Supplementary Table 1. Interaction of COI1 and TIR1 with SCF subunits

	TIR1	COI1
ASK	**Direct Interaction: (positive)** The Arabidopsis TIR1-ASK1 complex was crystallized, revealing interaction with ASK1 at the N-terminal F-Box domain (Tan *et al.*, 2007). **Stability: (positive)** Steady-state levels of TIR1 increase in the presence of ASK1 (Dezfulian *et al.*, 2016). **Auxin Response: (positive)** The *ask1-1* mutant has a reduced auxin response (Gray *et al.*, 1999).	**Direct Interaction: (positive)** The Arabidopsis COI1-ASK1 complex was crystallized, demonstrating that ASK1 interacts with COI1 at the N-terminal F-Box domain (Sheard *et al.*, 2010). **Stability: (positive)** *ask1-1* mutants have reduced levels of COI1 in immunoblot analysis, using α-COI1; whereas ASK1 being present in a yeast pBridge expression system leads to an increased COI1 protein levels (Yan *et al.*, 2013). **JA Response: (positive)** The *ask1-1* mutant has reduced expression levels of *vsp2* in the presence of MeJA (Yan *et al.*, 2013).
CULLIN	**Direct Interaction: (positive)** Immunoprecipitates and subsequent western blot analysis provide evidence that TIR1 interacts with CUL1 through TIR1's N-terminal H1 helix (Yu *et al.*, 2015). **Stability: (negative)** Inhibition of CUL1 interaction leads to increased TIR1 protein levels based on comparing histochemical staining in tagged GUS mutant and GUS wild-type lines (Yu *et al.*, 2015). **Auxin Response: (positive)** The *cul1-7* mutant is less responsive to 2,4-D (synthetic auxin) treatment compared to the wild type and also degrades IAA1-LUC at a slower rate (Gilkerson *et al.*, 2009).	**Direct Interaction: (?)** The interaction is assumed to be indirect, via the SCF^{COI1} complex. Although, it cannot be ruled out that COI1 may interact directly with CUL1, through its F-Box H1 helix (Zheng *et al.*, 2002b). CUL1 is able to be co-immunoprecipitated with COI1 (Xu *et al.*, 2002). **Stability: (positive)** The *axr6-1/+* and *axr6-2/+* CUL1 heterozygous mutants show reduced COI1 protein levels (Yan *et al.*, 2013). **JA Response: (positive)** When treated with MeJA, AtCUL1 mutants, *axr6-1* and *axr6-2*, show a reduced expression of JA responsive genes compared to wild-type lines (Ren *et al.*, 2005).
RBX	**Direct Interaction: (negative)** Indirect Interaction with RBX1 through CULLIN1 of the SCF^{TIR1} complex (Gray *et al.*, 2002). **Stability: (?)** We could not find any substantial evidence that RBX1 changes the TIR1 protein levels. **Auxin Response: (dynamic)** As part of the SCF complex, RBX1 facilitates substrate ubiquitination (Zheng *et al.*, 2002a). However, overexpression of *RBX1* leads to a reduced auxin response (Gray *et al.*, 2002).	**Direct Interaction: (negative)** COI1 indirectly interacts with RBX1 when assembled into the SCF^{COI1} complex (Zheng *et al.*, 2002b). **Stability: (?)** We could not find any substantial evidence that RBX1 changes the COI1 protein levels. **JA Response: (positive)** When induced, *AtRbx1*(RNAi) mutants show a reduction in the expression of JA responsive genes (Xu *et al.*, 2002). In *RBX1* overexpression lines, the JA response is unaffected (Gray *et al.*, 2002).

ALF4	**Direct Interaction: (negative)** Indirect interaction through the SCF^{TIR1} complex. ALF4 interacts with RBX1 in Y2H and co-immunoprecipitation experiments (Bagchi *et al.*, 2018). **Stability: (negative)** In an *alf4* mutant, there is an increase of TIR1 abundance (Bagchi *et al.*, 2018). **Auxin Response: (negative)** ALF4 reduces the ability of RBX1 and E2 to ubiquitinate substrates (Bagchi *et al.*, 2018).	
CAND1	**Direct Interaction: (negative)** The interaction is indirect through CULLIN (Zheng *et al.*, 2002a). **Stability: (negative)** Diminishing CULLIN-CAND1 interaction increases SCF^{TIR1} abundance (Zhang *et al.*, 2008). **Auxin Response: (dynamic)** The loss-of-function mutants display a reduced auxin response, this is thought to be based on dynamic cycling being necessary for proper SCF function (Zhang *et al.*, 2008).	**Direct Interaction: (negative)** The interaction is indirect through CULLIN (Zheng *et al.*, 2002a). **Stability: (no effect)** There is no reduction in the COI1 protein levels in *cand1-1* mutants (Feng *et al.*, 2004). **JA Response: (positive)** The *cand1-1* mutant has some JA response defects, whereas the *coi1-1 cand1-1* double mutant is fully insensitive (Feng *et al.*, 2004).
JAZ		**Direct Interaction: (positive)** The COI1-ASK1-JAZ1 degron peptide complex was crystallized with either JA-Ile or COR (Sheard *et al.*, 2010). Furthermore COI1 has shown interaction with a number of JAZ proteins although interaction with JAZ7 and JAZ8, which lack the canonical Jaz motif, has not been reported to our knowledge (Pauwels and Goossens, 2011; Shyu *et al.*, 2012). **Stability: (no effect)** When COI1 protein is co-expressed with JAZ1, it does not lead to increased COI1 protein levels (Yan *et al.*, 2013). **JA response: (negative)** JAZ protein interaction with COI1 leads to their ubiquitination and subsequent degradation, which prompts the JA response (Chini *et al.*, 2007; Thines *et al.*, 2007).

Aux/IAA	**Direct Interaction: (positive)** TIR1-ASK1 complex was crystallized with Aux/IAA substrate peptides (Tan *et al.*, 2007). **Stability: (?)** We did not find experimental evidence for an effect of Aux/IAAs on the stability of TIR1 proteins. **Auxin Response: (positive)** Aux/IAA protein interaction with TIR1 leads to their degradation and de-repression of ARFs, enabling the auxin response (Tan *et al.*, 2007).	
MED25		**Direct Interaction: (positive)** MED25 immunoprecipitates with COI1 (An *et al.*, 2017). **Stability: (positive)** The *med25-4* mutant displays reduced COI1 levels at the promoters of JA responsive genes (An *et al.*, 2017). **JA Response: (positive)** *med25-4* mutants have a reduced ability to degrade JAZ proteins in the presence of JA-Ile (An *et al.*, 2017).
Ubiquitination	**Direct Interaction: (positive)** TIR1 was demonstrated to be polyubiquitinated (Stuttmann *et al.*, 2009). **Stability: (negative):** Proteasome inhibitors stabilize TIR1 protein (Stuttmann *et al.*, 2009; Yu *et al.*, 2015). **Auxin Response (negative)** Once TIR1 is associated into the SCF^{TIR1} complex results in eventual proteasomal degradation of TIR1 (Stuttmann *et al.*, 2009; Yu *et al*., 2015).	**Direct Interaction: (positive)** COI1 is thought to be ubiquitinated on the K297 residue, leading to its degradation (Yan *et al.*, 2013). **Stability: (negative)** COI1 is thought to be ubiquitinated on K297, leading to its degradation. The 26 proteasome component mutant, *rpt5a-4,* has increased COI1 protein levels (Yan *et al.*, 2013). **JA Response: (negative)** The 26 proteasome component mutant, *rpt5a-4,* shows an enhanced JA sensitivity (Yan *et al.*, 2013).
S-Nitrosylation	**Direct Interaction: (positive)** Biotinylated TIR1 was detected in a biotin switch assays strongly supporting that a TIR1 residue (candidates being C140 or C480) has SNO modification (Terrile *et al.*, 2012). **Stability: (no effect)** Mutating the C140 residue did not result in a decrease in TIR1 protein stability (Dezfulian *et al.*, 2016). **Auxin Response: (positive)** An amino acid change of C140A led to abolishment of interaction with Aux/IAA proteins (Terrile *et al.*, 2012).	

Rubbylation	**Direct Interaction: (negative)** Rubbylation occurs on CUL1 in the SCF complex (Gray *et al.*, 2002). **Stability: (no effect)** RUB modification does not seem to affect the stability of the SCF complex (del Pozo *et al.*, 2002). **Auxin Response: (dynamic)** An *axr1* mutant, defective in RUB activation, lacks the ability for RUB modification of CUL1 and leads to decreases in the auxin response (del Pozo *et al.*, 2002). Overexpression of *RBX1* leads to hypermodification of CUL1 by RUB, which disrupts its function and leads to an impaired auxin response (Gray *et al.*, 2002).	**Direct Interaction: (negative)** Rubbylation occurs on CUL1 in the SCF complex (del Pozo *et al.*, 2002; Gray *et al.*, 2002). **Stability: (no effect)** RUB modification does not seem to affect the stability of the SCF complex (del Pozo *et al.*, 2002). **JA Response: (positive)** *axr1* mutants are slightly JA insensitive, indicating that RUB modification is necessary for the JA response (del Pozo *et al.*, 2002; Xu *et al.*, 2002). The *axr1-3* mutant shows a reduced JA root growth inhibition (Schwechheimer *et al.*, 2002).
COP9	**Direct Interaction: (negative)** COP9 interacts with SCF^{TIR1} subunits and specifically interacts with AtCUL1 in a yeast two-hybrid analysis (Schwechheimer *et al.*, 2001). CSN forms a complex with CULLIN (Cavadini *et al.*, 2016). **Stability: (positive)** A COP9 signalosome subunit mutant, *csn2-5*, has reduced TIR1 protein levels (Stuttmann *et al.*, 2009). **Auxin Response: (positive)** Plants with reduced COP9 show a reduced auxin response (Schwechheimer *et al.*, 2001).	**Direct Interaction: (negative)** CSN forms a complex with CULLIN (Cavadini *et al.*, 2016). COP9 interacts with SCF^{COI1} (Feng *et al.*, 2003). **Stability: (no effect)** We could not find any experimental evidence demonstrating a change in COI1 protein abundance. In CSN mutants, the integrity of the SCF complex was not affected (Schwechheimer *et al.*, 2002). **JA Response: (positive)** Plants with antisense *CSN5* show a reduced JA response (Schwechheimer *et al.*, 2002). Genome expression profiles demonstrate that functional COP9 is crucial for the JA response (Feng *et al.*, 2003).

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