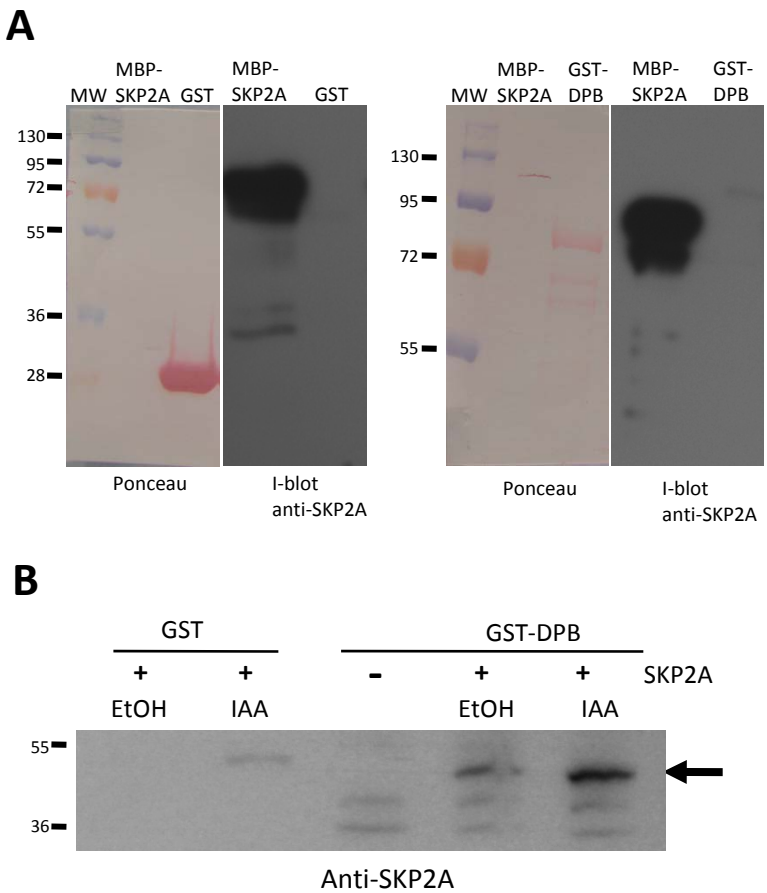


Supplemental Figure 2. Alignment of SKP2A and SKP2B protein sequences.

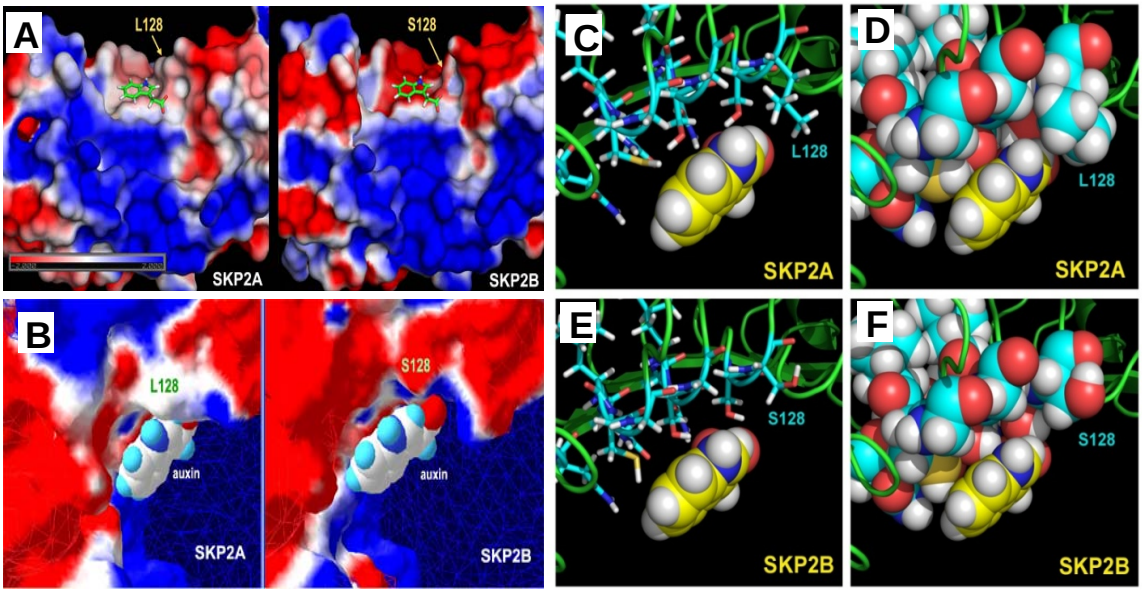
Arrow indicates the position 128 that corresponds to a leucine in SKP2A and to a serine in SKP2B.



Supplemental Figure 3. Scatchard analysis.
Pull-down reactions were carried out with MBP-SKP2A in the presence of 25, 50, 75, 100, 200, 500, 750 or 1000 nM [^{3}H]-IAA. The retained [^{3}H]-IAA in the amylose beads after three washes was measured by scintillation counting. Each value is the mean of three independent measures. To determinate the non-specific binding (NSB), similar experiments were carried out with 100 µM of cold IAA. The Specific binding was calculated by subtracting the NSB from the total. The Kd, B-maxima and R square were calculated using nonlinear regression analyses using the Graph Pad Prism5software.



Supplemental Figure 4. SKP2A interacts with DPB *in vitro*.
A) Anti-SKP2A does not recognize the GST or GST-DPB proteins. The MBP-SKP2A (0,01 µg), GST (4 µg) or GST-DPB (1 µg) were separated in s SDS-PAGE and analyzed by immunoblotting (I-blot) with affinity purified IgGs anti-SKP2A. Arrow indicates the MBP-SKP2A. MW is molecular weight.
B) GST-DPB, but not GST, also interacts with His-SKP2A. To check that the interaction showed between GST-DPB and MBP-SKP2A was not due to the presence of the MBP tag in the protein, we constructed a version of 6x-histidine tag of SKP2A using the pQE system (Qiagen) that was expressed in bacteria. The interaction was analyzed in the presence of ethanol (solvent control) or 1 µM of IAA. As shown in the figure, the IAA enhances the interaction between GST-DPB and His-SKP2A. Arrow indicates the His-SKP2A protein



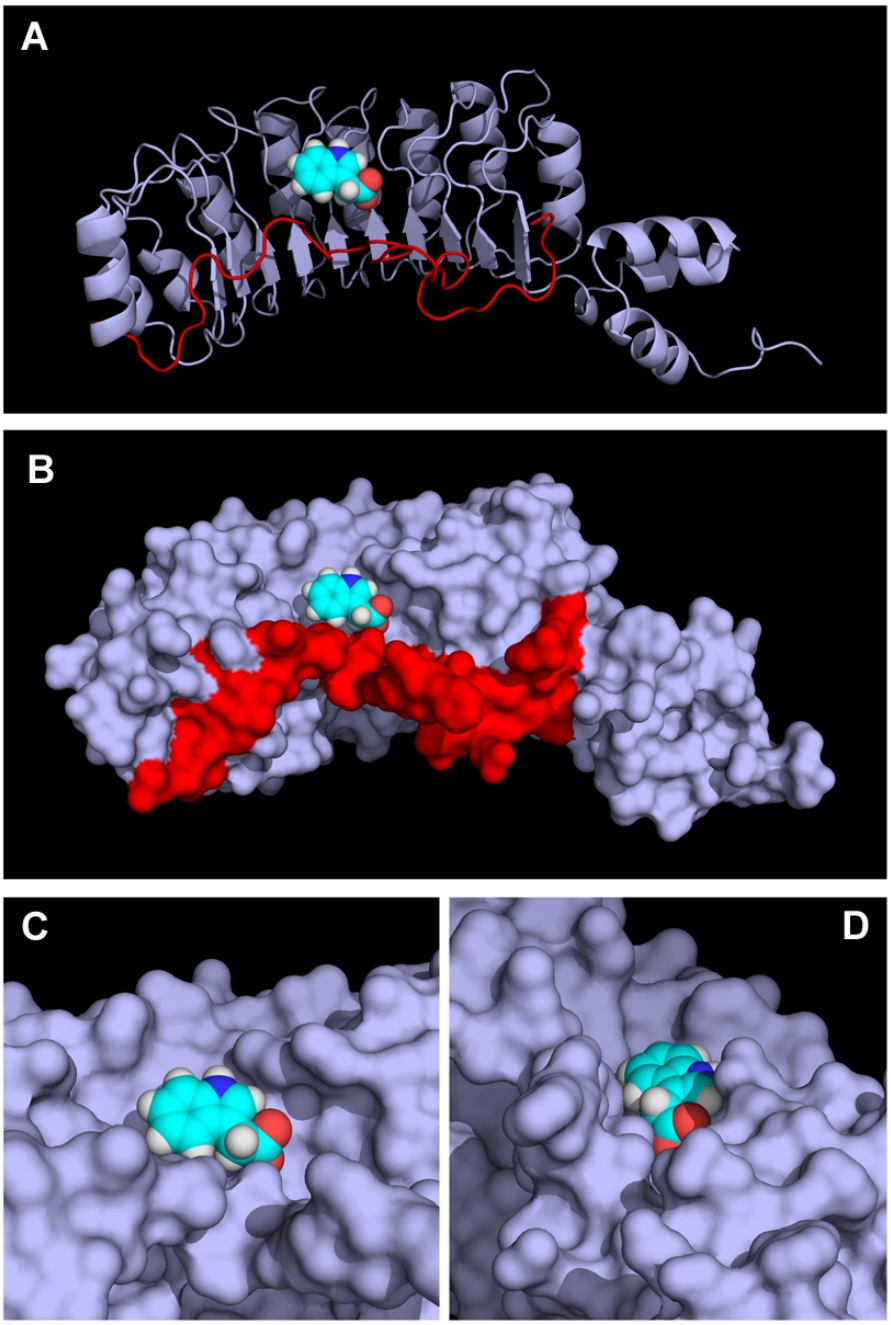
Supplemental Figure 5. Geometry of auxin at its binding site and steric effect of residue 128.

A) Electrostatic potential of SKP2A and SKP2B**.** Molecular surface of SKP2A and SKP2B with the PB electrostatic potential mapped onto the surface color-coded from positive +2.0 *kT/e* (blue) to negative -2.0 *kT/e* (red). Close-up views of the concave side of the LRR domain with auxin molecule docked shown as a stick model. Arrows point out the surface of residue 128 (leucine in SKP2A or serine in SKP2B). Notice that in SKP2B the non-polar moiety of auxin molecule is surrounded by a negative potential surface that should hinder the binding.

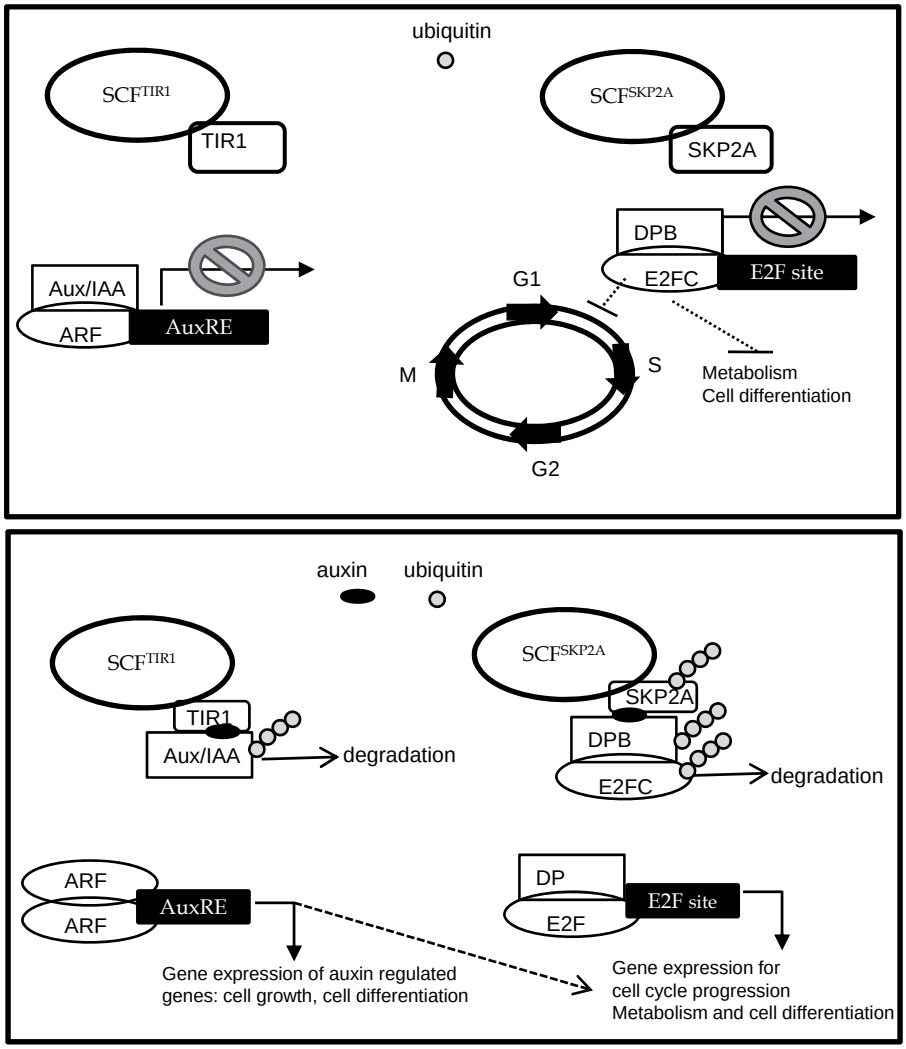
B) Steric effect of L128 in SKP2A and S128 in SKP2B on the binding of auxin shown as a CPK model within the protein Coulomb potential environment.

C, D) Auxin molecule shown as a CPK model at its binding site in SKP2A with protein residues (cyan frame) participating in the interaction represented as sticks (C) and CPK spheres(D) .

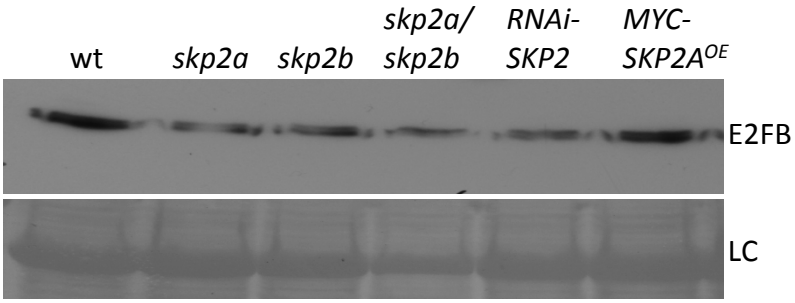
E, F) Same representation for SKP2B. Leucine at position 128 in SKP2A makes a “wall” that shapes the pocket. Serine at that position in SKP2B removes this wall and leaves a flatter surface at the binding site.



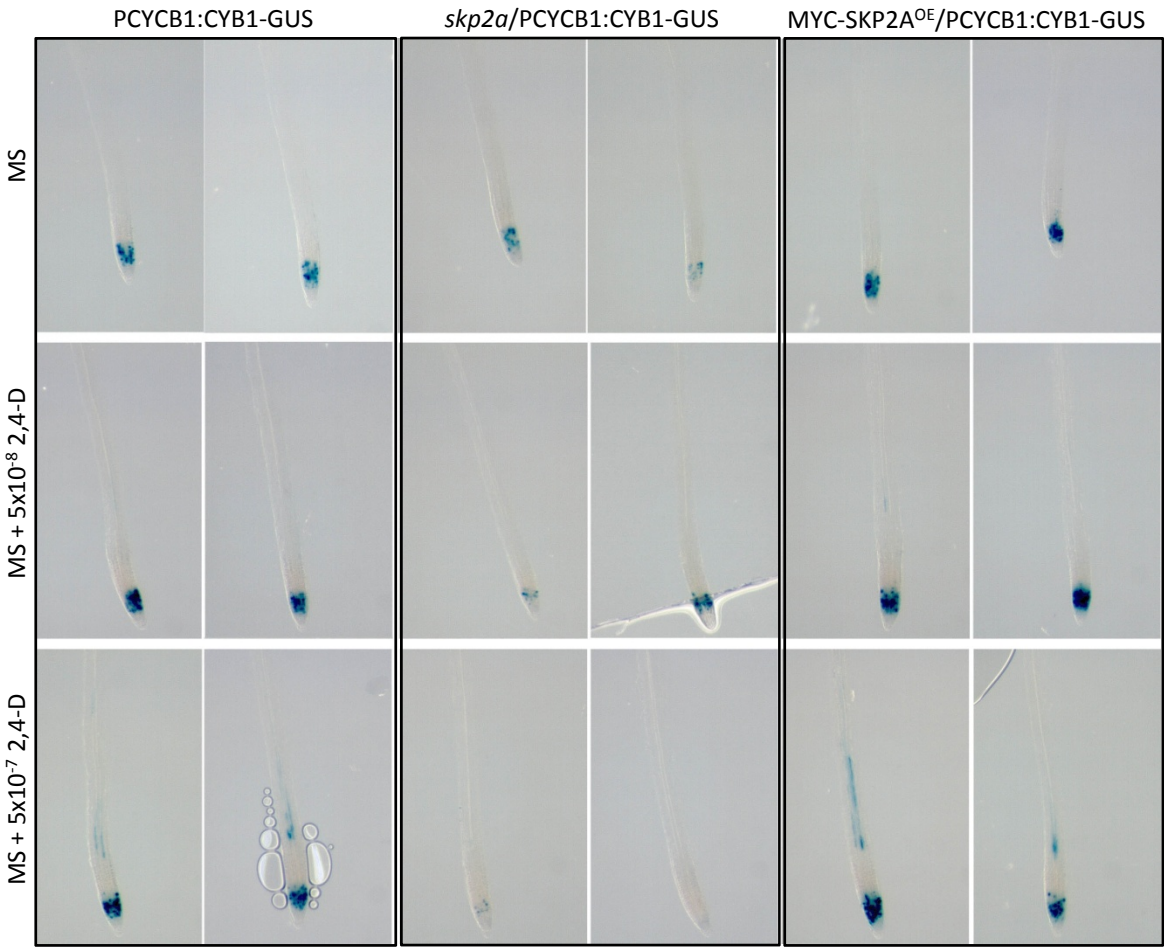
Supplemental Figure 6. Topography of the auxin binding pocket in complete SKP2A. A) Ribbon diagram of complete SKP2A with auxin shown as a CPK model at its binding site. The C-terminal coil composed of residues 295-332 is colored red. **B)** Surface representation of A. **C,D)** Close-up views of the binding pocket in the surface of complete SKP2A. The inclusion of the C-terminal coil segment adds an inner wall to the concave surface of the LRR solenoid that shapes the auxin binding pocket at its innermost part.



Supplemental Figure 7: Model of SKP2A-auxin regulation.
In a low auxin concentration, the SCF^{SKP2A} is found inactive. When the level of the hormone increased, auxin activates the SCF^{SKP2A} complex (promoting the formation of the complex or activating the E3 ligase activity) and promotes the Aux/IAA degradation by the TIR1, allowing the ARF-mediated gene expression. In this situation, the SCF^{SKP2A} ubiquitinates DPB and E2FC, promoting their degradation by the UPS. This will liberate the repression mediated by E2FC-DPB, allowing the activation of cell cycle, metabolism and cell differentiation genes. This, together with the ARF-mediated gene expression, will lead to the cell division and/or cell differentiation. In addition, to limit the over-function of the SCF^{SKP2A}, SKP2A is also ubiquitinated and degraded.



Supplemental Figure 8. E2FB stability is regulated by neither SKP2A nor SKP2B. Immunoblotting analyses of wild type (wt), *skp2a* or *skp2b* mutants (Ren et al, 2008), double *skp2a/skp2b* mutant, an RNAi-SKP2 line (Ren et al., 2008) and the overexpressing MYC-SKP2A line (Jurado et al., 2008), using the anti-E2FB IgGs. Total protein as extracted from 6 days old plants in TAP buffer containing proteases inhibitors and 50 µM of MG132. The proteins were separated in a SDS-PAGE and transferred to a PVDF membrane, which was analyzed by immunoblotting using IgGs against E2FB (kindly provided by Lieven De Veylder). LC is the loading control, corresponding to the Ponceau stained blot.



Supplemental Figure 9. *SKP2A* is necessary to control cell proliferation in response to auxin.

Five days old seedling carrying the CYCB1;1 degron fused to the GUS protein under the control of the CYCB1;1 promoter (PCYCB1:CYB1-GUS), *skp2a*/PCYCB1:CYB1-GUS or MYC-SKP2A^{OE}/PCYCB1:CYB1-GUS seedlings were transferred to MS medium alone or MS medium containing 5x10^{-8} M or 5x10^{-7} M of 2,4-D for 8 hours. Afterwards, these seedlings were stained for GUS activity. Representative pictures of root meristems of each genotype were chosen.