

A Non-Canonical Function of Arabidopsis ERECTA Proteins in Gibberellin Signaling

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46 **Short title: The Role of ERECTA and SWI3B in GA Signaling**

47 **ONE SENTENCE SUMMARY:** ERECTA leucine-rich receptor-like kinase and SWI3B subunit
48 of SWI/SNF chromatin remodeling complex cooperate in direct transcriptional control of *GID1*
49 genes in Arabidopsis.

50

51 **Authors Contributions**

52 TJS, ES, and SJD planned experiments and wrote the manuscript

53 SK and PC participated in the planning of some experiments

54 TJS, ES, SK, PC, SS, SA, BH, JAS, and ARF analyzed the data

55 ES, PC, SS, SK, PO, JS, MZ, JMS, RD, AM, MS, BH, MC, KN, ATR, EB, RF, AK, MAD, SA,
56 and TJS performed experiments

57 All authors read, edited, and approved the final manuscript

58 **Key Words:** Arabidopsis, ERECTA, ERECTA-LIKE1, ERECTA-LIKE2, LRR-RLK, SWI/SNF,
59 SWI3B, HER2, Chromatin

60

61 **Abbreviations and Acronyms:**

62 ERf, ERECTA family; ER, ERECTA; ERL1, ERECTA-LIKE 1; ERL2, ERECT-LIKE 2; LRR-
63 RLKs, leucine-rich repeat receptor-like kinases; CRC, chromatin remodeling complex;
64 SWI/SNF, Switch/Sucrose Nonfermenting; *GID1*, *GIBBERELLIN INSENSITIVE DWARF 1*; GA,
65 gibberellin; PAC, Paclobutrazol; qRT-PCR, quantitative real-time PCR; BFA, Brefeldin A; NLS,
66 nuclear localization signal; KDER, the kinase domain of ERECTA; TSS, transcription start site;
67 EGFR, epidermal growth factor receptor.

68

69 **Abstract**

70 The Arabidopsis ERECTA family (ERf) of leucine-rich repeat receptor-like kinases
71 (LRR-RLKs), comprising ERECTA (ER), ERECTA-LIKE 1 (ERL1) and ERECTA-LIKE 2
72 (ERL2), control epidermal patterning, inflorescence architecture, stomata development, and
73 hormonal signaling. Here we show that the *er/erl1/erl2* triple mutant exhibits impaired
74 gibberellin (GA) biosynthesis and perception alongside broad transcriptional changes. ERf
75 proteins interact in the nucleus, *via* kinase domains, with the SWI3B subunit of the SWI/SNF
76 chromatin remodeling complex (CRCs). The *er/erl1/erl2* triple mutant exhibits reduced SWI3B
77 protein level and affected nucleosomal chromatin structure. The ER kinase phosphorylates
78 SWI3B *in vitro*, and the inactivation of all ERf proteins leads to the decreased phosphorylation of
79 SWI3B protein *in vivo*. Correlation between DELLA overaccumulation and SWI3B proteasomal
80 degradation together with the physical interaction of SWI3B with DELLA proteins explain the
81 lack of RGA accumulation in the GA- and SWI3B-deficient *erf* mutant plants. Co-localization of
82 ER and SWI3B on *GIDI* (*GIBBERELLIN INSENSITIVE DWARF 1*) DELLA target gene
83 promoter regions and abolished SWI3B binding to *GIDI* promoters in *er/erl1/erl2* plants
84 supports the conclusion that ERf-SWI/SNF CRC interaction is important for transcriptional
85 control of GA receptors. Thus, the involvement of ERf proteins in transcriptional control of gene
86 expression, and observed similar features for human HER2 (Epidermal Growth Family Receptor-
87 member), indicate an exciting target for further studies of evolutionarily conserved non-canonical
88 functions of eukaryotic membrane receptors.

89

90 **Introduction**

91 The ERECTA family (ERf) of leucine-rich-repeat receptor-like kinases (LRR-RLKs)
92 consists of three members: ERECTA (ER), ERECTA-LIKE 1 (ERL1), and ERECTA-LIKE 2
93 (ERL2). ERf proteins carry extra-cellular leucine-rich repeats (LRRs), as well as transmembrane
94 and cytosolic kinase domains (Shpak et al., 2004; Torii et al., 1996, Kosentka et al., 2017).
95 Inactivation of *ERECTA* leads to inflorescence, pedicels, and siliques compaction, while the
96 individual loss of either *ERL1* or *ERL2* function has a limited effect on Arabidopsis development
97 (Shpak et al., 2004). ERf proteins are functionally redundant-their simultaneous inactivation
98 results in dramatic growth retardation, severe dwarfism, enlargement of the shoot apical meristem
99 (SAM), clustered stomata, and sterility. ERf regulates stem cell homeostasis *via* buffering
100 cytokinin responsiveness and auxin perception in SAM and modulating the balance between stem
101 cell proliferation and consumption (Shpak et al., 2004; Griffiths et al., 2006; Torii et al., 2007;
102 Chen et al., 2013; Shpak, 2013; Uchida et al., 2013; Zhang et al., 2021). ERECTA controls the
103 expression of genes associated with gibberellin (GA) metabolism (Uchida et al., 2012a)
104 restricting xylem expansion downstream of the GA pathway (Ragni et al., 2011). It additionally
105 regulates shade avoidance in a GA and auxin-dependent manner (Du et al., 2018) and
106 ethylene-induced hyponastic growth (Van Zanten et al., 2010).

107 Overexpression of ER variant lacking the C-terminal kinase domain (ER Δ K) caused more
108 severe developmental defects than complete inactivation of *ERECTA*, suggesting an interaction
109 of the kinase domain with important regulatory partners (Shpak, 2003). ERECTA interacts with
110 ERL1 and ERL2 to form receptor complexes recognizing two endodermis-derived peptide
111 hormones (EPFL4 and EPFL6), regulating vascular differentiation and stem elongation. ERf
112 proteins additionally form complexes with the receptor-like protein TOO MANY MOUTHS
113 (TMM), which controls stomatal differentiation by recognition of the secretory peptides
114 EPIDERMAL PATTERNING FACTOR 1 (EPF1), EPF2, and stomagen (Lee et al., 2012;
115 Uchida et al., 2012a; Lee et al., 2015b).

116 ERL2 has been found to undergo endocytosis (Ho et al., 2016), suggesting that ERf
117 proteins may play, as yet uncharacterized, regulatory roles upon internalization, in addition to
118 their functions as ligand-binding membrane receptors. ERECTA signaling, in tandem with the
119 SWR1 chromatin remodeling complex (CRC), controls the expression of the *PACLOBUTRAZOL*
120 *RESISTANCE 1 (PRE1)* family genes. This observation supports their role in the GA signaling

121 pathway, however, neither direct interaction between ERECTA and SWR1 nor the direct
122 influence of ERECTA signaling on chromatin structure or SWR1 activity has, as yet, been
123 demonstrated (Cai et al., 2017; Cai et al., 2021).

124 Here we show that the loss of all ERF proteins in the *er/erl1/erl2* triple mutant (*erf*) results
125 in broad transcriptomic changes affecting hormonal, developmental, and metabolic processes.
126 Inactivation of ERF proteins caused down-regulation of the GA receptor *GIDI* (*GIBBERELLIN*
127 *INSENSITIVE DWARF 1*) genes expression and decreased bioactive GA levels. The ER protein
128 undergoes endocytosis and enters the nucleus. All three ERF proteins interact in the nucleus with
129 the SWI3B core subunit of the SWI/SNF CRCs. The kinase domain of the ER protein exhibits the
130 ability to phosphorylate SWI3B protein. The physical interaction of SWI3B with RGA and
131 RGL1, together with identified correlation between DELLA accumulation and SWI3B
132 proteasomal degradation, provide an explanation as to why GA-deficient *erf* mutant plants did
133 not overaccumulate RGA. These data collectively suggest cooperation of ERF-signaling with
134 SWI/SNF in the modulation of gene transcription. The ER and SWI3B also co-localized in the
135 promoter regions of *GIDI* DELLA target genes. In the *erf* mutant, the binding of SWI3B to *GIDI*
136 promoters was abolished. These results collectively suggest that ERF proteins directly control GA
137 receptor expression by restricting recruitment of the SWI/SNF CRCs to its target *loci*.

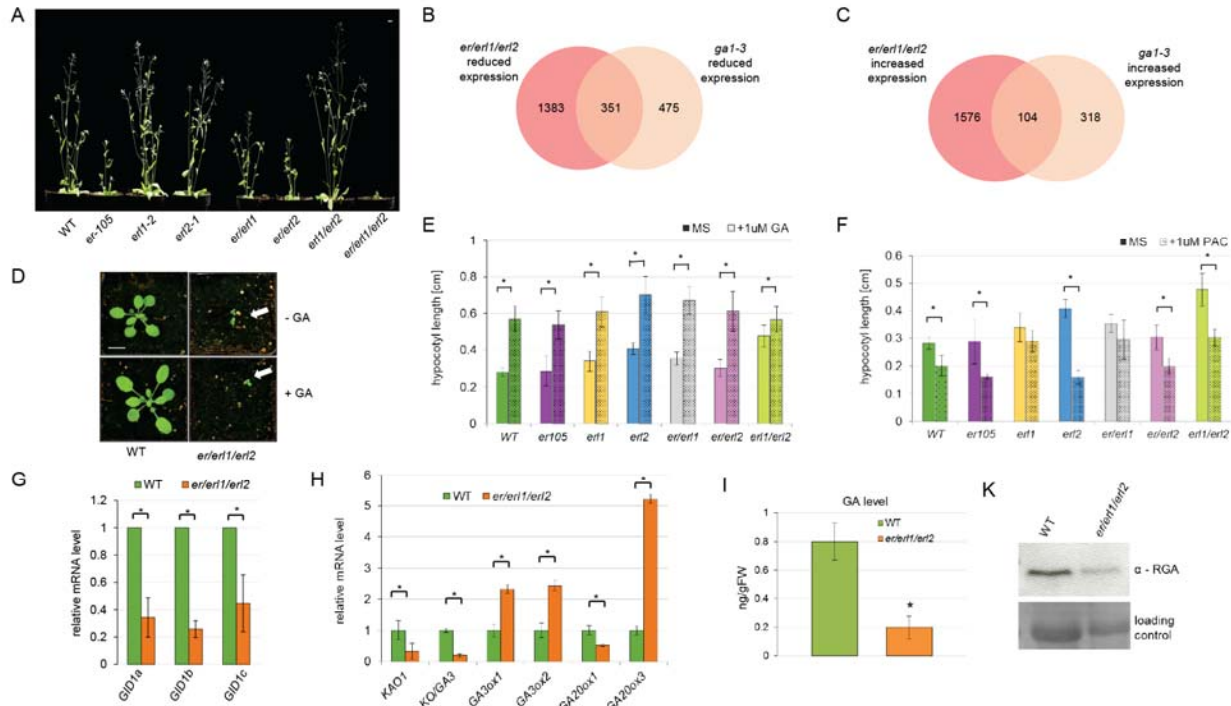
138

139 **Results**

140 **Inactivation of *Erf* Proteins Has a Broad Effect on the Arabidopsis Transcriptome**
141 **including GA Signaling.**

142 The Arabidopsis *er/erl1/erl2* plants exhibit severe dwarfism, dark green color, defects in
143 vascular development, stem elongation, and stomatal differentiation, as well as complete sterility
144 (Figure 1 A, Supplemental Figure 1A, (Shpak et al., 2004)).

145



146

147 **Figure 1.** *ERf* inactivation affects Arabidopsis development, causes transcriptomic changes

148 overlapping with the effect of *gal-3* mutation and impairs GA biosynthesis and signaling (See

149 also Figures S1, S2 and S3). A, Phenotypic changes conferred by combinations of *erf* mutations.

150 Scale bar= 1 cm. B, Overlapping down-regulated genes in *er/er1/er2* and *gal-3* plants. C,

151 Overlapping up-regulated genes in *er/er1/er2* and *gal-3* plants. D, The *er/er1/er2* plants

152 exhibit impaired GA response. 14-days old LD (12h day/12 night) grown WT and *er/er1/er2*,

153 sprayed twice a week with water (upper row) or 100μM GA₄₊₇ (lower row). Arrows-*er/er1/er2*

154 plants. Scale bar= 1cm. E, The GA response is retained to various levels in combinations of *erf*

155 mutants. Error bars-SD,* = P < 0.05, Student's *t*-test, n= 30 plants. F, The response of various *erf*

156 mutants to 1μM Paclobutrazol treatment. Error bars-SD,* = P < 0.05, Student's *t* test, n= 30

157 plants. G, The *er/er1/er2* mutant exhibits altered transcription of *GID1* GA receptor genes (error

158 bars-SD, P < 0.05, Student's *t*-test, three biological and technical replicates were assayed). H,

159 The *er/er1/er2* mutant displays altered GA biosynthesis and metabolism-related genes

160 expression (error bars-SD, P < 0.05, Student's *t*-test, three biological and technical replicates

161 were assayed). I, The *er/er1/er2* mutant exhibits dramatically reduced level of bioactive GA₄₊₇

162 gibberellin (error bars-SD, P < 0.05, Student's *t*-test, three biological and technical replicates

163 were assayed). J, The *er/er1/er2* mutant shows decreased level of the DELLA protein RGA.

164

165 Given the severe phenotypic alterations of the *er/erl1/erl2* plants, we performed transcript
166 profiling with Affymetrix ATH1 microarrays on RNA samples from aerial parts of the
167 *er/erl1/erl2* mutant and WT (wild type) adult plants (representing the most comparable stage of
168 the development) grown for 5 weeks under long-day conditions (16h day/8h night). Data analysis
169 identified 1734 versus 1680 genes showing >1.50-fold decrease and increase, respectively, of
170 transcript levels in *er/erl1/erl2* comparing to WT (Supplemental Figure 1B, Supplemental
171 Dataset 1 Sub-tables 1, 2). Gene Ontology (GO) terms of primary metabolism, developmental
172 processes, and response to hormones were enriched among the *er/erl1/erl2* down-regulated genes
173 (Supplemental Dataset 1 Sub-table 3). Among these, 27 genes were classified to GA-response
174 (Supplemental Table 1, Supplemental Dataset 1 Sub-tables 1, 3). The up-regulated genes were
175 classified into GO-terms of chloroplast-related metabolic and light-regulated transcription
176 processes, responses to cytokinin, and auxin degradation (Supplemental Dataset 1 Sub-Tables
177 2,4). Several genes acting in leaf epidermal and stomatal cell differentiation showed enhanced
178 transcription in the *er/erl1/erl2* mutant (Supplemental Table 2). In conclusion, the inactivation of
179 ERf altered transcriptional regulation of hundreds of targets, including a set of GA-regulated
180 genes.

181 Phenotypic traits exhibited by double and triple *erf* mutants resemble those of double and
182 triple *gid1abc* (*gibberellin insensitive dwarf 1a, b* and *c*) plants (Figure 1A; (Griffiths et al.,
183 2006)). Inactivation of *GID1abc* genes has a nearly identical effect on the Arabidopsis
184 transcriptome as the severe GA-deficient mutant *gal-3* (Willige et al., 2007), thus we compared
185 the transcriptomic data available for the *gal-3* mutant with those caused by inactivation of all *ERf*
186 genes.

187 We identified a large overlap of differentially expressed genes (DEG) in the *er/erl1/erl2*
188 and *gal-3* lines. Among 826 genes down-regulated in the *gal-3* line, 351 (about 42.5%) also
189 exhibited decreased expression in the *er/erl1/erl2* plants (Figure 1B), while 104 genes (about
190 24.6% of *gal-3* up-regulated genes) were up-regulated in both lines (Figure 1C). Only 33 genes
191 were up-regulated in *gal-3* but down-regulated in *er/erl1/erl2* (Supplemental Figure 1C), and
192 only 64 genes down-regulated in *gal-3* but up-regulated in *er/erl1/erl2* (Supplemental Figure
193 1D). DEG common to *gal-3* and *er/erl1/erl2* lines belonged to both DELLA (repressors of GA
194 pathway) -dependent and DELLA-independent classes (Cao et al., 2006), regardless of whether
195 they display co-regulation or contrasting regulation in these lines (Supplemental Figure 1E and

196 F). This suggests the involvement of Arabidopsis ERF proteins in the control of GA-related
197 processes. Therefore, we next tested the response of *er/erl1/erl2* plants to exogenously supplied
198 bioactive 100 μ M GA₄₊₇ and found that spraying of the *er/erl1/erl2* mutant grown under LD
199 condition (12h day/12h night) did not lead to increased leaf size by day 14 compared to the
200 remarkable expansion of control WT rosette leaves (Figure 1D, Supplemental Figure 2A). Thus
201 *er/erl1/erl2* displayed GA insensitivity. Nonetheless, the GA-treatment resulted in bolting of
202 *er/erl1/erl2* plants, only after over two months (Supplemental Figure 2B and C), indicating their
203 residual response to GA.

204 Although we showed that ERF proteins are involved in the GA response, it remained
205 unclear whether proper GA perception requires all ERF proteins. Thus, we tested the hypocotyl
206 response of single and double *erf* mutants in various combinations to the treatment with 1 μ M
207 GA₄₊₇ or 1 μ M Paclobutrazol (PAC), an inhibitor of GA biosynthesis. The GA response was
208 retained to various levels in all tested mutants (Figure 1E) while the *erl1* and *er/erl1* plants had an
209 impaired response to PAC and *erl1/erl2* displayed a significant reduction of hypocotyl length
210 (Figure 1F).

211 Upon crossing *er*, *er/erl1*, and *er/erl2* lines with the *gal-3* mutant, we observed only a
212 discrete enhancement of the *gal-3* phenotype. However most of the phenotypic changes
213 characteristic for *gal-3* mutation were retained, indicating that many of the *er*, *erl1*, or *erl2* single
214 or double mutant phenotypes are likely not exclusively a result of GA deficiency (Supplemental
215 Figure 2D and E).

216 We have proven that only parallel inactivation of all ERF proteins causes severe
217 impairment of the GA response. Quantitative real-time PCR (qRT-PCR) measurements of GA
218 response and biosynthesis genes expression revealed a parallel 2.5 to 3-fold reduction in the
219 transcript levels of all three *GIDI* GA-receptors in the *er/erl1/erl2* mutant compared to WT
220 (Figure 1G). The GA-receptor genes *GID1A/B* have been reported to be direct CHIP targets of
221 RGA, a major DELLA repressor of GA-signaling, which stimulates *GIDI* transcription (Zentella
222 et al., 2007). The *er/erl1/erl2* triple mutant also displayed altered expression of GA biosynthesis
223 genes compared to the WT: a 4-fold reduction of mRNA levels of *KAOI* (*ent*-kaurenoic acid
224 oxidase) and *KO* (*ent*-kaurene oxidase), a 2.5-fold increase of mRNA levels of GA-repressed
225 *GA3ox1* and *GA3ox2* (GIBBERELLIN 3 BETA-HYDROXYLASE 1 and 2), a 2-fold inhibition
226 and 5-fold up-regulation, respectively, of mRNA levels corresponding to the *GA20ox1* and

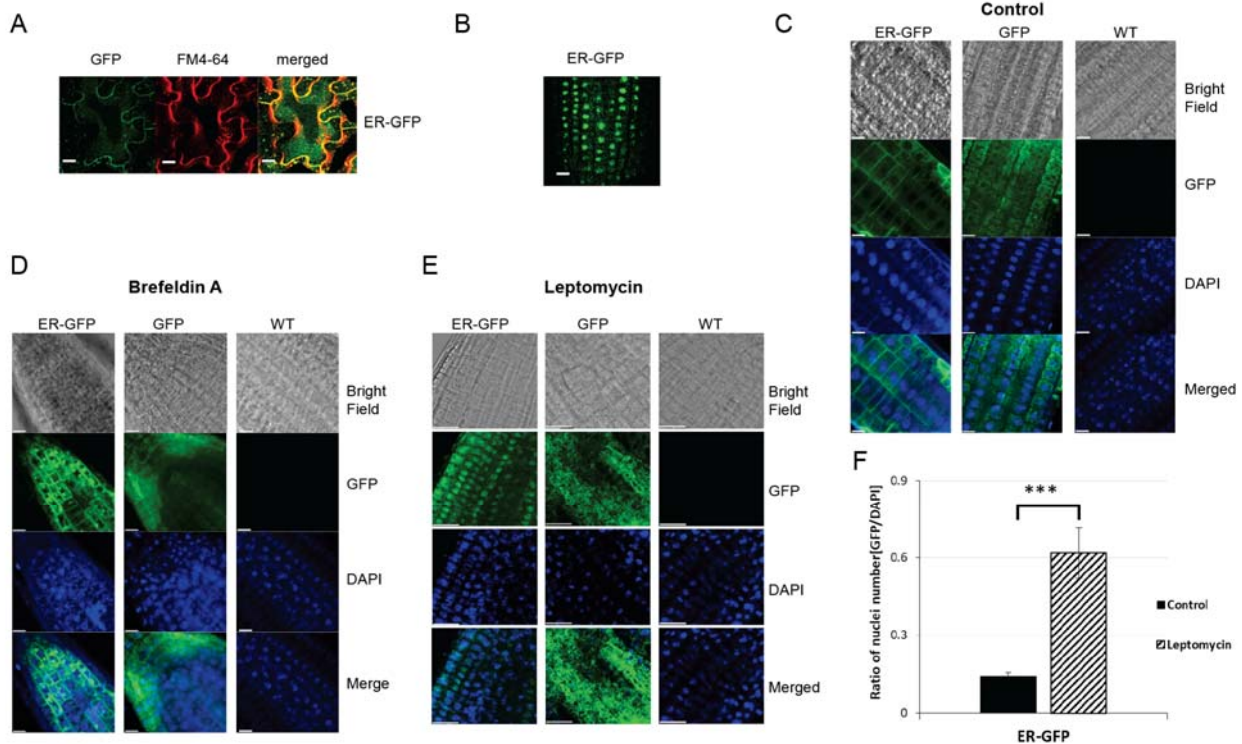
227 *GA20ox3* genes (Figure 1H). This indicated that the ERF proteins not only influence the
228 expression of GA receptors, but also genes associated with GA biosynthesis. We subsequently
229 found a substantial decrease of bioactive GA₄ as well as GA₁₂, and GA₂₄ intermediates in
230 *er/er11/er12* mutant (Figure 1I, Supplementary Figure 3). Counterintuitively, the Western blotting
231 using a specific antibody (Willige et al., 2007) detected reduced levels of RGA in the *er/er11/er12*
232 mutant plants (Figure 1J). Our results indicate that the parallel inactivation of all ERF proteins
233 results in co-ordinate deregulation of GA biosynthesis and response pathways in Arabidopsis.

234

235 **ERECTA (ER) Protein Undergoes Endocytosis and Migrates to the Nucleus.**

236 In analogy to some human membrane receptors internalizing to endosomes and migrating
237 to the nucleus (*i.e.*, Giri et al., 2005), the ERL2 member of the ERF undergoes endocytosis (Ho et
238 al., 2016). We next examined, in detail, the cellular localization of ER by creating C-terminal
239 GFP fusions with ER (Figure 2A) after verifying genetic complementation of the *er-105* mutation
240 by a 35S::ER-GFP construct (Supplemental Figure 4).

241



242
243 **Figure 2.** Subcellular localization of ERECTA protein (See also Figures S4 and S5). A,
244 ERECTA is localized in plasma-membrane and endosomes in epidermal cells of 7-days old
245 seedlings. ER-GFP, or free GFP visualized using GFP channel. FM4-64 specifically stains
246 plasma-membranes. Scale bar=10 μ m. B, Root-tip images of approximately two-week-old (14-17
247 days) ER-GFP seedlings showing nuclear localization of ERECTA protein at considerable
248 frequency. C, Root-tip images of 12-day-old ER-GFP seedlings serving as the control for D and
249 E. D, Brefeldin A treatment enhanced the localization of ERECTA protein in Brefeldin A (BFA)
250 bodies. Roots of 12-day-old Arabidopsis seedlings. E, Leptomycin B treatment enhanced the
251 nuclear localization of ERECTA Free GFP was used as a control in C, D, and E, cell nuclei were
252 stained with DAPI, scale bar= 50 μ m. F, Letomycin B enhances nuclear presence of ER protein.
253 The GFP/DAPI ratio calculated per area for roots of plants expressing ER-GFP protein.
254

255 As observed earlier (Shpak et al., 2005; Uchida et al., 2012a), a pool of the ER-GFP
256 protein was detected in association with plasma-membranes of the leaf epidermis. In addition, a
257 weak localization signal was detected in internal structures, which could represent endosomes
258 (Figure 2A). In guard cell pairs, ER-GFP protein was also detected in circles around the positions
259 of nuclei, which were visualized by propidium iodide staining (Supplemental Figure 5A and
260 Supplemental Movie 1). We also observed with considerable frequency ER protein in the nuclei
261 of roots of 14- to 17-day-old Arabidopsis plants (Figure 2B), however ER was mainly located in
262 the plasma membrane and endosome-like structures (Figure 2C).

263 To verify that the ER-GFP protein indeed undergoes endocytosis, we examined its
264 localization in Arabidopsis seedlings treated with 25 μ M Brefeldin A (BFA), a compound
265 preventing Golgi-mediated vesicular transport of membrane proteins to the plasma membrane
266 (Miller et al., 1992). We observed accumulation of the ER-GFP protein in BFA bodies within 30-
267 40 min after BFA treatment leading to its accumulation at the nuclei periphery 120 min after
268 BFA application (Figure 2D, Supplemental Figure 5B). The 4h long 200 nM leptomycin B
269 treatment (a compound blocking nuclear export by EXPORTINS (Haasen et al., 2002)) resulted
270 in the ER accumulation in the cell nuclei (Figure 2E, F). ER thus appeared to behave similarly to
271 certain human plasma-membrane receptors in migrating into the nucleus (Hung et al., 2008; Chen
272 and Hung, 2015).

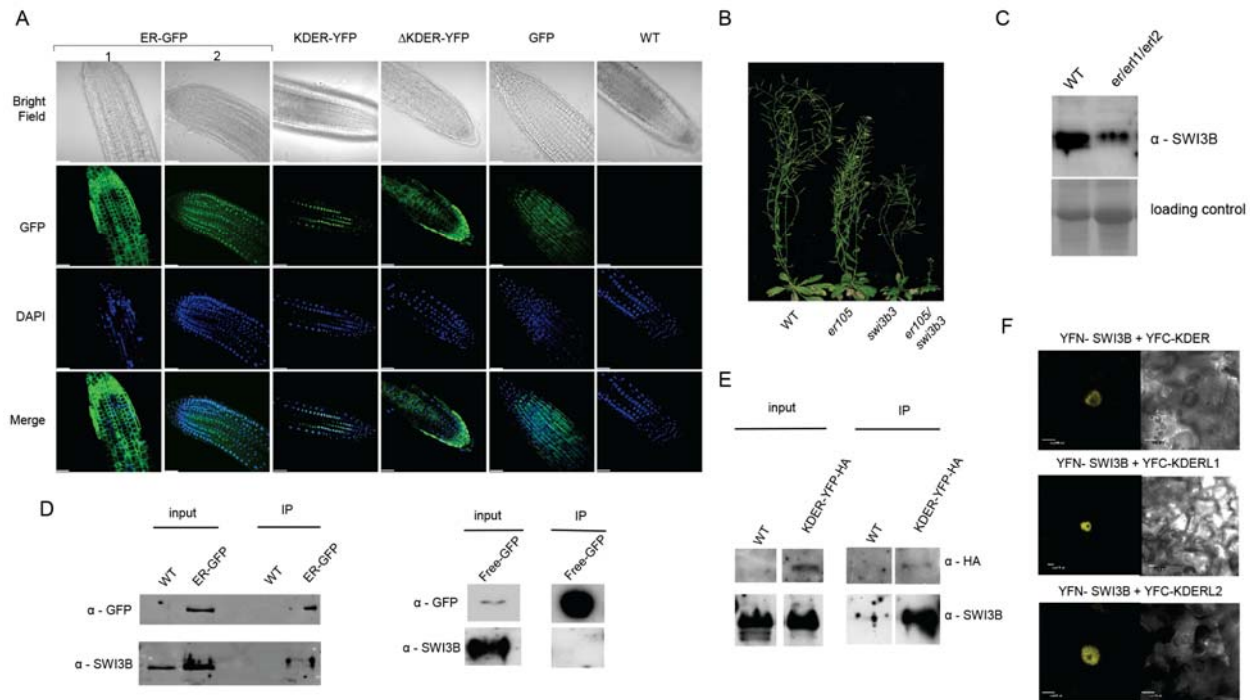
273 We noted that ERL1 and ERL2 proteins carry a monopartite nuclear localization signal
274 (NLS) sequence in their kinase domains, while the ERL1 kinase domain carries an additional
275 bipartite NLS identified using cNLS Mapper (Kosugi et al., 2009b). The NLS signal in the ER
276 protein was not recognized, however, all ERf proteins show evolutionally conserved amino acid
277 sequences in this region (Supplemental Figure 6A). Using the NetNES1.1 (la Cour et al., 2004)
278 server, we predicted the existence of specific for AtXPO1/AtCRM1 exportin (Haasen et al.,
279 2002) leucine-rich nuclear export signals (NES) in all ERf proteins (Supplemental Figure 6A).
280 The subsequent Western-blotting analysis of nuclear extracts (Supplemental Figure 6B)
281 confirmed the nuclear presence of ER. In addition to the expected full-length forms (140 kDa),
282 we also detected shorter (~75 kDa) versions of the ERECTA protein with the C-terminal GFP tag
283 and smaller products of degradation, including free GFP, suggesting an analogy to the human
284 Epidermal Growth Factor Receptor (EGFR), (Chen and Hung, 2015). The detection of N-
285 terminally truncated forms of the ERECTA protein carrying a kinase domain resembled the

286 recently reported fate of the XA21 LRR-RLK immune receptor in rice (Park and Ronald, 2012),
287 where its C-terminal kinase domain enters the nucleus to interact with the OsWRKY62
288 transcriptional regulator.

289 To assess whether the ERECTA kinase domain (KDER) is imported into the nucleus, we
290 fused the C-terminal part of ERECTA, harboring the KDER, to a YFP-HA tag (Supplemental
291 Figure 6C) and expressed this construct in the *er-105* mutant. The presence of KDER-YFPHA
292 was detected exclusively in cell nuclei (Figure 3A). Furthermore, KDER-YFPHA expression
293 partially restored the *er-105* rosette and cauline leaf phenotype to WT values (Supplemental
294 Figure 7A, B). Still, it failed to genetically complement the defect of stem elongation
295 (Supplemental Figure 7C). Partial genetic complementation of the *er-105* mutation and nuclear
296 localization of KDER prove that the KDER has a receptor-domain independent signaling
297 function. Interestingly, by contrast to the full length and kinase domain of ERECTA protein
298 (Figure 3A), truncated ER Δ kinase form of ERECTA protein fused to YFP-HA (Δ KDER-YFP-
299 HA) did not enter into the nucleus proving the presence of functional NES and NLS sequences in
300 the C-terminal part of ERECTA protein.

301

302



303

304 **Figure 3.** Nuclear function of ERf proteins (See also Figures S6, S7, S8, S9, and S10). A, Root-
 305 tip images of approximately two-week-old (14-17 days) plants expressing ER-GFP, KDER-YFP-
 306 HA (the kinase domain of the ER protein), ER Δ K-YFPHA (truncated ER protein lacking the
 307 kinase domain) proteins indicating that the kinase domain is necessary for the nuclear localization
 308 of ER protein. WT and GFP expressing plants-negative controls. Panel 2 in the ER-GFP indicates
 309 nuclear localization of ER protein appearing at considerable frequency. Cell nuclei were stained
 310 with DAPI. Scale bar=25 μ m. B, The *er-105/swi3b3* double mutant shows more retarded growth
 311 than either *er-105* or *swi3b3* plants. Scale bar= 1cm. C, The *er/er1/erl2* triple mutant exhibits
 312 reduced SWI3B protein level. D, ER-GFP or free GFP (negative control) pull-down from the
 313 nucleus and anti-SWI3B western blotting indicate a specific ER-SWI3B interaction. E,
 314 Immunoprecipitation of KDER-YFP-HA from the nucleus indicated that the kinase domain of
 315 ER interacts with SWI3B. F, ER, ERL1, and ERL2 kinase domains interact with SWI3B in the
 316 nucleus. Bimolecular Fluorescence Complementation assay (BiFC) in epidermis of tobacco
 317 leaves. Scale bar = 10 μ m.

318

319 Upon nuclei fractionation (Sarnowski et al., 2002), the ERECTA protein was detected in
320 the nuclear membrane, soluble nuclear-protein fraction, and chromatin, with its major presence
321 within the nuclear matrix. The nuclear fractions contained both full-length and N-terminally
322 truncated ER forms (Supplemental Figure 8) suggest that ER could be involved in either
323 transcriptional regulation or other nuclear functions.

324

325 **ERf Proteins Physically Interact with the SWI3B Core Subunit of SWI/SNF CRC**

326 The weak *swi3b-3* allele (Sáez et al., 2008) carrying, in the *er-105* background, a point
327 mutation in the *SWI3B* gene encoding a core subunit of the SWI/SNF chromatin remodeling
328 complex (CRC) exhibit severe dwarfism, altered leaf shape, delayed flowering and reduced
329 fertility (Figure 3B). We, therefore, introgressed the *swi3b-3* mutation into WT and found that the
330 phenotypic alterations related to *swi3b-3* were much weaker (slight reduction of growth rate,
331 leading to decreased plant height, Figure 3B) than the phenotypic traits exhibited by the *er-*
332 *105/swi3b-3* as well as single *er-105* mutation. The severe phenotypic alterations exhibited by the
333 *er-105/swi3b-3* plants indicated the likely existence of a strong genetic interaction between the
334 ERECTA signaling pathway and SWI3B-containing SWI/SNF CRCs. This observation is in line
335 with *i*) the direct binding of 15 out of 27 potential ERf target genes related to the GA signaling
336 pathway (Supplemental Table 1) by SWI/SNF CRCs (Sacharowski et al., 2015; Archacki et al.,
337 2016; Li et al., 2016); *ii*) the unexpected broad transcriptional changes and severe effects on
338 Arabidopsis development and hormonal signaling pathways observed in the *er/erl1/erl2* mutant,
339 and *iii*) the well-recognized function of SWI/SNF CRC in hormonal crosstalk including GA
340 signaling (Sarnowska et al., 2013; Sarnowska et al., 2016).

341 We next assessed the level of the SWI3B protein in *er/erl1/erl2* plants. We found a
342 significant decrease in the SWI3B protein abundance (Figure 3C), further suggesting that the ERf
343 signaling pathway may influence the proper function of SWI3B-containing SWI/SNF CRCs.
344 Additionally, the SWI3B was found to bind ER-GFP but not free GFP (Figure 3D). Similarly, co-
345 immunoprecipitation indicated that SWI3B interacts with the kinase domain of ER (Figure 3E).

346 Next, we performed BiFC assays (Hu et al., 2002) in epidermal cells of *Nicotiana*
347 *benthamiana* and confirmed the SWI3B and ER kinase domain interaction. The YFC-RFP served
348 as a control unrelated protein with broad intracellular localization (Figure 3F, Supplemental
349 Figure 9). We also detected the interaction of SWI3B with the kinase domain of the ERL1 or

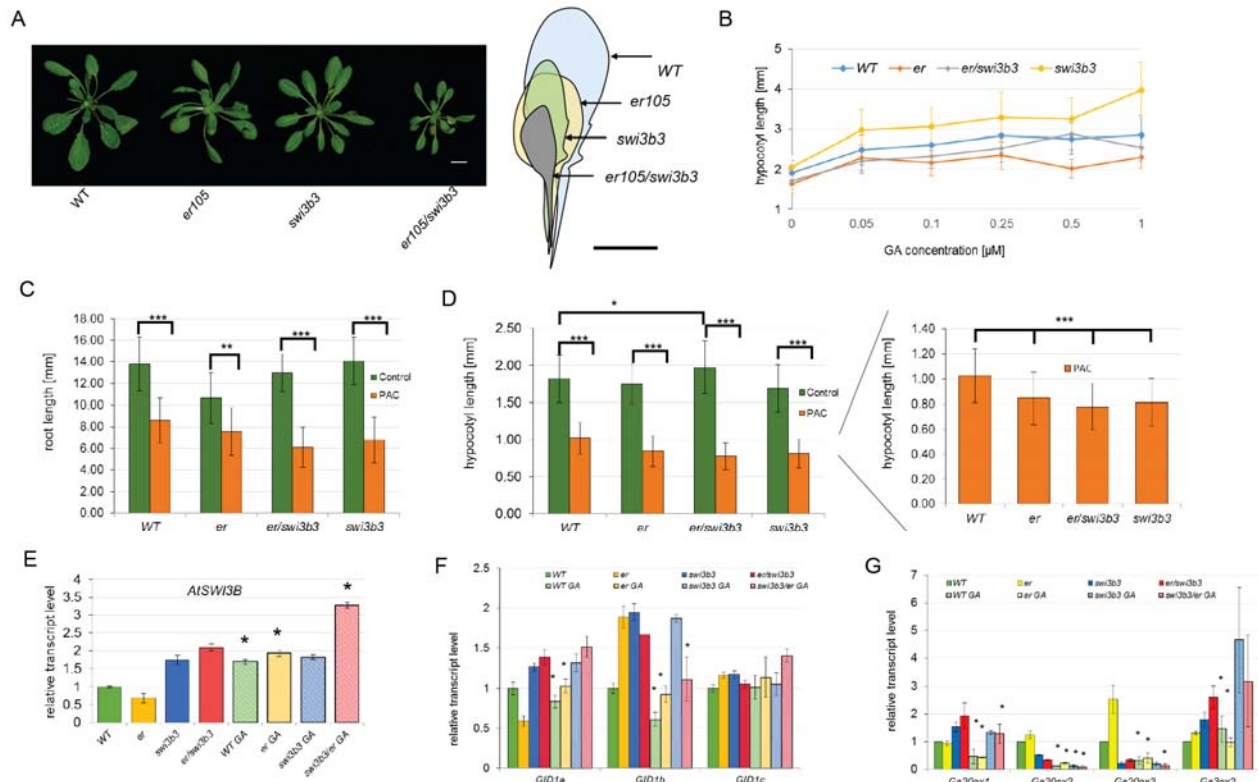
350 ERL2 (Figure 3F, Supplemental Figure 9), indicating the existence of direct interdependences
351 between the ERf signaling pathway and SWI/SNF–dependent chromatin remodeling.

352 Moreover, we found similar interactions in the nuclei of human cells for HER2
353 (Epidermal Growth Factor Receptor- family member), a membrane receptor acting in a non-
354 canonical signaling mode including translocation to the nucleus (Lee et al., 2015a), and BAF155
355 a SWI3-type subunit of human SWI/SNF CRCs (Supplemental Figure 10). Thus, our data
356 indicate that the phenomenon observed for ERf and SWI3B is not limited to Arabidopsis but
357 rather may be a general feature of SWI/SNF CRCs and membrane receptors.

358
359 ***ERECTA* and *SWI3B* Interact Genetically and *er/elr1/erl2* Plants Exhibit Alteration in**
360 **Chromatin Status.**

361 The *er-105/swi3b-3* double mutant exhibited more severe phenotypic traits than both single *er-*
362 *105* and *swi3b-3* mutant lines (Figure 4A), supporting the observed physical interdependences
363 between ER and SWI3B.

364



365
 366 **Figure 4.** ER and *SWI3B* interact genetically and affect both GA biosynthesis and response
 367 pathways (See also Figures S11 and S12). A, The *er-105/swi3b-3* double mutant exhibits more
 368 retarded growth than the *er-105* and *swi3b-3* (three-weeks old plants). Graphical alignment of
 369 corresponding leaves. Scale bar= 1 cm. B, The hypersensitivity of 1-week-old *swi3b-3* hypocotyl
 370 to GA treatment is abolished by introducing *er-105*. C, Roots of all tested 1-week-old genotypes
 371 similarly respond to PAC treatment (error bars-SD, * $P < 0.01$, ** $P < 0.001$, *** $P < 0.0001$,
 372 Student's *t*-test). D, Hypocotyls of all tested 1-week-old genotypes similarly respond to PAC
 373 treatment, right panel- hypocotyl length comparison for PAC treated plants only (error bars-SD,
 374 * $P < 0.01$, ** $P < 0.001$, *** $P < 0.0001$ Student's *t*-test). E, *swi3b-3* weak, point mutant line and
 375 *er-105/swi3b-3* exhibit elevated *SWI3B* transcript level, the *SWI3B* expression is elevated after
 376 supplementation with bioactive GA_{4+7} in all genotypes except *swi3b-3* (error bars-SD, $P < 0.05$,
 377 Student's *t*-test). F, The examination of *GID1* genes indicated that almost all examined lines
 378 responded to GA treatment, but the *swi3b-3* line was insensitive for GA-induced transcriptional
 379 changes (error bars-SD, $P < 0.05$, Student's *t*-test). G, The examination of GA biosynthesis genes
 380 indicated that almost all examined lines responded to GA treatment, but the *swi3b-3* line was
 381 insensitive for GA-induced transcriptional changes except *GA20ox2* expression (error bars-SD, P
 382 < 0.05 , Student's *t*-test).

383
384 The treatment with bioactive GA₄₊₇ gibberellins indicated hypersensitivity of *swi3b-3* to GA
385 demonstrated by hypocotyl length, while the response of *er-105* was reduced. By contrast, the
386 GA hypersensitivity of *swi3b-3* was abolished by introduced *er-105* mutation (Figure 4B). All
387 tested genotypes were responding to PAC treatment in a similar way (Figure 4C, D). The higher
388 expression of *SWI3B* was visible in the case of the *swi3b-3* mutant, which was even more
389 pronounced in *er-105/swi3b-3*. The expression of *SWI3B* was elevated after supplementation with
390 bioactive GA₄₊₇ in all genotypes, except for the *swi3b-3* line (Figure 4E). The examination of
391 *GIDI* and GA biosynthesis genes indicated that almost all examined lines responded to GA
392 treatment while the *swi3b-3* line was insensitive for induced by GA transcriptional changes
393 except for *GA20ox2* expression (Figure 4F, G). Collectively, our results further indicate that both
394 ERF signaling and SWI3B-containing CRCs play together an important role in the fine-tuning of
395 GA signaling in Arabidopsis.

396 To verify the biological effect of observed interactions between ERF signaling and SWI3B-
397 SWI/SNF, we analyzed the chromatin status, nuclei shape and chromocenters number in the
398 *er/erl1/erl2* mutant plants. We found that *er/erl1/erl2* plants exhibit increased chromocenter
399 number and altered spindle-like nuclei shape (Supplemental Figure 11A, B). We furthermore
400 screened the effect of inactivation of ERfs on genome-wide nucleosome positioning in chromatin
401 using *micrococcal nuclease* protection assays followed by deep sequencing (MNase-seq) and
402 confirmatory MNase-qPCR in WT and *er/erl1/erl2* plants.

403 We found that inactivation of ERF proteins has a broad influence on the global
404 nucleosomal chromatin structure in Arabidopsis- *erf* exhibited 41519 nucleosome occupancy
405 changes, 13924 "fuzziness" changes and 4055 nucleosome position changes (Supplemental
406 Figure 12A) and alterations in the presumable regulatory regions upstream of the transcription
407 start site (TSSs) (Supplemental Figure 12B, Supplemental Dataset 1, Sub-Table 9-14).

408 Among genes with down-regulated expression and altered nucleosome positioning in the
409 *er/erl1/erl2* mutant were 14 GA-related genes (*ATBETAFRUCT4*, *XERICO*, *PRE1*, *MYBR1*,
410 *MYB24*, *MIF1*, *HAI2*, *ZPF6*, *GA20ox1*, *CGA1*, *XTH24*, *GID1b*, *RGL1*, and *GIS3*) Interestingly,
411 seven of them (*ATBETAFRUCT4*, *PRE1*, *MYBR1*, *MIF1*, *XTH24*, *GID1b*, and *RGL1*) were
412 already observed to be directly targeted by the BRM ATPase of the SWI/SNF CRC (Archacki et
413 al., 2016; Li et al., 2016).

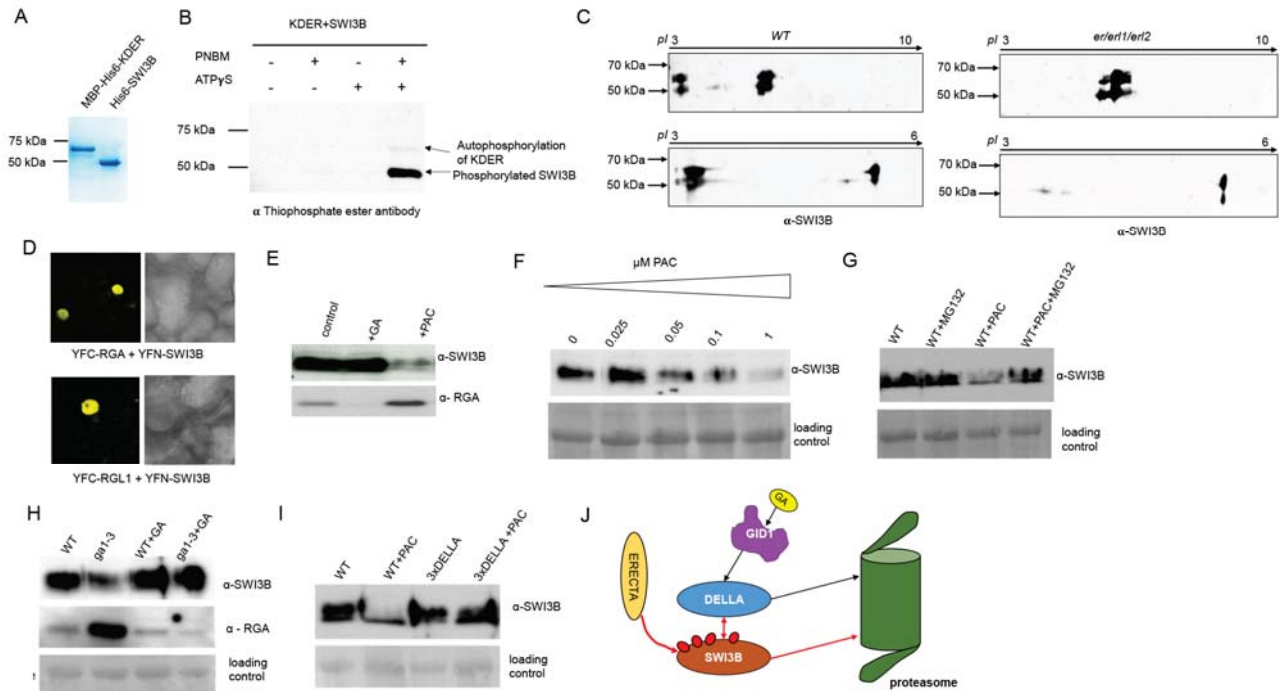
414 An Integrated Gene Browser (IGB) view of *PRE1*, *GID1a,b* promoter regions indicated
415 (Supplemental Figure 12D) various nucleosome alterations on promoter regions of these genes in
416 the *er/erl/erl2* mutant pointing out impaired chromatin remodeling in the absence of functional
417 ERF proteins. The selected changes were confirmed by MNase-qPCR (Supplemental Figure 12E).

418

419 **The Inactivation of ERF proteins Affects SWI3B Protein Phosphorylation**

420 We tested the ability of KDER to phosphorylate SWI3B protein. We overexpressed,
421 purified, and subsequently used MBP-His6-KDER and His6-SWI3B (Figure 5A, Supplemental
422 Figure 13A) for non-radioactive *in vitro* kinase assay. The existence of a strong band
423 corresponding to phosphorylated SWI3B protein and a weaker band of autophosphorylated
424 KDER was indicated (Figure 5B, Supplemental Figure 13B, C). The confirmatory mass-
425 spectrometry analysis resulted in the identification of the active phosphorylation sites at KDER
426 and in the SWI3B protein (Supplemental Figure 13D, E). Interestingly three of four KDER-
427 dependent phosphorylation sites were located in SWI3B in SWIRM and SANT domains
428 (Supplemental Figure 13E, F), providing a valuable hint that the ERF family proteins may be
429 responsible for the SWI3B phosphorylation.

430



431
 432 **Figure 5.** Erf proteins are responsible for the phosphorylation of SWI3B protein, while DELLA
 433 proteins control SWI3B protein abundance (See also Figures S13, S14, and S15). A, Coomassie
 434 staining of MBP-His6-KDER and His6-SWI3B proteins purified from bacteria. B, Western blot
 435 with anti-Thiophosphate ester antibody (ab92570; Abcam) showing *in vitro* SWI3B
 436 phosphorylation by KDER. C, 2D Western blot assay with anti SWI3B antibody indicating *in*
 437 *vivo* phosphorylation alteration of SWI3B protein in *er/er11/er12* mutant. D, SWI3B and RGA
 438 and RGL1 proteins in the nuclei of living cells. Bimolecular Fluorescence Complementation
 439 assay (BiFC) in epidermis of tobacco leaves. Scale bar = 10 μ m. E, The amounts of SWI3B and
 440 RGA proteins in plants are oppositely regulated by PAC treatment. F, The disappearance of
 441 SWI3B protein is PAC-dose dependent. G, The PAC-dependent degradation of SWI3B is
 442 abolished by the MG132 treatment, a known proteasome inhibitor. H, The *gal-3* mutant
 443 constitutively accumulating DELLA proteins exhibits the decreased level of SWI3B, which is
 444 restored to WT levels upon GA treatment. I, The triple DELLA mutant exhibits a WT-like level
 445 of SWI3B protein, and the PAC treatment does not influence SWI3B level in this background. J,
 446 Schematic model highlighting Erf and DELLA impact on the SWI3B protein.

447

448 To verify this possibility the *in vivo* phosphorylation analysis using 2D-IEF-PAGE
449 combined with the Western blot was performed. The alteration in the SWI3B proteins isoelectric
450 point (*pI*) in *er/erl1/erl2* mutant was observed. The bands corresponding to phosphorylated
451 SWI3B form were nearly absent in *er/erl1/erl2* plants, indicating a severe defect in SWI3B
452 phosphorylation (Figure 5C). This data strongly supports the regulatory function of the ERF
453 proteins on the SWI3B subunit of SWI/SNF CRC.

454 455 **Accumulation of DELLA Proteins Correlates with Increased Proteasomal Degradation of** 456 **SWI3B**

457 Our previous study demonstrated that SWI3C, a partner of SWI3B, physically interacts
458 with DELLA proteins (Sarnowska et al., 2013). We also found that the Arabidopsis lines with
459 impaired SWI/SNF CRCs- *brm* and *swi3c* exhibit decreased level of bioactive GA₄ gibberellins
460 level (Sarnowska et al., 2013; Archacki et al., 2013), but they do not accumulate RGA DELLA
461 protein similarly as in case of *er/erl1/erl2* plants (Supplemental Figure 14 and Figure 1J). To
462 address this unusual phenomenon, we used a BiFC assay to analyze the interaction between
463 DELLA and SWI3B. The interaction between either RGA or RGL1 protein and SWI3B was
464 found (Figure 5D, Supplemental Figure 15). No YFP signal was detected in control cells.

465 To understand the functional consequences of the detected interactions between SWI3B
466 and DELLA proteins, we analyzed the amounts of SWI3B and RGA proteins in GA or PAC-
467 treated plants (Figure 5E). Surprisingly, we observed the PAC-dose-dependent disappearance of
468 SWI3B protein (Figure 5F). To check if the degradation of SWI3B under these conditions
469 depended on the proteasome, we tested the effect of MG-132 on the SWI3B level. MG-132
470 treatment caused increasing SWI3B abundance in PAC treated plants (Figure 5G), suggesting
471 that the degradation of SWI3B observed in parallel to accumulation of DELLAs occurs *via* the
472 proteasome. We also observed increased degradation of SWI3B in the *gal-3* mutant in which
473 DELLA proteins are constitutively accumulated (Figure 5H), but we did not observe enhanced
474 SWI3B degradation in PAC-treated 3xDELLA (Archacki et al., 2013) collectively suggesting
475 that binding of SWI3B by DELLA proteins may be a primary cause of its proteasomal
476 degradation (Figure 5I, J). Thus, the accumulation of DELLA proteins should lead to the same
477 consequences as the elimination of SWI3B protein or SWI3B-containing SWI/SNF CRCs. This
478 conclusion is strongly supported by the lack of RGA protein accumulation in GA deficient *brm*

479 and *swi3c* lines with inactivated other subunits of SWI/SNF CRC (Archacki et al., 2013;
480 Sarnowska et al., 2013). Therefore, it could be indeed expected that GA and SWI3B-deficient
481 *er/er11/er12* mutant will also not accumulate RGA protein because, in the case of SWI/SNF CRC
482 impairment, the DELLA accumulation seems to be irrelevant. Collectively, our results provide
483 new insight into the functioning of the ERECTA family proteins and DELLA proteins and their
484 mutual impact on the SWI3B-containing SWI/SNF CRCs.

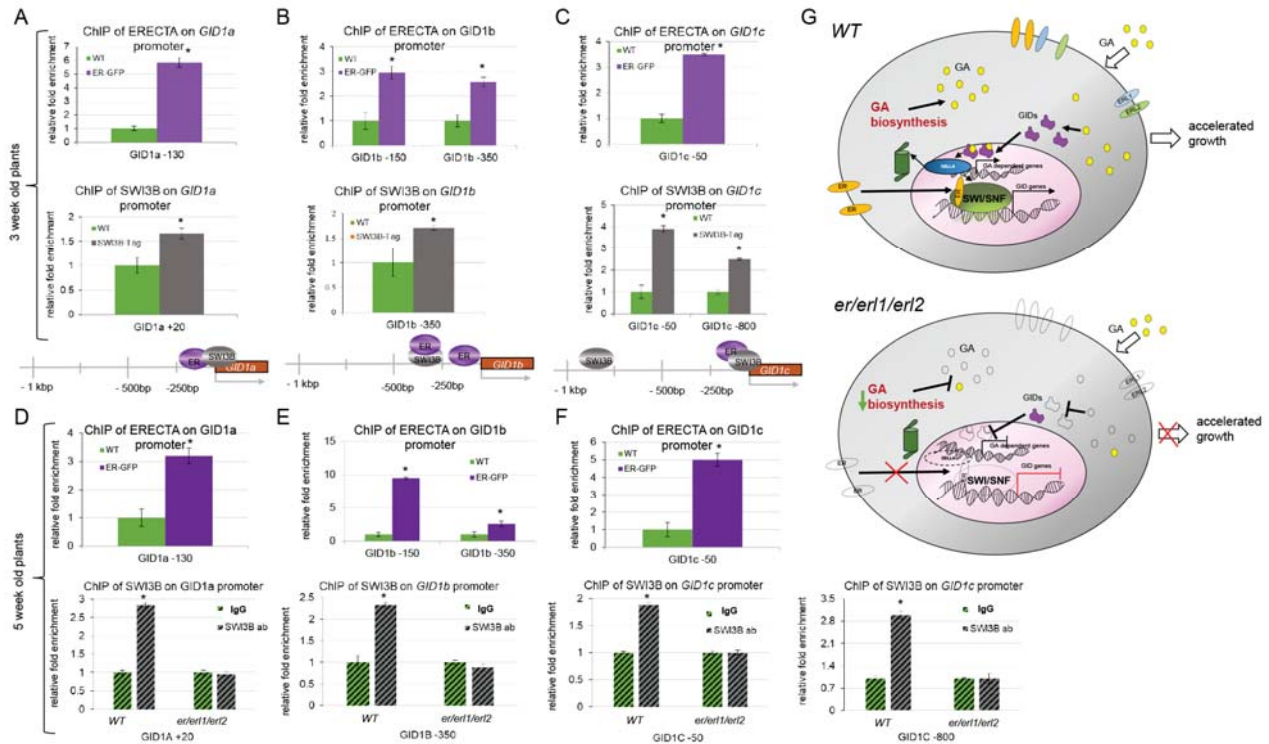
485

486 **Binding of ERECTA and SWI3B to Promoter Regions of the *GIDI* Genes**

487 The *er/er11/er12* mutant displays an impaired response to exogenous GA treatment and a
488 consistently decreased expression of all three *GIDI* genes. ERf proteins interact with the SWI3B,
489 inactivation of *ERf* proteins results in nucleosomal chromatin structure alterations and decreased
490 abundance of SWI3B and its phosphorylated form in Arabidopsis, and there is an intriguing
491 interdependence between the control of the SWI3B level and DELLA protein accumulation,
492 therefore we examined if ER and the SWI3B participate in transcriptional control of the *GIDI*
493 genes previously reported as targets for DELLA (Rosa et al., 2015). The ChIP analysis on *GIDI*
494 promoters was performed using nuclei purified from 3-week-old seedlings expressing the ER-
495 GFP and the SWI3B-HIS-STREP-HA proteins.

496 The binding of ER-GFP was detected around -130bp upstream of the TSS in the *GIDIa*
497 promoter region while SWI3B bound around the TSS of the *GIDIa* promoter (Figure 6A). The
498 ER protein was targeted to two regions around -150bp and -350bp from the TSS in the *GIDIb*
499 promoter, SWI3B was localized only -350bp upstream TSS (Figure 6B). ER and SWI3B were
500 similarly cross-linked to the -100bp region of the *GIDIc* promoter, but SWI3B was also mapped
501 further upstream to -800bp (Figure 6C).

502



503
 504 **Figure 6.** ERf proteins enter the nucleus where ERECTA protein binds the *GID1* promoters
 505 similarly to the SWI3B subunit of SWI/SNF CRC (See also Figures S16 and S17). A, ERECTA
 506 protein binds to promoter regions of the *GID1a* gene in a region targeted by the SWI/SNF
 507 complex in three-week-old plants. (error bars refer to SD, $P < 0.05$, Student's *t*-test, three
 508 biological and technical replicates were performed). B, ERECTA and SWI3B core subunit of
 509 SWI/SNF CRCs target promoter regions of *GID1b* gene in three-weeks old plants (error bars
 510 refer to SD, $P < 0.05$, Student's *t*-test, three biological and technical replicates were performed).
 511 C, SWI3B binds to the promoter region of the *GID1c* gene in two different regions. One of them
 512 is targeted by ERECTA protein in three-week-old plants. D, ERECTA protein binds to promoter
 513 regions of the *GID1a* gene in a region targeted by the SWI/SNF complex in five-week-old plants.
 514 (error bars refer to SD, $P < 0.05$, Student's *t*-test, three biological and technical replicates were
 515 performed). E, ERECTA targets promoter region of *GID1b* gene in five-week-old plants (error
 516 bars refer to SD, $P < 0.05$, Student's *t*-test, three biological and technical replicates were
 517 performed). F, ERECTA binds to the promoter region of the *GID1c* gene in three-week-old
 518 plants. The bottom panel in D-F: the binding of native SWI3B protein to its target sites in *GID1a*-
 519 *c* promoter regions is abolished in 5-week-old *er/er1/erl2* triple mutant plants. G, A model
 520 describing the non-canonical nuclear function of ERf proteins in the GA signaling pathway.

521
522 Inspection of ER and SWI3B binding to the *GID1a-c* promoter regions in 5-week-old
523 WT, ER-GFP, and the *er/erl1/erl2* mutant demonstrated that ER binds the same *GID1* promoter
524 regions as in the case of 3-week-old plants (Figure 6 D-F). The SWI3B binding to the promoter
525 of *GID1* genes was abolished by the inactivation of ERF proteins in *er/erl1/erl2* plants. The
526 inactivation of *ER*, *ERL1*, or *ERL2* did not affect the binding of SWI3B to *GID1a-c* promoter
527 regions in single *er105*, *erl1*, and *erl2* mutant lines (Supplemental Figure 16). Our study provides
528 evidence that the three ERF proteins have redundant functions regarding proper SWI3B
529 recruitment since only the simultaneous absence of all ERfs proteins abolished SWI3B binding to
530 the *GID1a-c* promoters.

531 Of note, we found a similar binding of HER2 (EGFR-family) receptor and BAF155
532 subunit of SWI/SNF CRCs to the promoter regions of human *BRCA1* and *FBP1* genes
533 (Supplemental Figure 17), indicating that the phenomenon observed for ER may be a general
534 mechanism controlling gene expression that is maintained between kingdoms.

535

536 **Discussion**

537 Inactivation of ERf LRR-RLK family members results in various defects in Arabidopsis
538 growth and development. While it is well established that ERf proteins play distinct roles in the
539 control of epidermal patterning, stomatal development, meristem size, inflorescence architecture,
540 and hormonal signaling, the exact mechanisms underlying the regulatory functions of ERf
541 proteins in these processes are largely unknown (*e.g.*, Chen and Shpak, 2014; Chen et al., 2013b;
542 Qi et al., 2004; Van Zanten et al., 2010; Kosentka et al., 2019).

543 Here we show that the inactivation of Arabidopsis ERf proteins has a broad effect on
544 various regulatory processes, including hormonal signaling, and suggest that these sum responses
545 underlie the severe developmental defects exhibited by the *er/erl1/erl2* mutant. We demonstrate
546 that parallel inactivation of all *ERf* proteins results in severe deregulation of the GA signaling
547 pathway as evidenced by the impairment of GA perception and GA biosynthesis. When taken
548 together, these findings, alongside the identification of NLS and NES sequences in ERf proteins
549 and our demonstration of their translocation into the nucleus, suggest a novel, non-canonical
550 function of ERf proteins (Figure 6G).

551 Our study also reveals an analogy of this system to the previously described XA21 LRR
552 immune receptor in rice (Park and Ronald, 2012) and to the non-canonical signaling mode of the
553 human Epidermal Growth Factor Receptor (EGFR) family (Lee et al., 2015a). Although it should
554 be stressed that these two classes of plant and animal epidermal receptors carry completely
555 unrelated sequences from one another and from the system we describe here (Supplemental Table
556 3), suggesting that the translocation of the membrane receptors to the nucleus may be a general
557 paradigm maintained between plant and animal kingdoms. In addition to their canonical
558 membrane receptor functions, holoreceptor and truncated forms of EGFRs are imported into
559 nuclei *via* ER-mediated retrograde transport, although some of them lack known NLSs (Chen and
560 Hung, 2015).

561 In the nucleus, the EGFR receptors can bind to DNA, interact with various transcription
562 factors. Thereby, nuclear forms of EGFRs are implicated in the control of cell proliferation, DNA
563 replication and repair, and transcription (Chen and Hung, 2015), so their functions extend far
564 beyond the regulation of epidermal patterning.

565 We found here that the Arabidopsis ERECTA LRR-RLK receptor similarly translocates
566 from the plasma membrane into the nucleus. Both intact and N-terminally truncated forms of

567 ERECTA were detectable in the nucleus. A truncated ERECTA carrying only the kinase domain
568 localizes exclusively in the nucleus and partially complements the leaf developmental defects
569 caused by the *er-105* mutation implying a ligand-independent non-canonical signaling function of
570 the ERECTA kinase domain.

571 Furthermore, our data show that the ERF proteins interact through their kinase domains
572 with the SWI3B, and ER can phosphorylate the SWI3B core subunit of the SWI/SNF CRC.
573 SWI/SNF plays a pivotal role in the hormonal crosstalk regulation in both humans and plants
574 (Sarnowska et al., 2016). Moreover, we show that analogously as ER protein, the HER2 member
575 of the EGFR family directly interacts with the BAF155 subunit of human SWI/SNF and co-
576 localizes with BAF155 on some gene promoters providing evidences that such system is likely
577 maintained between kingdoms.

578 Parallel inactivation of all ERF proteins results in alterations of genome-wide nucleosomal
579 chromatin structure and altered transcriptional activity of a large number of genes. The binding of
580 SWI3B to its target regions in the *GID1a-c* promoters is retained in *er-105*, *erl1*, and *erl2* single
581 mutant lines. By contrast, the *er/erl1/erl2* mutant plants exhibited a reduction in phosphorylation
582 SWI3B protein level and abolished proper SWI3B binding to *GID1* promoter regions together
583 with decreased expression of *GID1a-c* genes, indicating a strong and direct effect of the ERF
584 signaling pathway on SWI/SNF-dependent chromatin remodeling. The *er/erl1/erl2* mutant plants
585 are characterized by a decreased level of endogenous gibberellins; however, they do not
586 accumulate DELLA proteins, similar to the SWI/SNF mutants. Thus, we have demonstrated that
587 the *er/erl1/erl2* mutant plants exhibit severe deregulation of the gibberellin signaling pathway
588 and SWI/SNF-dependent chromatin remodeling. This is, in turn, an attractive explanation of the
589 observed insensitivity of *er/erl1/erl2* mutant to the application of exogenous gibberellin. DELLA
590 proteins are involved in the sequestration of various transcription factors and chromatin
591 remodeling complexes (Phokas and Coates, 2021). In this study, we extend the existing
592 knowledge on DELLA functioning by providing evidence for the existence of the DELLA-
593 SWI3B regulatory module and explaining why some GA-deficient mutant lines with impaired
594 SWI/SNF chromatin remodeling complex do not accumulate DELLA proteins (Figure 5G).

595 Collectively our finding that plant ERF proteins play an important, non-canonical nuclear
596 function, *i.e.*, bind directly to chromatin and control the proper recruitment of SWI/SNF CRCs,
597 which are strongly involved in controlling regulatory processes including hormonal crosstalk

598 (Sarnowska et al., 2016), may be a general paradigm for other classes of plant and mammalian
599 membrane receptor kinases.

600

601 **Methods**

602 **Plant Material and Growth Conditions**

603 The *Arabidopsis thaliana* ecotype Columbia was used as wild type (WT) in all
604 experiments. The following *Arabidopsis* mutants were used for analysis: *er-105*, *er-105/swi3b-3*
605 (Sáez et al., 2008), *er/er11/er12* plants (Shpak et al., 2004) and *erf* lines in various combinations
606 (Torii et al., 1996), the 35S::GFP *Arabidopsis* line has been obtained from NASC (N67775).
607 Seeds were sown on soil or plated on ½ Murashige and Skoog medium (Sigma-Aldrich)
608 containing 0.5% sucrose and 0.8% agar. Plants were grown under long day (LD) condition (12h
609 Day/12h Night or 16h Day/8h Night). For GA response tests, plants were sprayed twice a week
610 with 100 μM GA₄₊₇ or water (control) for a fast response the 2h of GA₄₊₇ treatment was
611 performed.

612 **Construction of Transgenic Lines**

613 Genomic sequences of *ERECTA*, cDNAs of *ERECTA* kinase domain and truncated
614 *ERECTA* lacking kinase domain (ERΔK) were cloned into binary vector p35S::GW::GFP (F.
615 Turck, Max-Planck-Institut für Züchtungsforschung, in the case of *ERECTA*), and into pEarley
616 Gate 101 (in the case of the ER kinase domain and ΔKDER; (Earley et al., 2006). Plants were
617 transformed using *Agrobacterium tumefaciens* GV3101 (pMP90) by floral-dip method (Davis et
618 al., 2009). The STOP codon of the *SWI3B* genomic sequence was replaced with HIS-STREP-HA
619 using the recombineering method (Bitrián et al., 2011), moved into pCB1 vector (Heidstra et al.,
620 2004), and transformed into *swi3b-2* *Arabidopsis* mutant line.

621 **RNA Extraction and qRT-PCR Analysis**

622 Total RNA was isolated from adult (5-week-old) plants using an RNeasy plant kit
623 (Qiagen), treated with a TURBO DNA-free kit (Ambion). Total RNA (2.5μg) was reverse
624 transcribed using a first-strand cDNA synthesis kit (Roche). qRT-PCR assays were performed
625 with SYBR Green Master mix (Bio-Rad) and specific primers for PCR amplification.
626 Housekeeping genes *PP2A* and *UBQ5* (AT1G13320 and AT3G62250, respectively) were used as
627 controls. The relative transcript level of each gene was determined by the $2^{-\Delta\Delta C_t}$ method
628 (Schmittgen and Livak, 2008). Each experiment was performed using at least three independent
629 biological replicates. qRT-PCR primers are listed in Supplemental Dataset 3.

630 **Transcript Profiling and Gene Ontology Analysis**

631 RNA was isolated from adult (5-week-old) WT and *er/erl1/erl2* plants using a Plant
632 RNeasy kit (Qiagen) according to the manufacturer's protocol. Transcriptomes were analyzed
633 using 150ng of total RNA as starting material. Targets were prepared with a cDNA synthesis kit
634 followed by biotin labeling with the IVT labeling kit (GeneChip 39IVT Express; Affymetrix) and
635 hybridized to the ATH1 gene chip for 16h as recommended by the supplier. The raw data were
636 analyzed using GenespringGX according to the manual (guided workflow). GO-TermFinder was
637 used for GO analyses of selected groups of genes (Boyle et al., 2004).

638 **Nuclear Fractionation**

639 Nuclei were isolated from 2g of leaves of 3-weeks old Arabidopsis seedlings according to
640 the method previously described by Gaudino and Pikaard (1997). Subsequent nuclear
641 fractionation was performed using the high-salt method, with modifications (Sarnowski et al.,
642 2002).

643 **Protein Interaction Study, Confocal Imaging, Subcellular Localization, Brefeldin A and** 644 **Leptomycin B Treatment, DAPI Staining**

645 Protein interaction was analyzed by performing the immunoprecipitation of ER-GFP or
646 KDER-YFP-HA from nuclei from 4 g of Arabidopsis plants (Saleh et al., 2008). The nuclear
647 extracts were incubated with 25 μ L of GFP Magnetic Trap beads (Chromotek) according to
648 manufactures instructions. The presence of SWI3B protein was determined by western blot
649 analysis using anti-SWI3B antibody (Sarnowski et al., 2002).

650 The interaction between human proteins was analyzed by immunoprecipitation of HER2
651 and BAF155 from viscolase treated nuclear extracts prepared, according to Jancewicz et al. 2021.
652 The presence of HER2 and BAF155 was determined by Western blot analysis using anti HER2
653 (CST, 12760) and anti BAF155 (CST, 11956) antibodies.

654 To obtain YFN-ERL1, YFC-ERL1, YFC-ERL2, and YFC-KDER fusions for BiFC (Hu et
655 al., 2002) analysis, cDNAs encoding ERL1 and ERL2 proteins and ERECTA, ERL1, and ERL2
656 C-terminal kinase domains were PCR amplified and cloned into the binary vectors pYFN43 or
657 pYFC43 (Belda-Palaz3n et al., 2012). The *in vivo* interactions between proteins were detected by
658 BiFC using Leica TCS SP2 AOBS, a laser scanning confocal microscope (Leica Microsystems).
659 Tobacco (*Nicotiana benthamiana*) epidermal cells were infiltrated using *Agrobacterium*
660 *tumefaciens* GV3101 (pMP90) carrying plasmids encoding ERL1, ERL2, or KDER fusions and
661 the p19 helper vector and analyzed by confocal microscopy 3 d later. YFN-RFP and YFC-RFP

662 fusions were used to detect transformed cells in the BiFC assays (Sarnowska et al., 2013); at least
663 five nuclei were analyzed in three separate experiments.

664 The vesicle trafficking inhibitor BFA (Sigma Aldrich) was used at the 25 μ M
665 concentration at the following time points 40 min, 90 min, and 120 min. The NES-dependent
666 nuclear export inhibitor Leptomycin B was used at the 200 nM concentration 4h before
667 microscopy observation. Nuclei were stained with 4',6-diamidino-2-phenylindole (DAPI) at the
668 1 μ g/mL concentration for 30 min. The observation was carried out on the root tip of about two
669 weeks old plants incubated directly before in 1/2 MS alone or with the addition of proper
670 compound (BFA or Leptomycin B, respectively). Every time 30 min before the end of
671 incubation, DAPI was added.

672 **Chromatin Immunoprecipitation**

673 ChIP experiments were performed as described previously (Sacharowski et al., 2015) on
674 three or five-week-old WT, ER-GFP, SWI3B-His-Strep-HA, and *er/er1/erl2* plants cross-linked
675 under vacuum using formaldehyde (final concentration: 1%) and Bis-(sulfosuccinimidyl)
676 glutarate (final concentration: 1mM). For ER-GFP, chromatin immunoprecipitation was
677 performed with GFP-Trap M (Chromotek). For SWI3B ChIP experiments, NiNTA Agarose
678 (Qiagen) or, in the case of anti-SWI3B antibody, the magnetic protein A and G dynabeads
679 (Dynal) were used. ChIP enrichment was determined using qPCR, and relative fold change was
680 calculated using the $2^{-\Delta\Delta C_t}$ method (Schmittgen and Livak, 2008). The TA3 retrotransposon was
681 used as negative control (Pastore et al., 2011). Primers used in ChIP experiments are listed in
682 Supplemental Dataset 3.

683 Chromatin from the SKBR-3 human cell line was immunoprecipitated according to
684 (Komata et al., 2014 and Jancewicz et al., 2021). Recovered chromatin was incubated O/N at 4°C
685 with the following antibodies: anti BAF155 (CST, 11956), antiHER2 (CST, 12760), and Normal
686 Rabbit IgG (CST, 2729, mock control). Results were calculated based on the $2^{-\Delta\Delta C_t}$ (Schmittgen
687 and Livak, 2008). The relative fold enrichment of the analyzed sample represents the fold change
688 with reference to IgG (mock) sample. A set of primers used for ChIP-qPCR analysis is listed in
689 Supplementary Dataset 3.

690 **MNase Mapping of Genome-wide Nucleosome Positioning and MNase-qPCR**

691 The nuclear extraction, MNase treatment, subsequent NGS analyses, and confirmatory
692 MNase-qPCR were performed according to (Sacharowski et al., 2015) on 5 week old plant
693 material.

694 **Gibberellin Analysis**

695 About 200 mg of frozen materials from 5 week old plants were used to extract and purify the GA
696 as described in Plackett et al. (2012) with minor modifications. GA was quantified using MS/MS
697 analysis using 4000 Triple Quad (AB Sciex Germany GmbH, Darmstadt, Germany) in multiple
698 reaction monitoring (MRM) scan with electrospray ionization (ESI) as described in Salem et al.,
699 (2016). The mass spectrometry attached to UPLC system (e.g., Waters Acquity UPLC system,
700 Waters, Machester, UK) separation was achieved on a reversed phase C18-column (100 mm ×
701 2.1 mm 1.8 μm).

702 ***In vitro* Phosphorylation Analysis and *in vivo* Kinase Assay**

703 SWI3B-6xHis was overexpressed and purified, according to Sarnowski et al. (2002). The
704 KDER (pDEST-6xHis-MBP vector) was purified using tandem purification using MBP and Ni-
705 NTA resins. *In vitro* kinase assay was performed according to the method described by (Allen et
706 al., 2007). Phosphorylation was detected by Western blot analysis using an anti-Thiophosphate
707 ester antibody (ab92570; Abcam) and by the MS/MS analysis.

708 *In vivo* phosphorylation analysis was performed using a 2D western blot assay on nuclear
709 extracts from 5 weeks old WT and *er/er1/er2* plants (Saleh et al., 2008). For isoelectrofocusing
710 (IEF), nuclear proteins were prepared according to Kubala et al. (2015). The IEF was performed
711 on the 7cm length gel strips with immobilized pH gradients 3-10 and 3-6 (BioRad). After IEF,
712 the equilibration of immobilized pH gradient was performed according to Wojtyla et al. (2013).
713 The SWI3B protein was detected by Western blotting using an anti SWI3B antibody (Sarnowski
714 et al., 2002). The *in vivo* phosphorylation was identified based on the changes of SWI3B
715 isoelectric point (*pI*) (Mayer et al., 2015).

716 **Accession Numbers**

717 Microarray and MNase-seq data are available in the ArrayExpress database
718 (www.ebi.ac.uk/arrayexpress) under E-MTAB-5595 and E-MTAB-5830 accession numbers,
719 respectively.

720

721 **Supplemental Data**

722
723
724 **Supplemental Figure 1.** Comparative analysis of genes with altered expression in *er/er11/erl2*
725 and *gal-3* mutants. A, Combinations of *erf* mutants have distinct effects on height of Arabidopsis
726 plants. Error bars refer to SD,* = $P < 0.05$, Student's *t* test. B, Genes showing altered expression
727 in the *er/er11/erl2* mutant plants. C, Venn diagrams indicating genes contrastingly up-regulated in
728 *er/er11/erl2* and down-regulated in *gal-3* plants. D, Venn diagrams indicating genes contrastingly
729 down-regulated in *er/er11/erl2* and up-regulated in *gal-3* plants. E, ERF proteins control the
730 expression of genes down-regulated in *gal-3*, which are either DELLA repressed or DELLA
731 independent. F, ERF proteins control the expression of genes up-regulated in *gal-3*, which are
732 either DELLA activated or DELLA independent.

733
734 **Supplemental Figure 2.** The *er/er11/erl2* mutant displays severely impaired response to
735 exogenous GA treatment and slightly enhance *gal-3* phenotypic traits. A, The 14 days old
736 *er/er11/erl2* plants treated with GA₄₊₇ did not show rosette expansion indicating defects in GA
737 response. Error bars refer to SD,* = $P < 0.05$, Student's *t* test, n= 30 plants. B, Two-month-old
738 GA₄₊₇ treated *er/er11/erl2* plants show accelerated flowering compared to mock treated control.
739 Scale bar= 1cm. n= 30 plants. C, Two-months old *er/er11/erl2* plants. Scale bar= 1cm. n= 30
740 plants. D, Three-weeks old *gal-3* plants crossed with *erf* mutants in various combinations show
741 mostly the phenotypic traits characteristic for *gal-3*. Scale bar= 1 cm. E, The phenotypic traits of
742 5-weeks old plants carrying *erf* mutations crossed with *gal-3* in various combinations grown in
743 long day conditions. Scale bar= 1 cm.

744
745 **Supplemental Figure 3.** *er/er11/erl2* plants exhibit deficiency in gibberellin intermediates.
746 Left panel: *er/er11/erl2* mutant exhibits dramatically reduced level of GA₁₂ and GA₂₄ gibberellin
747 intermediates (error bars-SD, $P < 0.05$, Student's *t*-test, three biological and technical replicates
748 were assayed). Right panel: schematic representation of alteration in GA biosynthesis pathway in
749 *er/er11/erl2* plants.

750
751 **Supplemental Figure 4.** 35S::ERECTA-GFP construct complements the *er-105* mutation.

752

753 **Supplemental Figure 5.** ERECTA protein is detected in various cell compartments including
754 endosomes and accumulate in the nuclei periphery after BFA treatment. A, In cell pairs of
755 stomata, ERECTA-GFP was detected in circles around the positions of nuclei. Scale bar=10 μ m.
756 B, Accumulation of ERECTA protein in the BFA bodies after Brefeldin A treatment. Note the
757 enhanced presence of BFA bodies after 40 min (mid column) and 90 min (right column) BFA
758 treatment. Scale bar=10 μ m.

759
760 **Supplemental Figure 6.** The ERL1 and ERL2 proteins carry defined NLS in their kinase
761 domains. A, The NLS prediction in the kinase domain of ERL1 and ERL2 proteins has been done
762 using cNLS mapper (Kosugi et al., 2009a; Kosugi et al., 2009b). NLS score in range 5-7 means
763 that protein is partially localized in the nucleus and cytoplasm. Bottom panel: The alignment of
764 ERECTA, ERL1, and ERL2 protein sequences (part of kinase domains carrying NLS) using
765 PRALINE indicates high amino-acid sequence conservation between analyzed proteins.
766 Consistency is determined within range 1-10, where 1 means least conserved substitution and 10-
767 the most conserved substitution (Simossis et al., 2005). B, Western blot analysis with anti-GFP
768 antibody confirms nuclear localization of ERECTA protein which undergoes proteolytic
769 processing. The samples were standardized by western blotting with anti H3 antibody. C,
770 Schematic presentation of full length and deletion variants of ERECTA protein used for the
771 localization study. ER – ERECTA protein with complete amino acids sequence; Δ KDER –
772 truncated ERECTA protein lacking kinase domain; KDER – the kinase domain of ERECTA
773 protein.

774
775 **Supplemental Figure 7.** The kinase domain of ERECTA (KDER) has ability to complement the
776 *er-105* leaf phenotypic traits. A, Rosette leaves of WT (upper), *er-105* (mid), and *er-105*/KDER-
777 YFP (lower panel). Graphical alignment of corresponding leaves indicating partial
778 complementation of *er* phenotypic traits by KDER. Scale bar= 1cm. B, Cauline leaves of WT
779 (upper), *er-105* (mid), and *er-105*/KDER-YFP (lower panel). Graphical alignment of
780 corresponding laves indicating partial complementation of *er* phenotypic traits by KDER. Scale
781 bar= 1cm. C, The kinase domain of ERECTA cannot restore all (i.e., stem elongation) phenotypic
782 traits of the *er-105* mutant line. Scale bar= 1cm.

783

784 **Supplemental Figure 8.** ERECTA protein enters to the nucleus and localizes in various sub-
785 nuclear fractions.

786
787 **Supplemental Figure 9.** Negative controls for bimolecular fluorescence complementation assay.
788 Negative controls for BiFC interaction analysis of ER, ERL1, ERL2 kinase domains fused to
789 YFC and SWI3B fused to YFN, including the RFP channel. Scale bar 10 μ m.

790
791 **Supplemental Figure 10.** Human SWI3-type BAF155 co-precipitates with HER2 EGFR family
792 membrane receptor from human cells nuclei.

793
794 **Supplemental Figure 11.** *er/erl1/erl2* mutant plants show affected chromatin organization
795 demonstrated as altered chromocenters number.
796 Upper panel: exemplary pictures of WT and *er/erl1/erl2* nuclei.
797 Lower panel: calculation of chromocenters (n=20 nuclei for each genotype).

798
799 **Supplemental Figure 12.** ERF proteins inactivation has a severe impact on genome-wide
800 nucleosome positioning. A, Nucleosome changes identified in the *er/erl1/erl2* triple mutant
801 plants. B, Genome-wide nucleosome distribution patterns surrounding the transcription start site
802 (TSS). C, Nucleosome distribution patterns surrounding the TSS of GA-related genes showing
803 altered expression in the *er/erl1/erl2* triple mutant plants. D, The alteration of nucleosomal
804 structure on *PRE1*, *GID1a*, and *GID1b* loci misexpressed in the *er/erl1/erl2* triple mutant plants
805 and targeted by the SWI/SNF CRC. Red boxes indicate nucleosome alterations. E, Confirmatory
806 MNase-qPCR for selected genes with altered nucleosomes.

807
808 **Supplemental Figure 13.** Kinase domain of ERECTA phosphorylates SWI3B protein. A,
809 Western blot with anti His6 antibody for detection of MBP-His6-KDER and His6-SWI3B
810 proteins purified from bacteria. B, Western blot with anti-Thiophosphate ester antibody
811 indicating no phosphorylation of SWI3B protein in the absence of KDER (negative control). C,
812 Western blot with anti-Thiophosphate ester antibody (ab92570; Abcam) showing
813 autophosphorylation of KDER in the absence of SWI3B protein. D, Identification of active
814 phosphorylation sites in KDER by MS/MS analysis. E, Identification of active phosphorylation

815 sites in SWI3B by MS/MS analysis. F, KDER phosphorylates SWI3B at the SWIRM and SANT
816 domains.

817
818 **Supplemental Figure 14.** GA-deficient mutant lines with inactivated subunits of SWI/SNF
819 complexes do not accumulate RGA DELLA protein. A, GA-deficient *swi3c* plants are unable to
820 over accumulate RGA protein. B, GA-deficient *brm* plants are unable to over accumulate RGA
821 protein.

822
823 **Supplemental Figure 15.** Negative controls for bimolecular fluorescence complementation
824 assay.

825 Negative controls for BiFC interaction analysis of SWI3B, RGA, and RGL1 fused to YFC, and
826 SWI3B fused to YFN, including the RFP channel. Scale bar 10 μ m.

827
828 **Supplemental Figure 16.** Proper SWI3B binding to *GID1a-c* promoter regions is abolished in
829 the *er/erl1/erl2* whereas occurs in single *er-105*, *erl1* or *erl2* mutant lines. A, SWI3B targets
830 promoter region of *GID1a* gene in five-weeks old plants WT, *er-105*, *erl1* or *erl2* but is abolished
831 in triple *er/erl1/erl2* mutant plants (error bars refer to SD, $P < 0.05$, Student's t test, three
832 biological and technical replicates were used). B, SWI3B targets promoter region of *GID1b* gene
833 in five-weeks old plants WT, *er-105*, *erl1* or *erl2* but is abolished in triple *er/erl1/erl2* mutant
834 plants (error bars refer to SD, $P < 0.05$, Student's t test, three biological and technical replicates
835 were used). C, SWI3B targets promoter region of *GID1c* gene in five-weeks old plants WT, *er-*
836 *105*, *erl1* or *erl2* but is abolished in triple *er/erl1/erl2* mutant plants (error bars refer to SD, $P <$
837 0.05 , Student's t test, three biological and technical replicates were used).

838
839 **Supplemental Figure 17.** Human SWI3-type BAF155 targets together with HER2 EGFR family
840 membrane receptor *FBP1* and *BRCA1* genes *loci*. A, BAF155 subunit of human SWI/SNF
841 complex binds *Fructose-1,6-Bisphosphatase locus* together with HER2 member of EGFR
842 membrane receptor family. B, BAF155 subunit of human SWI/SNF complex binds *BRCA1 locus*
843 together with HER2 member of EGFR membrane receptor family.

844

845 **Supplemental Table 1.** Genes classified to “Response to Gibberellin” GO term and showing
846 down-regulated expression level in the *er/erl1/erl2* mutant.

847
848 **Supplemental Table 2.** Genes with up-regulated expression in *er/erl1/erl2* mutant plants
849 classified to GO-terms of leaf epidermal and stomatal cell differentiation.

850
851 **Supplemental Table 3.** Functional analogies between arabidopsis ERF proteins and the human
852 EGFR membrane receptors.

853
854 **Supplemental dataset 1.** Comparative analysis of transcript profiling and MNase-seq data.

855 **Sub-table 1.** Transcript profiling using ATH1 microarray analysis to identify genes down-
856 regulated in *er/erl1/erl2* mutant line.

857 **Sub-table 2.** Transcript profiling using ATH1 microarray analysis to identify genes up-regulated
858 in *er/erl1/erl2* mutant line.

859 **Sub-table 3.** GO analysis of genes down-regulated in *er/erl1/erl2* mutant line.

860 **Sub-table 4.** GO analysis of genes up-regulated in *er/erl1/erl2* mutant line.

861 **Sub-table 5.** Comparative transcript profiling analysis for genes down-regulated in *er/erl1/erl2*
862 and *gal-3* mutants lines.

863 **Sub-table 6.** GO analysis of *er/erl1/erl2* and *gal-3* down-regulated genes.

864 **Sub-table 7.** Comparative transcript profiling analysis for genes up-regulated in *er/erl1/erl2* and
865 *gal-3* mutants lines.

866 **Sub-table 8.** GO analysis of genes up-regulated in *gal-3* and *er/erl1/erl2*.

867 **Sub-table 9.** Genes with altered nucleosome positioning in promoter region -3000 to TSS.

868 **Sub-table 10.** GO analysis of genes with altered nucleosome positioning identified in promoter
869 region -3000 to TSS.

870 **Sub-table 11.** Genes with altered nucleosome positioning identified in promoter region -3000 to
871 TSS and down-regulated in *er/erl1/erl2* microarray.

872 **Sub-table 12.** Genes with altered nucleosome positioning identified in promoter region -3000 to
873 TSS and up-regulated in *er/erl1/erl2* microarray.

874 **Sub-table 13.** GO analysis for genes with altered nucleosome positioning identified in promoter
875 region -3000 to TSS and down-regulated in *er/erl1/erl2* microarray.

876 **Sub-table 14.** GO analysis for genes with altered nucleosome positioning identified in promoter
877 region -3000 to TSS and up-regulated in *er/erl1/erl2* microarray.

878

879 **Supplemental dataset 2.** Comparison of ER, ERL1, and ERL2 protein sequences with
880 highlighted important domains including NLS.

881 ClustalW was used to align ER, ERL1, and ERL2 sequences. Under conserved amino acid is
882 asterisk mark, highly similar or similar amino-acids are marked :: and . respectively. LRR domain
883 is marked as gray background, amino-acids crucial for EPFs or TMM interaction are indicated
884 with bold. Transmembrane domain, juxtamembrane domain, and kinase domain are indicated
885 with bold green, orange, and blue color fonts, respectively (Kosentka et al., 2017). The predicted
886 NLS sequence is marked in a dotted line frame (Kosugi et al., 2009b).

887
888 **Supplemental Dataset 3.** Primers used in this work.

889 **Supplemental Movie 1. The ERECTA protein undergoes endocytosis.**

890 **Supplemental Movie 2. The ERECTA protein undergoes endocytosis.**

891

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896 GA measurements and the Max Planck-Genome-Center Cologne
897 (<http://mpgc.mpipz.mpg.de/home/>) for performing the transcript profiling and MNase-seq
898 analysis described in this study.

899

900 **FIGURE LEGENDS**

901 **Figure 1.** *ERf* inactivation affects Arabidopsis development, causes transcriptomic changes
902 overlapping with the effect of *gal-3* mutation and impairs GA biosynthesis and signaling (See
903 also Figures S1, S2 and S3). A, Phenotypic changes conferred by combinations of *erf* mutations.
904 Scale bar= 1 cm. B, Overlapping down-regulated genes in *er/erl1/erl2* and *gal-3* plants. C,
905 Overlapping up-regulated genes in *er/erl1/erl2* and *gal-3* plants. D, The *er/erl1/erl2* plants
906 exhibit impaired GA response. 14- days old LD (12h day/12 night) grown WT and *er/erl1/erl2*,
907 sprayed twice a week with water (upper row) or 100 μ M GA₄₊₇ (lower row). Arrows-*er/erl1/erl2*
908 plants. Scale bar= 1cm. E, The GA response is retained to various levels in combinations of *erf*
909 mutants. Error bars-SD,* =P < 0.05, Student's *t*-test, n= 30 plants. F, The response of various *erf*
910 mutants to 1 μ M Paclobutrazol treatment. Error bars-SD,* =P < 0.05, Student's *t* test, n= 30
911 plants. G, The *er/erl1/erl2* mutant exhibits altered transcription of *GID1* GA receptor genes (error
912 bars-SD, P < 0.05, Student's *t*-test, three biological and technical replicates were assayed). H,
913 The *er/erl1/erl2* mutant displays altered GA biosynthesis and metabolism-related genes
914 expression (error bars-SD, P < 0.05, Student's *t*-test, three biological and technical replicates
915 were assayed). I, The *er/erl1/erl2* mutant exhibits dramatically reduced level of bioactive GA₄₊₇
916 gibberellin (error bars-SD, P < 0.05, Student's *t*-test, three biological and technical replicates
917 were assayed). J, The *er/erl1/erl2* mutant shows decreased level of the DELLA protein RGA.

918
919 **Figure 2.** Subcellular localization of ERECTA protein (See also Figures S4 and S5). A,
920 ERECTA is localized in plasma-membrane and endosomes in epidermal cells of 7-days old
921 seedlings. ER-GFP, or free GFP visualized using GFP channel. FM4-64 specifically stains
922 plasma-membranes. Scale bar=10 μ m. B, Root-tip images of approximately two-week-old (14-17
923 days) ER-GFP seedlings showing nuclear localization of ERECTA protein at considerable
924 frequency. C, Root-tip images of 12-day-old ER-GFP seedlings serving as the control for D and
925 E. D, Brefeldin A treatment enhanced the localization of ERECTA protein in Brefeldin A (BFA)
926 bodies. Roots of 12-day-old Arabidopsis seedlings. E, Leptomycin B treatment enhanced the
927 nuclear localization of ERECTA Free GFP was used as a control in C, D, and E, cell nuclei were
928 stained with DAPI, scale bar= 50 μ m. F, Letomycin B enhances nuclear presence of ER protein.
929 The GFP/DAPI ratio calculated per area for roots of plants expressing ER-GFP protein.

930

931 **Figure 3.** Nuclear function of ERf proteins (See also Figures S6, S7, S8, S9, and S10). A, Root-
932 tip images of approximately two-week-old (14-17 days) plants expressing ER-GFP, KDER-YFP-
933 HA (the kinase domain of the ER protein), ER Δ K-YFPHA (truncated ER protein lacking the
934 kinase domain) proteins indicating that the kinase domain is necessary for the nuclear localization
935 of ER protein. WT and GFP expressing plants-negative controls. Panel 2 in the ER-GFP indicates
936 nuclear localization of ER protein appearing at considerable frequency. Cell nuclei were stained
937 with DAPI. Scale bar=25 μ m. B, The *er-105/swi3b3* double mutant shows more retarded growth
938 than either *er-105* or *swi3b3* plants. Scale bar= 1cm. C, The *er/er11/erl2* triple mutant exhibits
939 reduced SWI3B protein level. D, ER-GFP or free GFP (negative control) pull-down from the
940 nucleus and anti-SWI3B western blotting indicate a specific ER-SWI3B interaction. E,
941 Immunoprecipitation of KDER-YFP-HA from the nucleus indicated that the kinase domain of
942 ER interacts with SWI3B. F, ER, ERL1, and ERL2 kinase domains interact with SWI3B in the
943 nucleus. Bimolecular Fluorescence Complementation assay (BiFC) in epidermis of tobacco
944 leaves. Scale bar = 10 μ m.

945
946 **Figure 4.** ER and *SWI3B* interact genetically and affect both GA biosynthesis and response
947 pathways (See also Figures S11 and S12). A, The *er-105/swi3b-3* double mutant exhibits more
948 retarded growth than the *er-105* and *swi3b-3* (three-weeks old plants). Graphical alignment of
949 corresponding leaves. Scale bar= 1 cm. B, The hypersensitivity of 1-week-old *swi3b-3* hypocotyl
950 to GA treatment is abolished by introducing *er-105*. C, Roots of all tested 1-week-old genotypes
951 similarly respond to PAC treatment (error bars-SD, *P < 0.01,** P < 0.001, ***P<0.0001,
952 Student's *t*-test). D, Hypocotyls of all tested 1-week-old genotypes similarly respond to PAC
953 treatment, right panel- hypocotyl length comparison for PAC treated plants only (error bars-SD,
954 *P < 0.01,** P < 0.001, ***P<0.0001 Student's *t*-test). E, *swi3b-3* weak, point mutant line and
955 *er-105/swi3b-3* exhibit elevated *SWI3B* transcript level, the *SWI3B* expression is elevated after
956 supplementation with bioactive GA₄₊₇ in all genotypes except *swi3b-3* (error bars-SD, P < 0.05,
957 Student's *t*-test). F, The examination of *GID1* genes indicated that almost all examined lines
958 responded to GA treatment, but the *swi3b-3* line was insensitive for GA-induced transcriptional
959 changes (error bars-SD, P < 0.05, Student's *t*-test). G, The examination of GA biosynthesis genes
960 indicated that almost all examined lines responded to GA treatment, but the *swi3b-3* line was

961 insensitive for GA-induced transcriptional changes except *GA20ox2* expression (error bars-SD, P
962 < 0.05, Student's *t*-test).

963
964 **Figure 5.** ERF proteins are responsible for the phosphorylation of SWI3B protein, while DELLA
965 proteins control SWI3B protein abundance (See also Figures S13, S14, and S15). A, Coomassie
966 staining of MBP-His6-KDER and His6-SWI3B proteins purified from bacteria. B, Western blot
967 with anti-Thiophosphate ester antibody (ab92570; Abcam) showing *in vitro* SWI3B
968 phosphorylation by KDER. C, 2D Western blot assay with anti SWI3B antibody indicating *in*
969 *vivo* phosphorylation alteration of SWI3B protein in *er/erl1/erl2* mutant. D, SWI3B and RGA
970 and RGL1 proteins in the nuclei of living cells. Bimolecular Fluorescence Complementation
971 assay (BiFC) in epidermis of tobacco leaves. Scale bar = 10µm. E, The amounts of SWI3B and
972 RGA proteins in plants are oppositely regulated by PAC treatment. F, The disappearance of
973 SWI3B protein is PAC-dose dependent. G, The PAC-dependent degradation of SWI3B is
974 abolished by the MG132 treatment, a known proteasome inhibitor. H, The *gal-3* mutant
975 constitutively accumulating DELLA proteins exhibits the decreased level of SWI3B, which is
976 restored to WT levels upon GA treatment. I, The triple DELLA mutant exhibits a WT-like level
977 of SWI3B protein, and the PAC treatment does not influence SWI3B level in this background. J,
978 Schematic model highlighting ERF and DELLA impact on the SWI3B protein.

979
980 **Figure 6.** ERF proteins enter the nucleus where ERECTA protein binds the *GID1* promoters
981 similarly to the SWI3B subunit of SWI/SNF CRC (See also Figures S16 and S17). A, ERECTA
982 protein binds to promoter regions of the *GID1a* gene in a region targeted by the SWI/SNF
983 complex in three-week-old plants. (error bars refer to SD, P < 0.05, Student's *t*-test, three
984 biological and technical replicates were performed). B, ERECTA and SWI3B core subunit of
985 SWI/SNF CRCs target promoter regions of *GID1b* gene in three-weeks old plants (error bars
986 refer to SD, P < 0.05, Student's *t*-test, three biological and technical replicates were performed).
987 C, SWI3B binds to the promoter region of the *GID1c* gene in two different regions. One of them
988 is targeted by ERECTA protein in three-week-old plants. D, ERECTA protein binds to promoter
989 regions of the *GID1a* gene in a region targeted by the SWI/SNF complex in five-week-old plants.
990 (error bars refer to SD, P < 0.05, Student's *t*-test, three biological and technical replicates were
991 performed). E, ERECTA targets promoter region of *GID1b* gene in five-week-old plants (error

992 bars refer to SD, $P < 0.05$, Student's *t*-test, three biological and technical replicates were
993 performed). F, ERECTA binds to the promoter region of the *GID1c* gene in three-week-old
994 plants. The bottom panel in D-F: the binding of native SWI3B protein to its target sites in *GID1a-*
995 *c* promoter regions is abolished in 5-week-old *er/er11/er12* triple mutant plants. G, A model
996 describing the non-canonical nuclear function of ERF proteins in the GA signaling pathway.
997

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999

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