

Figure S1. The double mutant *hae hsl2* is totally deficient in abscission. (a) Inflorescence of wild type Arabidopsis plant. Red arrow points to first siliques with absciced floral organs. (b) *hae hsl2* double mutant. Orange and yellow arrows indicate silique positions 16 and 20, respectively, counting from the top of the inflorescense. (c) Attached floral organs of the *hae hsl2* double mutant are turgid at least until position 16.

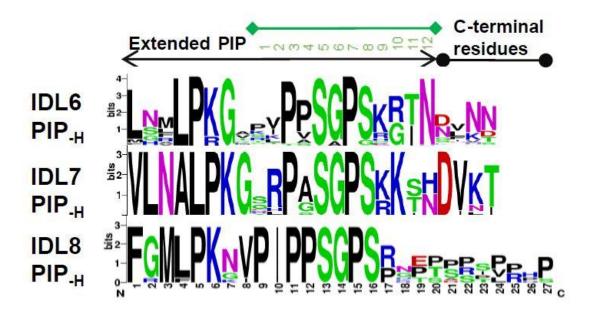


Figure S2. Conserved residues in propeptides of angiosperm IDL 6.8 orthologues numbered from the start of the extended PIP motif to the C terminal end. Sequences were aligned using Muscle, and consensus sequences were generated with logo@compbio berkeley edu. The PIP residues are numbered 1.12 Compared to IDA and IDLA 1.5 IDL 6.8 lack the His (in position 11 The Arg His Asn (of IDA has been shown to interact with the correceptor SERK 1.22.

LRR	Arabiopsis thaliana HAE LxxLxxLxLxNxLSGx-IPxx-LGx		Arabidopsis thaliana HSL2 LxxLxxLxLXxNxLSGxIPxx-LGx		Elais guineensis EgHSL2 LxxLxxLxLxxNxLS-GxIPxxLGx
1	88 LPSLHSLSLYNNSINGS-LSADDFDT	97	IRTLINITLSQNNLNGTIDSAPLSL	121	IPTLRHLSLAWNDLGGGLPAADVAL
2	113 CHNLISLDLSENLLVGS-IPKSLPFN	122	CSKLQNLILNQNNFSGKLPEF-SPE	118	CSGLEVINISNNFFV-GRIPEFSGE
3	138 LPNLKFLEISGNNLSDT-IPSS-FGE	146	FRKLRVLEL ESN LFTGEIPQS-YGR	142	FPRLRLLDLSGNNFS-GDIPPSFGR
4	162 FRKLESLNLAGNFLSGT-IPAS-LGN	170	LTALQVLNL <mark>N</mark> GNPLSGIVPAF-LGY	166	FPSLRVLSLY <mark>S</mark> NLIS-GRIPSFLAN
5	186 VTTLKELKLAYNLFSPSQIPSQ-LGN	190	LTELTHLDLA <mark>y</mark> isdpspipst-lg <mark>n</mark>	190	LTELIEFNLGENPFRSGSLPPEIGN
6	211 LTELQVLWLAGCNLVGP-IPPS-LSR	219	LSNLTDLRLTHSNLVGEIPDS-IMN	215	LTKLEVLWLPFVNLV-GEIPDSVGN
7	235 LTSLVNLDLTFNQLTGS-IPSW-ITQ	243	LMLLENLDLAMNGLTGEIPES-IGT	239	LANLKILDLSNNGLT-GRIPASIGR
8	259 LKTVEQIELFNNSFSGE-LPES-MGN	267	LESVY <mark>QIELYDN</mark> RLSGKLPES-IGN	263	LRSVEKMELWRNQLS-GELPQSLGN
9	283 MTTLKRFDASMNKLTGK-IPDN-LN-	2 <mark>91</mark>	LTELRNFDVSQNNLTGELPEK-IA-	287	LTSLFAFDASENKLT-GKLPE-GLA
10	306 LLNLESLNLFENMLEGP-LPES-ITR	314	ALQLI <mark>SFNLN</mark> DNFFTGGLPDV-VAL	310	GLNLTSLALNDNRMD-GEISTVLAQ
11	330 SKTLSELKLFNNRLTGV-LPSQ-LGA	338	NPNLVEFKIFNNSFTGTLPRN-LGK	334	NPHLVELKLENNNFS-GELPSGLGR
12	354 NSPLQYVDLSYNRFSGE-IPAN-ICG	362	FSEISEF DVST NRFSGELPPY-L	358	YSYLTNV <mark>DVS</mark> GNQFA-GRLPPDL <mark>C</mark> S
13	378 EGKLEYLILIDNSFSGE-ISNN-LGK	386	RRKLQ KIITF SNQLSGEIPES-YGD	382	RGMLESLVAFGNRFS-GELPQSYAD
14	402 CKSLTRVRLSNNKLSGQ-IPHGFWG-	410	CHSLNYIRMADNKLSGEVPAR-FWE	409	CRTLDYVRIQNNELS-GKVPDLFWS
15	426 LPRLSLAELSDNSFTGS-IPKTIIG-	434	LPLTRLELANNNQLQGSIPPS-ISK	430	LPKLYHLELRGNKLE-GSLPPNISR
16	450 AKNL <mark>SNLR</mark> ISKNRFSGS-IPNE-IGS	458	arhl <mark>so</mark> leisannfsgvipvk-lcd	454	AKNL <mark>TQ</mark> IIISDNKFS-GQIPPEICD
17	474 LNGIIEISGAENDFSGE-IPES-LVK	482	lrdl <mark>rv</mark> idlsrnsflgsipsc-ink	478	LMELRTFDAGNNQFS-SGLPLCIAD
18	498 LKQL <mark>SR</mark> LDLSKNQLSGE-IPRE-LRG	506	lkni <mark>er</mark> vemqenmidgeipss-vss	502	LTKLQVLDLQQNNFS-GEIPA-GG-
19	522 WKNLNELNLANNHLSGE-IPKE-VGI	530	CTELTELNLSNNRLRGGIPPE-LGD	524	WTEL <mark>AB</mark> INLSMNRFS-GEIPRSLGD
20	546 LPVLNYLDLSSNQFSGE-IPLE-LQN	554	LPVLNYLDLSNNQLTGEIPAE-LLR	548	LPVLTYLDLSGNQLS-GEIPPELTN-
21	570 L-KLNVLNLSYNHLSGK-IPPL-YA-	578	L- <mark>KLN</mark> QFNVSDNKLYGKIPSFQ-	572	L- <mark>KLNHLNL</mark> SGNDLS-GKIPDGF-
22	592 NKIYAHDFIGNPGLCVD-LDGLCR	600	QD <mark>IF</mark> RPSFLGNPNLCAPNLDPIRPC	594	DTSFFLPSLLGNPDLCSSSGLKTFRRC

Figure S3. Structure-based sequence alignment of the 21 leucine-rich repeats (LRRs) comparing Arabidopsis AtHAE and AtHSL2 with EgHSL2 from oil palm. Residue numbers are found in the columns to the left of the respective alignments. The plant LRR consensus sequence making up the scaffold of the ectodomain is shown on top of the alignments and is shaded in grey throughout the LRRs. Residues shown from the AtHAE-AtIDA crystal structure to mediate hydrophobic interactions with AtIDA, and corresponding conserved residues in AtHSL2 and EgHSL2, are highlighted in blue, residues contributing to hydrogen bond interactions and/or salt bridges are shown in red. N-glycosylation sites are indicated in green, cysteine residues involved in disulphide bridge formation in orange, and residues involved in binding of AtSERK1 in yellow.