

1 **Transcriptomic and epigenetic responses shed light on soybean resistance to *Phytophthora***  
2 ***sansomeana***

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17 **Running Title:** Genetic and epigenetic responses of soybean to *Phytophthora sansomeana*

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20 **Key words:** *Phytophthora sansomeana*, soybean, transcriptomics, long non-coding RNAs, DNA  
21 methylation

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25 **Abstract**

26 Phytophthora root rot caused by oomycete pathogens in the *Phytophthora* genus poses a  
27 significant threat to soybean productivity. While resistance mechanisms against *Phytophthora*  
28 *sojae* have been extensively studied in soybean, the molecular basis underlying immune  
29 responses to *Phytophthora* *sansomeana* remains unclear. We investigated transcriptomic and  
30 epigenetic responses of two resistant (Colfax and NE2701) and two susceptible (Williams 82 and  
31 Senaki) soybean lines at four time points (2, 4, 8, and 16 hours post inoculation, hpi) after *P.*  
32 *sansomeana* inoculation. Comparative transcriptomic analyses revealed a greater number of  
33 differentially expressed genes (DEGs) upon pathogen inoculation in resistant lines, particularly  
34 at 8 and 16 hpi, predominantly associated with ethylene and reactive oxygen species-mediated  
35 defense pathways. Moreover, DE transposons were predominantly up-regulated after inoculation  
36 and enriched near genes in Colfax. A long non-coding RNA (lncRNA) within the mapped region  
37 of the resistance gene exhibited exclusive up-regulation in the resistant lines after inoculation,  
38 potentially regulating two flanking *LURP-one-related* genes. Furthermore, DNA methylation  
39 analysis revealed increased CHH methylation levels in lncRNAs after inoculation, with delayed  
40 responses in Colfax compared to Williams 82. Overall, our results provide comprehensive  
41 insights into soybean responses to *P. sansomeana*, highlighting potential roles of lncRNAs and  
42 epigenetic regulation in plant defense.

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48 **Introduction**

49 Plants are constantly challenged by diverse pathogens, including bacteria, fungi, viruses, and  
50 oomycetes. To counter these biotic stresses, plants have evolved diverse defense mechanisms (J.  
51 D. G. Jones & J. L. Dangl, 2006). Cell surface receptor proteins called pattern recognition  
52 receptors in plants recognize pathogen-associated molecular patterns (PAMPs), eliciting PAMP-  
53 triggered immunity (PTI) (Boller & He, 2009). Additionally, plants have intracellular nucleotide  
54 binding site-leucine rich repeat receptors (NBS-LRRs or NLRs) that can recognize pathogen  
55 effectors, leading to effector-trigger immunity (ETI) (Dodds & Rathjen, 2010; Ngou et al., 2022).  
56 Both PTI and ETI could facilitate rapid cellular responses, such as calcium influx,  
57 reprogramming of the expression of defense-responsive genes (Amorim et al., 2017;  
58 Thirugnanasambantham et al., 2015), and activation of mitogen-activated protein kinases  
59 (MAPKs) that coordinate immune responses and modulate other defense-related genes (Meng &  
60 Zhang, 2013). Plant defense against pathogens also involves the production of defense  
61 phytohormones, including ethylene (ET), salicylic acid (SA), jasmonic acid (JA),  
62 brassinosteroids (BR), and auxin. These hormones collaboratively work to regulate immune  
63 responses in plants by serving as the primary molecules in the induced defense signaling network  
64 (Bürger & Chory, 2019; Pieterse et al., 2009). Additionally, in response to pathogenic attacks,  
65 plants produce specific compounds such as phytoalexins (Ahuja et al., 2012; Hammerschmidt,  
66 1999) and reactive oxygen species (ROS) (Mohammadi et al., 2021; Tyagi et al., 2022).  
67 Transcriptional changes for the coordination of these multiple pathways collectively contribute  
68 to the complex defense system that enables plants to effectively counteract pathogen challenges.  
69 In addition to transcriptional responses in protein encoding genes, plants react to  
70 environmental stresses by rapidly altering the transcription and activity of other genetic elements,

71 such as long non-coding RNAs (lncRNAs) and transposable elements (TEs) (Guo et al., 2021;  
72 Hou et al., 2019; Klein & Anderson, 2022). lncRNAs are a class of RNAs longer than 200  
73 nucleotides that lack protein-coding potential (Chekanova, 2015; Mattick et al., 2023). Despite  
74 their inability to code for proteins, lncRNAs play crucial roles in regulating gene expression both  
75 in *cis* and in *trans* through mechanisms such as transcriptional or post-transcriptional regulation,  
76 chromatin modification, RNA processing and stability, and scaffold interactions (Sharma et al.,  
77 2022; Zhang et al., 2020). While high-throughput RNA sequencing technologies have enabled  
78 the identification of a significant number of lncRNAs in plants associated with defense responses  
79 against fungal, viral, and bacterial infections (Di et al., 2014; Joshi et al., 2016; Seo et al., 2017;  
80 Sun et al., 2020; Wang et al., 2015; Wang et al., 2017; Xin et al., 2011; Yu et al., 2020; Zhang et  
81 al., 2013; Zhu et al., 2014), functional characterization of these lncRNAs remains limited.  
82 Particularly noteworthy is the scarcity of studies that have explored the specific roles of  
83 lncRNAs in conferring resistance to pathogenic oomycetes. A primary focus of lncRNAs to  
84 oomycetes has been on *Phytophthora infestans*, the causal pathogen responsible for late blight in  
85 tomato (Cui et al., 2020; Cui et al., 2017; Su et al., 2023). These identified lncRNAs modulate a  
86 range of genes that control the activation of multiple phytohormones and the accumulation of  
87 ROS, thereby enhancing the plants' resistance to the pathogen.

88 DNA methylation is a fundamental epigenetic process that regulates transcription activity,  
89 transposon mobility, and chromatin stability in response to both abiotic and biotic stresses (Arora  
90 et al., 2022; Dowen et al., 2012; Hewezi et al., 2018). In plants, DNA methylation commonly  
91 occurs in three cytosine sequence contexts CG, CHG, and CHH (where H represents A, T, or C)  
92 (Law & Jacobsen, 2010; Matzke & Mosher, 2014). The alternation of methylation levels in  
93 genes and TEs plays a pivotal role in orchestrating the dynamic rewiring of plant genomes

94 during stress responses to pathogen virulence (Cambiagno et al., 2018; Liu & Zhao, 2023;  
95 Zervudacki et al., 2018). For instance, a study investigating DNA methylation variants associated  
96 with soybean cyst nematode parasitism reveals distinct methylation patterns across three cytosine  
97 contexts (Rambani et al., 2020). Furthermore, methylation changes have been noted in lncRNAs  
98 of several species (Ding et al., 2012; He et al., 2014; Li et al., 2021; Zhao et al., 2016). A notable  
99 example is the rice lncRNA LDMAR, which experiences increased methylation in its promoter  
100 region due to a point mutation, resulting in abnormal pollen development (Ding et al., 2012).  
101 Nevertheless, the specific mechanisms underlying the methylation changes of lncRNAs in the  
102 context of plant immunity remains largely unexplored.

103 Phytophthora root rot (PRR) is one of the most destructive diseases in soybean (*Glycine*  
104 *max*), causing substantial soybean yield losses worldwide (Allen et al., 2017; Kaufmann &  
105 Gerdemann, 1958; Sahoo et al., 2021; Sandhu et al., 2005). Historically, this disease has been  
106 attributed to the soil-borne hemibiotrophic oomycete pathogen, *Phytophthora sojae*, which  
107 primarily infects soybean (Dorrance et al., 2008; Malwick & Grunden, 2004; TYLER, 2007). The  
108 resistance to *P. sojae* (*Rps*) genes have been effective in controlling PRR, with over 40 *Rps*  
109 genes/alleles identified as crucial regulators modulating diverse pathways against *P. sojae*  
110 (Anderson & Buzzell, 1992; Burnham et al., 2003; Lin et al., 2014; Polzin et al., 1994; Sahoo et  
111 al., 2021; Sandhu et al., 2005; Zhou et al., 2022). Many of these *Rps* genes belong to the NBS-  
112 LRR family, which can directly or indirectly recognize the corresponding effectors from a  
113 variety of pathogens (Ngou et al., 2022; Wu et al., 2018). In 2009, *Phytophthora* *sansomeana*  
114 was differentiated from the *Phytophthora megasperma* complex as a causal agent of root rot  
115 across a broader range of hosts, including soybean, carrot, pea, white clover, gerbera, and maize  
116 (Detranaltes et al., 2022; Hansen et al., 2009; Lin et al., 2021; Rojas et al., 2017; Zelaya-Molina

117 et al., 2010). The prevalence of *P. sansomeana* in soybean, particularly in North America and  
118 Northeast Asia, has led to significant agricultural losses (Alejandro Rojas et al., 2017; Lin et al.,  
119 2021; Malwick & Grunden, 2004; Rahman et al., 2015; Tang et al., 2010). In a recent study on  
120 the pathogenicity of oomycete species, it was determined that *P. sansomeana* has a more virulent  
121 impact on root reduction in soybean seedlings compared to *P. sojae* (Alejandro Rojas et al.,  
122 2017). However, unlike *P. sojae*, no resistant genes have been identified in soybean for *P.*  
123 *sansomeana*, leaving the molecular and physiological mechanisms underlying defense or stress  
124 responses to this pathogen poorly understood. Therefore, further exploration into the genetic  
125 control of immune responses to *P. sansomeana* becomes imperative to understand its  
126 pathogenicity and interactions with soybean for fortifying crop defenses.

127 Here, we conducted comprehensive analyses of transcriptome landscapes, including an in-  
128 depth investigation of differential transcriptions of genes, TEs, and lncRNAs, along with DNA  
129 methylation patterns in response to *P. sansomeana* to unravel the genetic and epigenetic basis of  
130 defense mechanisms in the resistant soybean lines by comparison with the susceptible lines. Our  
131 comparative analysis revealed contrasting transcriptomic and epigenetic responses between the  
132 resistant and susceptible lines. Specially, we observed the specific expression of a number of  
133 differentially expressed genes (DEGs) linked to ET-mediated defense responses, along with ROS  
134 generation with increased hydrogen peroxide levels within the resistant lines. Furthermore, the  
135 DE TEs were prominently up-regulated after inoculation, particularly in proximity to genes in  
136 Colfax. We also identified a DE lncRNA within the mapped region of the resistance gene in  
137 Colfax that exhibited significant increase in expression exclusively within the resistant lines  
138 following pathogen inoculation. This lncRNA holds the potential to regulate adjacent *LURP-one-*  
139 *related (LOR)* genes, known for their involvement in defense-related mechanisms against

140 pathogenic oomycetes in *Arabidopsis* (Baig, 2018; Knoth & Eulgem, 2008). Additionally, our  
141 DNA methylation analysis revealed increased CHH methylation levels in lncRNAs after  
142 inoculation, at a later time point in the resistant lines compared to an earlier time point in the  
143 susceptible lines, suggesting distinctive epigenetic responses of these different soybean lines.  
144 Together, our study provides valuable insights into the molecular and physiological mechanisms  
145 underlying the defense responses of soybean to the newly recognized pathogen *P. sansomeana*.

146

## 147 **Materials and Methods**

### 148 **Plant growth, inoculation, and sample collection**

149 Four soybean lines, Colfax, NE2701, Senaki, and Williams 82 were grown in the greenhouse at  
150 Michigan State University. For each of the four biological replicates, we divided the seedlings  
151 from each line into two groups: one group was inoculated with *P. sansomeana*, while the other  
152 group was mock-inoculated without the pathogen. The cultivation of *P. sansomeana* followed  
153 established protocols typically used for studying *P. sojae* (Dorrance et al., 2008). In each  
154 replicate, ten seedlings from each line were challenged with the *P. sansomeana* isolate MPSI7-  
155 22 using the standard hypocotyl inoculation method (Lin et al., 2021). Tissues were collected  
156 from each line and condition at four designated time points: 2 hours post inoculation (hpi), 4 hpi,  
157 8 hpi, and 16 hpi. Specifically, stem tissues were collected from 7-8 seedlings in each replicate  
158 by excising a 2-3 cm segment from the wounded site. These collected tissues were rapidly frozen  
159 using liquid nitrogen and subsequently stored at -80°C. Additionally, the remaining seedlings  
160 were retained to closely monitor the progression of symptoms and assess survival rates for a  
161 period up to one week post inoculation.

162

163 **RNA sequencing and data analysis**

164 Total RNA was extracted from approximately 100 mg of frozen stem tissues for each sample  
165 using the Qiagen RNeasy Plant Mini-Kit (Qiagen, Valencia, CA) according to the  
166 manufacturer's instructions. Subsequently, library preparation and RNA sequencing were  
167 conducted at Novogene (Sacramento, CA) using the Illumina HiSeq 4000 and NovaSeq 6000  
168 platform. The sequencing yielded a total of 30 to 47 million 150 bp read pairs per sample, which  
169 were used for further transcriptome analyses.

170 The quality of the sequenced reads was assessed using FastQC v0.11.7  
171 (<http://www.bioinformatics.babraham.ac.uk/projects/fastqc/>). The raw reads from the RNA-seq  
172 data were trimmed using Trimmomatic v0.36 to remove adapters and low-quality reads (Bolger  
173 et al., 2014). The trimmed reads were then aligned to the soybean reference genome, Williams  
174 82 genome assembly v4.0 (Wm82.a4), using HISAT v2.2.1 (Kim et al., 2015; Valliyodan et al.,  
175 2019). Additionally, uniquely mapped reads were retained, and reads with multiple matches were  
176 maintained by opting the shortest alignments or selecting a single random alignment for each  
177 instance of palindromic multiple mapping using selected scripts from COMEX v2.1 (Lee et al.,  
178 2021; Pietzenuk et al., 2016). The number of reads per gene for each sample was quantified  
179 using HTSeq v0.11.2 (Putri et al., 2022).

180 Differentially expressed genes (DEGs) between the control and inoculated samples were  
181 identified, and principal component analysis (PCA) of count data was conducted to assess data  
182 quality and sample distance using the R package *DESeq2* v1.12.3 (Love et al., 2014). Significant  
183 DEGs were identified using adjusted *P*-values from Wald-Test with FDR correction (Benjamini  
184 & Hochberg, 1995). Normalized counts obtained from *DESeq2* were used to compare transcript  
185 levels for genes across samples. Furthermore, co-expressed gene clusters were identified for each

186 line using Clust v1.18.1 (Abu-Jamous & Kelly, 2018). One or two clusters per line, displaying  
187 similar patterns such as clusters up-regulated after inoculation, were selected to identify co-  
188 expressed genes specific to the resistant or susceptible lines.

189 To determine biological categories of DEGs or genes selected in cluster analysis, gene  
190 ontology (GO) enrichment analysis was conducted using g:GOst functional profiling in  
191 g:Profiler (Raudvere et al., 2019). This analysis employed a hypergeometric test to identify  
192 significantly overrepresented GO terms (adjusted  $P < 0.01$ ). In addition, heatmaps depicting  
193 relative expression levels per gene for different pathways related to the response to *P.*  
194 *sansomeana* were generated. We manually curated a custom annotated list of genes potentially  
195 involved in response pathways, including signaling or synthesis of ET, JA, SA, BR, and MAPK.  
196 Normalized counts of these genes were used for measuring transcript levels, and gene clustering  
197 based on Euclidean distance was performed using the R package *pheatmap*.

198

### 199 **Analysis of transposable elements**

200 To explore the transcriptional activities of TEs, the mapped and corrected reads obtained from  
201 RNA-seq were counted using HTSeq v0.11.2 based on the TE annotation of the Wm82.a4  
202 genome (Valliyodan et al., 2019). We focused on six superfamilies of DNA transposons and four  
203 superfamilies of retrotransposons (see results). To compare the overall transcript levels of each  
204 TE superfamily across different lines and conditions, we used normalized counts divided by the  
205 length of each TE (NCPK; normalized counts per kilobase of TE length) as described in (Lee et  
206 al., 2021). Additionally, we counted the numbers and proportions of transcribed TEs (NCPK >  
207 0.5) per superfamily per sample. Moreover, differentially transcribed TEs were identified using  
208 the R package *DESeq2* v1.12.3, the same as the method employed in the DEG analysis.

209 Subsequently, we calculated the proportions of TE superfamilies among the DE TEs and  
210 compared them to the genome-wide levels. To determine the genomic locations of DE TEs, we  
211 examined whether they were located within or in close proximity to genes, as well as in  
212 pericentromeric regions and chromosomal arms based on annotations for TEs and genes using  
213 the *intersect* and *window* functions in bedtools v2.30.0 (Quinlan & Hall, 2010).

214

## 215 **LncRNA identification and analysis**

216 To identify lncRNAs, the transcriptomic data was *de novo* assembled following the Cufflinks  
217 v2.2.1.1 workflow (Trapnell et al., 2010). To achieve this, trimmed reads from the RNA-seq data  
218 were mapped to the soybean reference genome, Wm82.a4, using HISAT 2.2.1, with the inclusion  
219 of the Cufflinks option (--dta-cufflinks). The resultant mapped reads were used for transcript  
220 assembly for each sample through Cufflinks. These assembled transcripts were then merged to  
221 generate the final transcriptome assembly using the Cuffmerge function.

222 LncRNAs (> 200 bp) were identified from the assembled and merged transcripts using  
223 Evolinc-I v1.7.5 (Nelson et al., 2017). This identification process categorized lncRNAs into three  
224 types: long intergenic non-coding RNAs (lncRNAs), sense-overlapping lncRNAs (SOTs), and  
225 antisense (AOTs). To differentiate TE-containing lncRNAs from non-TE lncRNAs, we  
226 incorporated TE sequence data obtained using gff3toolkit v2.0.3 (Chen et al., 2019), based on the  
227 manually refined annotation of the repeat-masked assembly for the Wm82.a4 genome with the  
228 sequence similarity bit score > 200 and E-value < 1E-20.

229 Furthermore, transcription patterns of four transcript types, including genes, non-TE  
230 lncRNAs, TE-containing lncRNAs, and TEs, were explored using NCPK as the measure of  
231 transcript levels. First, the proportion of transcribed transcripts (NCPK > 0.5) across different

232 conditions and lines using all samples was calculated for each transcript type. Subsequently,  
233 transcript levels of these transcripts were compared using  $\log(NCPK + 1)$ .

234 For differentially transcribed lincRNAs upon inoculation, a method similar to the DEG  
235 analysis was pursued. DE lincRNAs were separately analyzed for non-TE lincRNAs and TE-  
236 containing lincRNAs, using the R package *DESeq2* v1.12.3 (as detailed above). Extracted  
237 normalized counts were used to examine the transcript levels of DE lincRNAs across different  
238 lines and conditions. We further investigated target genes by analyzing DE lincRNAs that were  
239 exclusively identified in either resistant or susceptible lines. To uncover potential *trans*-target  
240 genes, correlation analysis was employed between the transcript levels of DEGs and DE  
241 lincRNAs. DEGs with correlation coefficients (*R*) greater than 0.85 or less than -0.85, alongside  
242 a *P*-value < 0.001, were considered potential *trans*-targets. Additionally, for potential *cis*-targets,  
243 genes or TEs located within 5 kb upstream and downstream of DE lincRNAs were extracted  
244 using bedtools v2.30.0.

245

#### 246 **qRT-PCR validation**

247 Total RNA for qRT-PCR was extracted from a separate biological replicate, distinct from the  
248 samples used for transcriptome sequencing, using the RNeasy Plant Mini with DNase I treatment  
249 according to the manufacturer's instructions (Qiagen, Valencia, CA). One  $\mu$ g of RNA was used  
250 for a 20- $\mu$ L first-strand cDNA synthesis reaction using High Capacity cDNA Reverse  
251 Transcription Kit (Thermo Fisher Scientific, Waltham, MA) following the manufacturer's  
252 guidelines. For each 10  $\mu$ L of qPCR reaction, 2  $\mu$ L of a 1/10 dilution of the synthesized cDNA  
253 was mixed with PowerTrack SYBR Green Master Mix (Thermo Fisher Scientific, Waltham, MA)  
254 and primer pairs. The thermal cycling involved initial denaturation at 95°C for 2 min, 40 cycles

255 at 95°C for 15 sec and 60°C for 1 min, followed by final elongation at 60°C for 10 min, with a  
256 dissociation step. Quantification of relative transcript levels (RTL) involved normalizing the  
257 transcript levels of genes of interest to the respective transcript level of the housekeeping gene  
258 *Cons4* that had lowest variation in transcript levels among housekeeping genes across different  
259 samples in our RNA sequencing dataset.

260

## 261 **Determination of hydrogen peroxide concentration**

262 Methods for the measurement of hydrogen peroxide (H<sub>2</sub>O<sub>2</sub>) concentration followed procedures  
263 previously described (Alexieva et al., 2001). In brief, tissue samples were ground in liquid  
264 nitrogen and mixed with 0.5 ml of 0.1% trichloroacetic acid. Following centrifugation, 0.25 ml  
265 of 0.1 M phosphate buffer (pH 7.0) and 1 ml of 1 M KI were added to 0.25 ml of the resulting  
266 supernatant. The absorbance was then measured at 390 nm using the spectrophotometer  
267 GENESYS 20 (Thermo Fisher Scientific, Waltham, MA) after the reaction rested for 1h in  
268 darkness. The quantification of H<sub>2</sub>O<sub>2</sub> was determined by a standard curve prepared using known  
269 concentrations of H<sub>2</sub>O<sub>2</sub>.

270

## 271 **Analysis of whole genome bisulfite sequencing data**

272 DNA was isolated from the same tissues of two of the four soybean lines, Colfax and Willaims  
273 82, at two time points (4 and 16 hpi) using the modified CTAB method. Library construction and  
274 subsequent sequencing were performed at Novogene. The raw reads were quality controlled by  
275 FastQC and trimmed using Trimmomatic (Bolger et al., 2014). The resulting clean reads were  
276 then mapped to the soybean reference genome v4.0 (Wm82.a4) using Bismark with the  
277 following parameters (-I 50, -N 1) (Krueger & Andrews, 2011; Valliyodan et al., 2019). To

278 eliminate PCR duplicates, the deduplication package under Bismark was utilized. Additional  
279 packages under Bismark, including Bismark methylation extractor, bismark2bedGraph and  
280 coverage2cytosine, were employed to extract methylated cytosines and count methylated and  
281 unmethylated reads following our previous research (Yin et al., 2022; Zhao et al., 2021).

282 Methylated levels for each cytosine contexts (CG, CHG, and CHH) were calculated by  
283 dividing the number of methylated reads by the total number of methylated and unmethylated  
284 reads (Schultz et al., 2012). The average methylation level for each cytosine context per  
285 transcript of non-TE lincRNAs was calculated for each line and condition using the *map* function  
286 in bedtools v2.30.0. Additionally, we examined the distribution of methylation proportions  
287 across three genomic regions: transcript bodies, 2 kb upstream, and 2 kb downstream of the  
288 lincRNAs. Mean methylation levels were calculated in 40 windows per region to assess the  
289 overall methylation patterns across lincRNA bodies and their flanking regions.

290

## 291 **Results**

### 292 **Two soybean lines, Colfax and NE2701, are resistant to *P. sansomeana***

293 We previously screened approximately 500 soybean lines to identify resistance to *P. sansomeana*  
294 and discovered two lines, Colfax and NE2701, which exhibited resistance to the pathogen in both  
295 field and greenhouse conditions. Alongside these two resistant lines, we included two susceptible  
296 lines, Senaki and Williams 82, the latter of which serves as the reference genome of soybean, for  
297 transcriptomic analyses. Four biological replicates from each line were planted under greenhouse  
298 conditions. The seedlings from each line were then divided into two groups: one group was  
299 inoculated with *P. sansomeana*, while the other group was mock-inoculated without the  
300 pathogen. We observed stem rot in the majority of Senaki and Williams 82 plants three days after

301 pathogen inoculation, while a few Colfax and NE2701 plants exhibited the symptom (Figure S1).  
302 Additionally, we evaluated the resistance of the four soybean lines by measuring their survival  
303 rates to *P. sansomeana* seven days post mock (control) and pathogen inoculation (inoculated) to  
304 validate resistance or susceptibility of the four selected soybean lines to the pathogen. The  
305 survival rates for all four lines were consistently 100% after mock inoculation (Figure 1). The  
306 average survival rates for both resistant lines, Colfax and NE2701, showed no significant  
307 difference between mock and pathogen inoculation. However, the two susceptible lines, Senaki  
308 and Williams 82, displayed significant susceptibility to *P. sansomeana* after pathogen  
309 inoculation, when compared to mock inoculation (Figure 1; ANOVA, followed by Tukey's HSD  
310 test;  $P < 0.05$ ). The average survival rates of Senaki and Williams 82 upon pathogen inoculation  
311 were approximately 70% and 80% lower than those after mock inoculation, respectively. These  
312 results demonstrate that Colfax and NE2701 exhibit resistance to *P. sansomeana*, while Senaki  
313 and Williams 82 are susceptible to the pathogen.

314

315 **Transcriptomic responses to pathogen inoculation reveal contrasting patterns in resistant  
316 and susceptible soybean lines**

317 To dissect the molecular responses to the pathogen in the resistant and susceptible lines, we  
318 conducted a comprehensive analysis of transcriptomic changes following inoculation. A total of  
319 64 soybean samples, including four different lines, four time points (2, 4, 8, and 16 hours post  
320 inoculation, hpi), and two treatments (pathogen vs. mock), each with two biological replicates,  
321 were used for transcriptome analyses. The principal component analysis (PCA) of the  
322 transcriptome revealed that the primary source of variation (PC1; explained 54% of total

323 variance) was attributed to four different time points, while the second component, PC2 (14%),  
324 was associated with inoculation types (Figure S2).

325 Next, we identified differentially expressed genes (DEGs) in response to *P. sansomeana*  
326 inoculation. Upon pathogen inoculation, the transcriptomic differences in both the resistant lines  
327 comprised a considerably greater number of DEGs than those in the susceptible lines (Figure 2a;  
328 Dataset S1). In the resistant line, Colfax, a total of 5,058 genes were significantly up-regulated,  
329 and 3,120 genes were down-regulated across the four time points in response to the pathogen.  
330 Similarly, in the other resistant line, NE2701, 2,611 genes were up-regulated, and 1,364 genes  
331 were down-regulated upon pathogen inoculation. In contrast, the susceptible lines, Senaki and  
332 Williams 82, exhibited a relatively smaller number of DEGs following inoculation. Senaki  
333 showed 1069 up-regulated and 158 down-regulated genes, while Williams 82 had 603 up-  
334 regulated and 256 down-regulated genes (Figure 2a).

335 To specifically identify DEGs unique to either the resistant or susceptible lines, we  
336 performed an intersection analysis of these DEGs between different soybean lines (Figure 2b). In  
337 the early stages at 2 and 4 hpi, only a limited number of DEGs were common in the two resistant  
338 lines. However, at 8 and 16 hpi, a substantial overlap was observed, with several hundred up-  
339 regulated DEGs being shared between Colfax and NE2701, while only a few dozen down-  
340 regulated DEGs were common to these two lines. Among the DEGs identified in the resistant  
341 lines, we identified *Glyma.09G210600* (Dataset S1), a homolog of *resistance to pseudomonas*  
342 *syringae 3 (RPS3)* in Arabidopsis, which encodes a NBS-LRR-type protein, known for its role as  
343 a R protein in the defense response (Dangl & Jones, 2001; Lin et al., 2014; Sandhu et al., 2005;  
344 van der Hoorn & Kamoun, 2008). Interestingly, it showed significant up-regulation in response

345 to the pathogen inoculation at 8 or 16 hpi exclusively in both resistant soybean lines (Figure S3;  
346 Dataset S1).

347 To elucidate the function of these DEGs, we performed gene ontology (GO) enrichment  
348 analysis. Enriched GO terms were only identified at 8 and 16 hpi in the resistant lines, and at 4  
349 hpi in the susceptible lines. At 8 hpi, the 204 up-regulated DEGs uniquely shared by the two  
350 resistant lines were primarily associated with ET-responsive functions, including the ethylene-  
351 activated signaling pathway (GO:0009873), cellular response to ethylene stimulus (GO:0071369),  
352 and response to ethylene (GO:0009723) (Figure 2c). In the molecular function category, we  
353 identified 24 genes exhibiting DNA-binding transcription factor activity (GO:0003700) that were  
354 exclusively up-regulated in the resistant lines at 8 hpi. These include multiple ethylene response  
355 factors (*ERFs*) and various other transcription factors. Moreover, in the Kyoto encyclopedia of  
356 genes and genomes (KEGG), the MAPK signaling pathway (KEGG:04016) was significantly  
357 overrepresented among the up-regulated genes at 8 hpi in the resistant lines.

358 At 16 hpi, the 308 commonly up-regulated DEGs in the resistant lines demonstrated the  
359 most significant enrichment in GO terms related to defense response (GO:0006952), response to  
360 stress (GO:0006950) or stimulus (GO:0050896), along with several other terms associated with  
361 stress or defense responses, such as hydrogen peroxide catabolic process (GO:0042744) and  
362 ROS metabolic process (GO:0072593) (Figure 2d). To illustrate the accumulation of hydrogen  
363 peroxide ( $H_2O_2$ ) accumulated in the resistant lines, we quantified the concentration of  $H_2O_2$  in  
364 the four lines at 16 hpi. We observed an increase in the concentration of  $H_2O_2$  in the two resistant  
365 lines upon *P. salsomeana* inoculation at 16 hpi (Figure 2e). In contrast, the two susceptible lines  
366 exhibited a lower concentration of  $H_2O_2$  under pathogen inoculation compared to mock  
367 inoculation.

368 In contrast, the susceptible lines had fewer common DEGs across all time points. Notably  
369 overrepresented GO terms in the susceptible lines were only detected at 4 hpi (Figure 2f). The  
370 majority of the significantly enriched GO classes for the down-regulated DEGs were  
371 predominantly associated with photosynthesis-related classes, including photosystem I  
372 (GO:0009522) and photosystem II (GO:0009523), and photosynthesis light harvesting  
373 (GO:0009765).

374 In addition to the differential transcriptomic responses between the resistant and susceptible  
375 lines, we also observed distinctive defense responses within the two resistant lines. In NE2701,  
376 the highest number of genes exhibited differential regulation as early as 2 hpi, in contrast to  
377 Colfax, where the most significant differences in DEGs emerged at a relatively later time point,  
378 16 hpi (Figure 2a,b). Enriched GO terms in the biological process category for DEGs exclusively  
379 at 2 hpi in NE2701 included regulation of the jasmonic acid mediated signaling pathway  
380 (GO:2000022) and plant-type cell wall organization (GO:0009664) (Table S1). On the other  
381 hand, in Colfax, up-regulated genes were most significantly enriched for the phosphate-  
382 containing compound metabolic process (GO:0006796), while down-regulated genes were  
383 notably associated with photosynthesis (GO:0015979) at 16 hpi. These findings suggest that in  
384 addition to common defense strategies shared by these two resistant lines, genetic variations  
385 between these two lines also contribute to the distinct transcriptomic changes in response to the  
386 pathogen *P. sansemeana*.

387

388 **Ethylene-responsive genes are mainly co-expressed in the two resistant lines in response to**  
389 **the pathogen inoculation**

390 To identify co-expressed genes in response to the pathogen across the four lines, we extracted  
391 gene clusters demonstrating similar patterns of gene regulation under different conditions for  
392 each line. From these grouped clusters in each line (Figure S4), we selected the clusters likely to  
393 contain co-expressed genes that were either up-regulated or down-regulated upon pathogen  
394 inoculation (Figure 3; Dataset S2). However, no clusters from Senaki and Williams 82 displayed  
395 down-regulation in response to the pathogen inoculation. Therefore, we focused only on the  
396 clusters associated with up-regulation (Figure S4). We identified two clusters (503 and 1606  
397 genes; Figure 3a) in Colfax, along with one cluster for each of the other soybean lines, NE2701  
398 (1091 genes; Figure 3b), Senaki (1891 genes; Figure 3c), and Williams 82 (981 genes; Figure  
399 3d), all exhibiting up-regulation upon pathogen inoculation.

400 In these gene clusters, numerous GO classes associated with diverse biological processes  
401 and molecular functions were significantly enriched (Dataset S3). To pinpoint co-expressed  
402 genes specific to the resistant lines, we intersected the genes from the selected up-regulated  
403 clusters and extracted 370 genes exclusively present in the resistant lines (Figure 3e). Among  
404 these genes, ET-responsive genes (response to ethylene, GO:0009723; cellular response to  
405 ethylene stimulus, GO:0071369; ethylene-activated signaling pathway, GO:0009873) were  
406 overrepresented as the main globally co-expressed genes up-regulated in response to the  
407 pathogen inoculation in the resistant lines (Figure 3f). These genes included several ethylene  
408 response factors (ERFs) like *ERF1*, *ERF15*, and *ERF98*, known for their regulatory role in ET-  
409 responsive genes (Gao et al., 2020; Thirugnanasambantham et al., 2015). Additionally, the  
410 ethylene-forming enzyme *1-aminocyclopropane-1-carboxylate oxidase 4* (*ACO4*) was part of  
411 this ET-responsive gene network (Table S2).

412

413 **The majority of the differentially transcribed TEs are up-regulated in response to *P.***  
414 ***sansomeana* in all lines, and those in Colfax are more enriched near genes**

415 As TEs are often subject to environmental changes (Casacuberta & González, 2013), we  
416 compared transcriptional patterns of TEs in the four soybean lines in response to *P. sansomeana*.  
417 Based on the annotation of the soybean reference genome (Wm82.a4), we examined the  
418 expression of TEs across ten superfamilies, including four retrotransposons (class I) and six  
419 DNA transposons (class II) (Figure 4; Figure S5). Out of the 324,333 TEs, only an average of 3%  
420 exhibited transcription, in either mock-inoculated or pathogen-inoculated plants. Notably,  
421 although long interspersed nuclear elements (LINEs) were not the most abundant TE superfamily  
422 in the soybean genome, they constituted the largest proportion of transcribed TEs at 15%  
423 compared to other TE superfamilies (Figure S5). In Colfax, more TEs in most superfamilies were  
424 transcribed upon the pathogen inoculation compared to the mock inoculation consistently at 2  
425 hpi, and predominantly at 16 hpi, particularly in DNA transposons. In Senaki, a similar overall  
426 increase in transcribed TEs upon inoculation was observed at 2 hpi, mirroring the trend noted in  
427 Colfax, except for DNA/*TcMar/Stowaway*, which exhibited reduced activation upon inoculation  
428 (Figure S5e). Overall, no significant differences in the total transcript levels of TEs were  
429 observed between the resistant and susceptible lines across most superfamilies (Figure S6).  
430 Interestingly, DNA/*PIF-Harbinger* showed higher transcript levels in both resistant lines at 2 hpi  
431 post the *P. sansomeana* inoculation, while the susceptible lines displayed comparable transcript  
432 levels between mock-inoculated and pathogen-inoculated plants (Figure S6d).

433 Next, we identified significantly differentially expressed TEs (DE TEs) between the mock  
434 and pathogen inoculated samples at different time points for each of the soybean lines (Dataset  
435 S4). The number of DE TEs varied among the four lines in response to the pathogen inoculation

436 (Figure 4a). In Colfax, comparable numbers of up-regulated and down-regulated DE TEs (25 and  
437 27, respectively) in response to the pathogen were detected only at 8 hpi. The proportion of TE  
438 superfamilies among the up-regulated TEs in Colfax mirrored the genome-wide level in the  
439 soybean genome, where approximately 70% of TEs represented retrotransposons and the  
440 remaining 30% were DNA transposons (Figure 4b). These up-regulated TEs were located mainly  
441 in pericentromeric regions (76%), similar to the genome-wide distribution (74%) (Figure 4c).  
442 Furthermore, more than 40% of the up-regulated DE TEs in Colfax were located proximal to  
443 genes in the genome (Figure 4d). In contrast, a larger proportion of the down-regulated DE TEs  
444 in Colfax consisted of DNA transposons (44%), particularly *CACTA* elements (37%).  
445 Interestingly, over two-thirds of the down-regulated DE TEs in Colfax were located in  
446 chromosomal arms, and a significant portion (90%) of these down-regulated DE TEs in Colfax  
447 were either within or in close proximity to genes (within 2 kb upstream or downstream), a  
448 marked contrast to the overall genome-wide distribution where less than 20% of TEs were  
449 positioned near genes. In Colfax, among the genes near or containing up-regulated DE TEs, a  
450 significantly up-regulated gene (*Glyma.16G135200*) exhibited an association with an up-  
451 regulated LTR/*Copia* element (ID 371175) (Figure 4e; Dataset S1). This gene encodes a  
452 Toll/interleukin-1-receptors (TIR)-NBS-LRR (TNL) protein (Swiderski et al., 2009), renowned  
453 for its regulatory role as a R protein in immune responses to *Phytophthora sojae* in soybean  
454 (Zhou et al., 2022). Additionally, in Colfax, among the DEGs with down-regulated DE TEs,  
455 three harbored DNA/*CACTA* elements within their coding regions (Dataset S4). Notably, two out  
456 of these three DEGs (*Glyma.19G016400* and *Glyma.13G063700*) were both ABC transporters  
457 that were also down-regulated upon inoculation (Dataset S1).

458 In NE2701, a considerably larger number of DE TEs (n=111) were up-regulated at a  
459 relatively earlier stage, 4 hpi (Figure 4a), compared to Colfax. The majority of up-regulated DE  
460 TEs in NE2701 were retrotransposons (90%), including LTR/*Copia* accounting for  
461 approximately half of all TEs (Figure 4b). Most (92%) of the up-regulated DE TEs in NE2701  
462 were located in pericentromeric regions, a proportion much higher than the overall genome-wide  
463 distribution (74%) (Figure 4c). Furthermore, in contrast to Colfax, the up-regulated DE TEs in  
464 NE2701 (95%) were primarily located outside gene regions (Figure 4d).

465 In the susceptible line, Senaki, a total of 272 DE TEs exhibited up-regulation at 2 hpi, while  
466 only one TE displayed down-regulation at the same time point (Figure 4a). Over half of the up-  
467 regulated DE TEs (58%) in Senaki were LTR/*Copia*, unlike the other lines and the genome-wide  
468 proportion. Nonetheless, the overall proportions of classes I and class II TEs were comparable to  
469 the genome-wide levels (Figure 4b). In the other susceptible line, Williams 82, a lower number  
470 of DE TEs (15) were observed to be up-regulated at 8 hpi. Out of the 15 up-regulated DE TEs,  
471 14 were retrotransposons. The DE TEs in both susceptible lines were primarily located in  
472 pericentromeric regions (85% and 93%, respectively) and outside gene regions (89% and 100%,  
473 respectively) (Figure 4c,d). Taken together, these data propose that pathogen attacks trigger the  
474 transcriptional activation of numerous TEs, and the transcriptional responses of TEs to *P.*  
475 *sansomeana* vary significantly across different soybean lines.

476

477 **LncRNA XLOC\_013220 is associated with the expression of its potential target genes**  
478 **following pathogen inoculation in the resistant lines**

479 LncRNAs have been reported to regulate gene expression in various biological processes  
480 including pathogen infection (Chekanova, 2015; Gil & Ulitsky, 2020). To investigate how

481 soybean lncRNAs respond to *P. sansomeana* and their potential interaction with gene expression,  
482 we first identified lncRNAs in soybean using the assembled and merged transcripts from the 64  
483 samples. Our analysis revealed a total of 43,759 lncRNA transcripts, comprising long intergenic  
484 non-coding RNAs (lincRNAs), sense-overlapping lncRNA transcripts (SOT), and antisense-  
485 overlapping lncRNA transcripts (AOT) (Table S3). The vast majority of the identified lncRNAs  
486 were lincRNAs (95%; 41,159), with a smaller proportion representing SOT or AOT (3% and 2%,  
487 respectively) (Figure 5a). Additionally, based on sequence similarities with TEs, we classified  
488 lincRNAs into non-TE lincRNAs and TE-containing lincRNAs, possibly indicating historical TE  
489 integration or exaptation events (Nelson et al., 2017). Among the identified lncRNAs,  
490 approximately 58% (27,672) were designated as TE-containing lincRNAs, while the remaining  
491 37% (13,487) were classified as non-TE lincRNAs (Figure 5a).

492 We next compared the proportion of transcription and transcript levels among non-TE  
493 lincRNAs, TE-containing lincRNAs, genes, and TEs. Among these four transcript types,  
494 transcribed genes showed the highest proportion, accounting for 74% of the total transcripts  
495 (Figure S7a). In the context of lincRNAs, non-TE lincRNAs exhibited a higher transcription  
496 proportion at 15%, in contrast to TE-containing lincRNAs with 12%. In contrast, TEs  
497 demonstrated the lowest transcription proportion at 3% compared to the other four transcript  
498 types. This trend was consistent when considering mean transcript levels, where transcribed  
499 genes exhibited significantly higher levels compared to the other transcript types (Figure S7b).  
500 Similarly, within the other three transcript types, non-TE lincRNAs exhibited higher mean  
501 transcript levels, while TEs displayed the lowest mean transcript levels, characterized by  
502 relatively larger variability compared to lincRNAs (Figure S7b).

503 Upon the *P. sambucus* inoculation, we observed differential expression in a total of 497  
504 unique lincRNAs compared to mock-inoculated plants (Figure 5b; Dataset S5). The majority of  
505 differentially expressed lincRNAs (96%) were non-TE lincRNAs across all four soybean lines,  
506 except for NE2701 at 4 hpi, where 131 TE-containing lincRNAs were up-regulated. Of these 131  
507 TE-containing lincRNAs, 38 (29%) directly overlapped with DE TEs. In contrast, both  
508 susceptible lines, Senaki and Williams 82, showed fewer numbers of DE lincRNAs, most of  
509 which were up-regulated following inoculation (Figure 5b).

510 We further investigated common lincRNAs exclusively present in either the resistant or  
511 susceptible lines. Remarkably, we identified eight non-TE lincRNAs exclusively up-regulated in  
512 the resistant lines at 8 and 16 hpi (Figure 5c,d). A correlation analysis between these eight  
513 lincRNAs and DEGs revealed a wide range of co-expressed genes with each lincRNA (Figure 5e;  
514 Dataset S6). Among these, XLOC\_013220 and XLOC\_098355 were linked to nine and two co-  
515 expressed DEGs, respectively. Additional non-TE lincRNAs, such as XLOC\_014629,  
516 XLOC\_084885, and XLOC\_063809, exhibited a relatively higher number of co-expressed DEGs.  
517 The enriched GO terms for the DEGs associated with XLOC\_014629 and XLOC\_084885  
518 primarily pertained to the biosynthesis of various secondary metabolites. In contrast, DEGs  
519 involved in salicylic acid biosynthesis were enriched for XLOC\_063809 (Table S4).

520 We also explored potential *cis*-target genes by identifying adjacent genes ( $\pm 5$  kb) to the  
521 eight non-TE lincRNAs in the resistant lines (Figure 5c,d). Among these lincRNAs, four  
522 lincRNAs (XLOC\_053188, XLOC\_014629, XLOC\_084885, and XLOC\_063809) had a single  
523 adjacent gene each (Dataset S7). However, these genes were not DEGs (Dataset S1).  
524 Remarkably, an intriguing lincRNA, XLOC\_013220, located between two flanking genes  
525 (*Glyma.03G040900* and *Glyma.03G041000*), both encoding LURP-one-related (LOR) proteins,

526 exhibited a significant correlation with the transcript level of these two genes (Figure 5e). LORs  
527 have been demonstrated to play a role in defense responses to oomycete pathogens in  
528 *Arabidopsis* (Baig, 2018; Knoth & Eulgem, 2008). Interestingly, both of these *LOR* genes were  
529 DEGs upon pathogen inoculation at both 8 and 16 hpi exclusively in the resistant lines (Dataset  
530 S1). Significantly, *Glyma.03G040900* was also identified as a potential *trans*-target gene,  
531 showing a strong correlation in transcript levels with the lincRNA ( $R = 0.85, P = 2.2\text{E-}16$ ). The  
532 up-regulation of this gene after pathogen inoculation in the resistant lines was further validated  
533 by qRT-PCR (Figure S8). Similarly, the transcript levels of the other *LOR* gene,  
534 *Glyma.03G041000*, also demonstrated a significant correlation with the lincRNA ( $R = 0.63, P =$   
535  $2.2\text{E-}08$ ). This particular lincRNA, XLOC\_013220, exhibited exclusive up-regulation in the  
536 resistant lines at 16 hpi, with a consistent trend also at 8 hpi, aligning with the expression  
537 patterns of the two flanking genes (Figure 5f; Dataset S1). This lincRNA is of particular interest  
538 as it is located within the mapped region of the resistance gene in the resistant line Colfax based  
539 on our mapping results (Lin et al., 2023). These findings strongly suggest that XLOC\_013220  
540 potentially plays a crucial role in regulating the expression of both neighboring *LOR* genes in  
541 response to the pathogen infection in the resistant lines.

542 Among the DE TE-containing lincRNAs, only one lincRNA was exclusively shared by the  
543 resistant lines (Figure S9). This lincRNA harbored a DE *CACTA* element (ID 294229, Dataset S4)  
544 embedded directly within its transcript region. Furthermore, we detected 49 DEGs that exhibited  
545 strong co-expression patterns with this specific lincRNA based on the correlation analysis  
546 (Dataset S8).

547

548 **CHH methylation levels in lincRNAs increase upon pathogen inoculation at the later time  
549 point in the resistant line compared to the susceptible line**

550 LncRNAs have been found to interact with DNA methyltransferase to mediate gene expression  
551 (Böhmdorfer et al., 2014; Zhao et al., 2016). In pursuit of a more profound understanding of the  
552 prospective role of lincRNAs in gene expression modulation, we investigated DNA methylation  
553 levels within non-TE lincRNAs between the resistant line Colfax and the susceptible line  
554 Williams 82. We first determined DNA methylation levels in the CG, CHG and CHH contexts  
555 within lincRNA transcript bodies, as well as in regions extending 2 kb upstream of the start  
556 positions and 2 kb downstream of the end positions. Strikingly, across all regions and in each of  
557 the three cytosine contexts, the resistant line Colfax demonstrated marginally lower methylation  
558 levels in lincRNAs compared to the susceptible line Williams 82 (Figure 6 and Figure S10),  
559 suggesting distinct methylation patterns in lincRNAs across different soybean genetic  
560 backgrounds.

561 To further understand the role of methylation in response to the pathogen, we examined the  
562 methylation changes upon pathogen inoculation. While no significant methylation changes in CG  
563 and CHG contexts were observed after pathogen inoculation in either the resistant or susceptible  
564 lines within 2 kb flanking regions and lincRNA bodies (Figure S10), CHH methylation levels  
565 increased in both lines following inoculation (Figure 6). Interestingly, the CHH methylation  
566 pattern and timing of response to the pathogen inoculation varied between the two lines. In the  
567 resistant line Colfax, CHH methylation levels in lincRNAs exhibited a more pronounced  
568 increase at 16 hpi, particularly within the transcript bodies (Figure 6a). In contrast, in the  
569 susceptible line Williams 82, CHH methylation levels in lincRNAs showed a more pronounced  
570 increase at the earlier time point of 4 hpi. The distribution of CHH methylation across 2 kb

571 flanking regions also showed earlier increases at 4 hpi in the susceptible line and delayed  
572 changes at 16 hpi in the resistant line (Figure 6b). These results suggest that lncRNAs in the  
573 resistant line may possess a relatively higher stability with respect to epigenetic changes in  
574 response to pathogen attack compared to those in the susceptible line.

575

## 576 Discussion

### 577 *P. sansomeana* is likely a hemibiotrophic oomycete pathogen

578 Our transcriptomic analyses demonstrated that genes involved in the ET signaling pathway were  
579 significantly up-regulated during later stages of *P. sansomeana* inoculation (8 and 16 hpi) in the  
580 resistant lines (Figures 2, 3, and Table S2). This coordinated activation likely interacts with the  
581 up-regulation of multiple transcription factors that initiate and modulate diverse defense  
582 responses. Among these transcription factors, *ERFs* have well-documented roles in intricate  
583 networks involving ET and other hormones to enhance stress tolerance in plants (Adie et al.,  
584 2007; Dubois et al., 2018; Thirugnanasambantham et al., 2015). Several up-regulated *ERF* genes  
585 associated with the ET signaling exhibited a co-expression pattern in response to *P. sansomeana*  
586 (Figure 3).

587 ET, in concert with JA, is a key signal molecule in host defense response against  
588 necrotrophic and later-stage hemibiotrophic pathogens (Huang et al., 2020; van Loon et al.,  
589 2006). Clear evidence shows that many *Phytophthora* spp. pathogens, such as *Phytophthora*  
590 *infestans* and *P. sojae*, follow a hemibiotrophic life cycle, initiating the infection cycle as  
591 biotrophs but switching to a necrotrophic lifestyle at later stages (Lee & Rose, 2010; Qutob et al.,  
592 2002; Zuluaga et al., 2016). In *P. sojae*, an avirulence (effector) gene product with an RxLR  
593 (Arginine-any amino acid-Leucine-Arginine) motif can interact with a corresponding soybean

594 *Rps* gene, resulting in the rapid host activation of defense responses and plant resistance  
595 (Arsenault-Labrecque et al., 2022; Dong et al., 2011; J. D. Jones & J. L. Dangl, 2006; Na et al.,  
596 2013; Ngou et al., 2022; Shan et al., 2004). As disease progresses, *P. sojae* feeds on dead plant  
597 tissues, causing severe lesions and leading to necrosis (Qutob et al., 2002). According to our  
598 mapping results, a single dominant resistance gene contributes major resistance to *P.*  
599 *sansomeana* (Lin et al., 2023), suggesting that *P. sansomeana* possesses similar genetic qualities,  
600 such as effectors. The up-regulation of the ET signaling pathway and the ROS metabolic process  
601 at 8 and 16 hpi from our transcriptomic data and the H<sub>2</sub>O<sub>2</sub> experiment demonstrated necrotrophic  
602 symptoms. Overall, these findings suggest that, similar to other *Phytophthora* spp. pathogens, *P.*  
603 *sansomeana* is likely a hemibiotrophic pathogen. Future investigation into the life cycle of *P.*  
604 *sansomeana* could provide additional evidence.

605

#### 606 **LncRNA XLOC\_013220 potentially regulates adjacent *LORs* upon pathogen invasion**

607 LncRNAs can regulate diverse defense mechanisms by influencing the expression of resistance-  
608 related genes at both transcriptional and post-transcriptional levels (Sharma et al., 2022). In this  
609 study, we identified several hundreds of *trans*-targets for DE lncRNAs upon pathogen  
610 inoculation in the resistant lines, providing potential candidate target genes or co-expressed  
611 downstream genes that are induced in response to *P. sansomeana* (Figure 5). These lncRNAs  
612 may directly control or indirectly influence pathways involved in defense responses, such as  
613 lignan and SA biosynthesis (Table S4).

614 Of these DE lncRNAs that co-expressed with DEGs, an especially intriguing instance  
615 involves the lncRNA XLOC\_013220, which experienced up-regulation upon pathogen  
616 inoculation. This lncRNA is of particular interest as it lies within the mapped region of the

617 resistance gene in the resistant line Colfax (Lin et al., 2023). More interestingly, it is positioned  
618 adjacent to the two *LOR* genes, presumably generated by tandem duplication. *LURP1*, previously  
619 identified to be up-regulated upon recognition of pathogenic oomycetes in plants, is a key gene  
620 controlling basal defense against the oomycete species *Hyaloperonospora parasitica* in  
621 *Arabidopsis thaliana* (Knoth & Eulgem, 2008). It is worth noting that *LUPR1* in *A. thaliana*  
622 possesses a W-box and two TGA-box motifs that might engage with members of the WRKY  
623 family, which play roles in both PTI and ETI responses. In addition, a *LOR* gene, belonging to  
624 the *LURP* cluster, has been recently unveiled as a contributor to basal defense against another  
625 oomycete, *Hyaloperonospora arabidopsis* (Baig, 2018). In *Arabidopsis*, both *LURP1* and *LOR*  
626 are involved in the SA-dependent pathway, orchestrating immune responses to these oomycete  
627 pathogens.

628 Given the close proximity and the highly correlated expression patterns between the  
629 lincRNA and the two *LOR* genes in the resistant lines, it is possible that this lincRNA can exert a  
630 regulatory role in the expression of its neighboring *LOR* genes, thereby contributing to defense  
631 responses in the resistant soybean lines against the oomycete pathogen *P. salsomeana*. The  
632 lincRNA could function as a *cis*-regulatory element, modulating the transcriptional activity or  
633 stability of the *LOR* genes (Gil & Ulitsky, 2020). Alternatively, the lincRNA might be involved  
634 in coordinating the expression of these genes within a larger regulatory network activated in  
635 response to the pathogen. Further investigation, such as functional studies or genetic  
636 manipulations, would be necessary to elucidate the precise role of the lincRNA in regulating the  
637 expression of the neighboring *LOR* genes during the pathogenic response.

638

639 **Resistant and susceptible lines exhibit distinct patterns of CHH methylation in lincRNAs**  
640 **against *P. sansomeana***

641 Our methylation data revealed that CHH methylation levels in lincRNAs and their flanking  
642 regions in response to the *P. sansomeana* inoculation increased at distinct time points in both the  
643 resistant and susceptible lines, while the CG and CHG methylation levels remained unchanged  
644 (Figure 6; Figure S10). Although the methylation levels of CHH cytosines are generally very low,  
645 they are remarkably abundant in the soybean genome (Song et al., 2013), making them potential  
646 candidates for buffering the global impact of environmental stresses, such as pathogen attacks,  
647 on transcriptional activation of TEs to maintain genome stability. Interestingly, although both the  
648 resistant and susceptible lines exhibited increased CHH methylation levels in response to *P.*  
649 *sansomeana*, the timing of this increase differed between them (Figure 6). In the resistant line, a  
650 more significant increase in CHH methylation levels in lincRNAs occurred at 16 hpi. In contrast,  
651 the susceptible line exhibited this increase at an earlier time point, 4 hpi. This divergence in  
652 timing may be attributed to their distinct genetic backgrounds or variations in the kinetics of their  
653 defense response. The resistant line seems to mount a delayed yet sustained CHH methylation  
654 response, contributing to a more gradual and prolonged defense response. On the other hand, the  
655 susceptible line experienced an earlier but potentially transient increase in global CHH  
656 methylation triggered by *P. sansomeana* infection.

657  
658 **Distinct genetic backgrounds may contribute to divergent defense strategies between the**  
659 **two soybean resistant lines against *P. sansomeana***  
660 In addition to common defense responses shared by both resistant lines, we further dissected  
661 unique strategies employed by each resistant line. In the resistant line Colfax, notable

662 transcriptomic changes, spanning genes, TEs, and lincRNAs, primarily occurred at 8 or 16 hpi in  
663 response to the pathogen. At 16 hpi, an exclusive and significantly enriched biological process in  
664 Colfax for the up-regulated genes was the phosphate-containing compound metabolic process,  
665 which encompasses crucial phosphorylation events (Table S1). This process might play a role in  
666 activating *ERFs*, crucial in ET-mediated immune responses (Adie et al., 2007;  
667 Thirugnanasambantham et al., 2015; Wang et al., 2022). By contrast, the resistant line NE2701  
668 exhibited larger proportions of differentially transcribed genes, TEs, and lincRNAs at relatively  
669 earlier time points, 2 or 4 hpi. Notably, the primary biological process initiated immediately after  
670 pathogen attack at 2 hpi in NE2701 was the JA-mediated defense signaling pathway being  
671 triggered.

672 The divergent patterns of defense responses between the two resistant lines were also  
673 evident in differentially transcribed TEs and lncRNAs. In Colfax, DE TEs were largely located  
674 within gene regions (Figure 4d). Of particular interest was the up-regulated TE (LTR/Copia)  
675 located closely downstream of the significantly up-regulated *TNL* gene (*Glyma.16G135200*),  
676 which showed a similar gene expression pattern to the TE (Figure 4e; Dataset S1). Recently, a  
677 *TNL* was newly identified in soybean, demonstrating resistance against Phytophthora root rot  
678 (Zhou et al., 2022), and has been shown to increase JA- and SA-mediated disease resistance. Our  
679 finding suggests a potential role for this TE as a *cis*-regulatory element, modulating the  
680 expression of the *TNL* gene in defense responses to the pathogen in Colfax.

681 On the other hand, the NE2701 resistant line showed a higher number of up-regulated TEs  
682 and lincRNAs in response to *P. sansomeana*, particularly at the relatively earlier time point, 4  
683 hpi (Figures 4 and 5). This suggests a more rapid alleviation of silencing of TEs in NE2701 in  
684 response to the pathogen compared to Colfax. Strikingly, unlike Colfax, these DE TEs in

685 NE2701 were located predominantly outside gene regions. Interestingly, over one-third of the  
686 up-regulated TEs in NE2701 overlapped with lincRNAs, which were also up-regulated post  
687 inoculation, suggesting that some of these TEs could serve as sources for lincRNAs that  
688 modulate transcriptomic responses to the pathogen (Sharma et al., 2022; Zhang et al., 2020).

689 It is worth noting that the different timing and patterns of transcriptional responses between  
690 the two resistant lines can be attributed to their unique genetic backgrounds. Given that NE2701  
691 is originally derived from a cross between Colfax and A91-701035, it is most likely that they  
692 share the same *R* gene responsible for the resistance against *P. samborum*. However, A91-  
693 701035 has a divergent genetic background due to consecutive crosses with multiple other  
694 soybean lines (Graef et al., 2005). As a result, there may be some additional minor alleles from  
695 A91-701035 that also contribute to the disease response, in addition to the *R* gene inherited from  
696 Colfax. These unique genetic backgrounds likely shape the defense strategies observed in each of  
697 the resistant lines, highlighting the potential benefits of leveraging diverse genetic backgrounds  
698 for breeding more resilient and effective soybean cultivars against pathogenic challenges.

699

## 700 **Acknowledgements**

701 We thank HiPerGator Supercomputer for providing us with the computational resources to  
702 perform the analysis.

703

## 704 **Funding**

705 This work was supported by the National Science Foundation under Award Number  
706 IOS2128023, the National Institute of General Medical Sciences of the National Institutes of  
707 Health under Award Number R15GM135874, as well as startup funds from Miami University

708 and the University of Florida to M.Z. We also express our gratitude for partial support from the  
709 Michigan Soybean committee, North Central Soybean Research Program, and Project GREEEN-  
710 Michigan's plant agriculture initiative to M.I.C. Additionally, we thank funding support from the  
711 Michigan Soybean Committee, Project GREEEN-Michigan's plant agriculture initiative,  
712 AgBioResearch at Michigan State University (Project No. MICL02013), North Central Soybean  
713 Research Program, United States Department of Agriculture National Institute of Food and  
714 Agriculture (Hatch project 1011788), and the United Soybean Board (24-209-S-A-1-A) to D.W.  
715 The funders had no role in study design, data collection and analysis, decision to publish, or  
716 preparation of the manuscript.

717

718 **Competing interests:** The authors declare no conflict of interest.

719

720 **Data Availability Statement**

721 The raw and processed data of mRNA and whole genome bisulfite sequencing presented in this  
722 study have been deposited in NCBI Gene Expression Omnibus under the accession number  
723 GSE240966, <https://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE240966>.

724

725 **Supporting information**

726 **Figure S1. Phenotypes of mock- and *P. sansomeana*-inoculated plants of the four soybean  
727 lines.** The photos were taken five days after mock- or pathogen-inoculation for four lines.

728 **Figure S2. Principal component analysis (PCA) of 64 soybean transcriptomes.** Variant  
729 stabilizing transformations of read counts were used for PCA to determine the sample distance in

730 four soybean lines. The percentages of variation explained by PC1 and PC2 are indicated in  
731 parentheses.

732 **Figure S3. Transcript levels of *Glyma.09G210600 (RPS3)*, exclusively up-regulated at 16 hpi**  
733 **in both resistant lines.** (a) Transcript levels of *RPS3* from RNA sequencing. Normalized counts  
734 were used for transcript levels at different time points for each line and condition. (b) qRT-PCR  
735 quantification of *RPS3*. Transcript levels of *RPS3* were normalized to *Cons4*, a constitutively  
736 expressed control gene. A distinct biological replicate independent of the samples used for RNA  
737 sequencing was used for relative transcript level (RTLs) quantifications.

738 **Figure S4. Identified co-expressed gene clusters in the four lines.** (a-d) All clusters grouped  
739 based on k-means clustering in Colfax (cf; a), NE2701 (ne; b), Senaki (sk; c), and Williams 82  
740 (wm; d). The X-axis texts represent control (c) and inoculated (t) samples at different time points.  
741 The complete list of genes in these clusters can be found in Dataset S2.

742 **Figure S5. Proportion of transcribed TEs in each superfamily.** (a-j) Proportion of transcribed  
743 TEs (NCPK; normalized counts per kilobase of TE length  $> 0.5$ ) in different lines and conditions.  
744 The proportions were averaged between the two replicates.

745 **Figure S6. Total transcript levels of TEs in each superfamily.** (a-j) The expression levels of  
746 TEs in ten superfamilies. The expression levels were determined by the mean normalized counts  
747 divided by the length of each TE (NCPK) for TEs within each superfamily, across different lines  
748 and conditions. The error bars represent SE.

749 **Figure S7. Transcription patterns of genes, lincRNAs and TEs.** (a) The proportion of  
750 transcribed transcripts for each transcript type. NCPK (normalized counts per kilobase of length  
751 of transcripts or genes) of all 64 samples was used for calculating the proportion of transcribed  
752 transcripts (NCPK  $> 0.5$ ). (b) Comparison of transcript levels for four transcript types.

753 Log(NCPK + 1) was used as a transcript level only for transcribed transcripts as depicted in (a).  
754 The red points indicate mean transcript levels. Different characters represent statistically  
755 significant differences between means (ANOVA followed by Tukey's HSD test;  $P < 0.05$ ).

756 **Figure S8. qRT-PCR quantification of *LOR1* (*Glyma.03G040900*).** Transcript levels of *LOR1*  
757 were normalized to *Cons4*, a constitutively expressed control gene. A distinct biological replicate  
758 independent of the samples used for RNA sequencing was used for transcript quantification.

759 **Figure S9. TE-containing lincRNAs exclusively differentially expressed in the resistant**  
760 **lines.** (a) The number of up-regulated TE-containing lincRNAs at 8 hpi. Among the different  
761 time points and treatments, only a single up-regulated lincRNA was found in common in the  
762 resistant lines. The red number represents the shared DE TE-containing lincRNA in the resistant  
763 lines. (b) Transcript levels of the DE TE-containing lincRNA, XLOC\_059371, under different  
764 lines and conditions. Moreover, it was exclusively detected in the resistant lines at 8 hpi. A total  
765 of 49 co-expressed DEGs were linked with this lincRNA ( $|R| > 0.85$ ;  $P < 0.001$ ), but no adjacent  
766 DEG was found within 10 kb upstream or downstream. The TE within this lincRNA is classified  
767 as DNA/CACTA (ID: 294229).

768 **Figure S10. No significant changes in CG and CHG methylation on lincRNA bodies and**  
769 **their flanking regions upon pathogen inoculation.** (a) Mean proportions of CG and CHG  
770 methylation in non-TE lincRNAs for each line and condition. The error bars represent  $\pm$  SE.  
771 Asterisks indicate statistically significant differences of means in inoculated plants compared to  
772 controls (Student's  $t$  test;  $P \geq 0.05$ , ns, not significant). (b) Distribution of CG and CHG  
773 methylation within 2 kb upstream and downstream of non-TE lincRNA transcript bodies. The  
774 mean methylation proportion was calculated in 40 windows for each of upstream, body, and  
775 downstream of transcripts. The lines were smoothed using LOESS.

776 **Table S1. Five most significantly enriched GO terms exclusive to each resistant line.**

777 **Table S2. Gene list in the enriched GO term, “response to ethylene” among co-expressed**  
778 **genes in the resistant lines (refer to Figure 3f).**

779 **Table S3. Summary of identified lncRNAs in soybean transcriptomes.**

780 **Table S4. Overrepresented GO terms of potential *trans*-target genes of DE lncRNAs (refer**  
781 **to Figure 5e).**

782 **Dataset S1. Description and normalized counts of DEGs upon inoculation in four soybean**  
783 **lines.**

784 **Dataset S2. Selected clusters for up-regulated genes upon inoculation.**

785 **Dataset S3. Enriched GO terms for genes in the selected clusters from Dataset S2.**

786 **Dataset S4. DE TEs upon inoculation and their location nearby or within DEGs or DE**  
787 **lncRNAs.**

788 **Dataset S5. DE lncRNAs upon inoculation with overlapping DE TEs.**

789 **Dataset S6. Correlated DEGs with DE non-TE lncRNAs as potential *trans*-targets ( $|R| >$**

790  **$0.85, P < 0.001$ .**

791 **Dataset S7. DEGs located within 5 kb upstream or downstream of DE non-TE lncRNAs as**  
792 **potential *cis*-targets.**

793 **Dataset S8. Correlated DEGs with the DE TE-containing lncRNA, XLOC\_059371,**  
794 **exclusively in the resistant lines ( $|R| > 0.85, P < 0.001$ ).**

795

796

797 **Figure legends**

798 **Figure 1. Survival rates of four soybean lines after mock (control) and *P. sansomeana***  
799 **inoculation (inoculated).** Survival seedlings were counted seven days post inoculation. The  
800 results are presented as mean  $\pm$  SE ( $n = 16$  per line per treatment). Different characters were used  
801 to denote statistically significant differences between means based on two-way ANOVA,  
802 followed by Tukey's HSD test ( $P < 0.05$ ).

803  
804 **Figure 2. Transcriptomic responses to pathogen inoculation reveal contrasting patterns in**  
805 **the resistant and susceptible soybean lines.** (a) The number of significant DEGs for four lines  
806 (adjusted  $P < 0.05$ ) at four different time points (2, 4, 8, and 16 hours post inoculation, hpi). (b)  
807 Shared and unique up- and down-regulated DEGs at each time point. The upper row indicates  
808 up-regulated DEGs, while the lower row represents down-regulated DEGs. Red numbers  
809 indicate DEGs exclusively shared in either the resistant or susceptible lines. (c) Overrepresented  
810 GO terms of 204 up-regulated DEGs exclusively in the resistant lines at 8 hpi. (d)  
811 Overrepresented GO terms of 308 up-regulated and 42 down-regulated DEGs exclusively in the  
812 resistant lines at 16 hpi. (e) Fold changes in  $H_2O_2$  concentration after *P. sansomeana* inoculation  
813 at 16 hpi.  $\log_2$  fold change was calculated based on  $H_2O_2$  concentration between mock- and  
814 pathogen-inoculated plants at 16 hpi. GO terms analysis (refer to Figure 2d) identified the  
815 enrichment of  $H_2O_2$  catabolic process and reactive oxygen species metabolic process for up-  
816 regulated genes in the resistant lines (Colfax and NE2701) after pathogen inoculation. The error  
817 bars represent SD (standard deviation) among three separate measurements. (f) Overrepresented  
818 GO terms of 22 up-regulated and 4 down-regulated DEGs exclusively in the susceptible lines at  
819 4 hpi. For panels c, d, and f, the numbers within the bars denote the count of genes enriched in

820 each GO term (BP, Biological process; CC, Cellular component; MF, Molecular function; KEGG,  
821 Kyoto encyclopedia of genes and genomes).

822

823 **Figure 3. Co-expressed gene clusters up-regulated upon pathogen inoculation in the**  
824 **resistant lines are enriched in ethylene-responsive genes.** (a-d) Co-expression gene clusters  
825 demonstrating similar patterns of up-regulation upon pathogen inoculation in Colfax (a), NE2701  
826 (b), Senaki (c), and Williams 82 (d). These clusters were selected from the overall clusters,  
827 grouped based on  $k$ -means clustering (see Figure S4). The X-axis indicates time points: 2, 4, 8,  
828 and 16 hpi. The detailed gene list for each selected cluster can be found in Dataset S2, with  
829 enriched GO terms available in Dataset S3. (e) Shared and unique up-regulated DEGs within  
830 clusters across the four lines after inoculation. (f) Overrepresented GO terms in the gene lists of  
831 shared genes exclusively in the resistant lines. A total of 370 genes marked in red in (e) were  
832 used for the GO analysis. All three GO terms are classified under the biological process category.

833

834 **Figure 4. The majority of differentially expressed TEs are up-regulated in response to *P.***  
835 ***sansomeana* and those in Colfax are enriched near genes.** (a) The number of up- and down-  
836 regulated TEs in four lines upon inoculation at different time points (2, 4, 8, and 16 hpi). (b) The  
837 percentage of differentially expressed (DE) TEs in each line. The term “genome-wide” indicates  
838 the TE composition in the soybean reference genome. Retrotransposons (class I) are represented  
839 by four families in reddish colors, while DNA transposons (class II) are represented by six  
840 superfamilies in bluish colors. (c) The percentage of DE TEs between pericentromeric and  
841 chromosomal arms. The leftmost pie indicates the locations of all TEs in the genome, serving as  
842 a control. (d) The percentage of DE TEs near or within genes. Each pie, except the “genome-

843 “wide” in (b-d), corresponds to the respective soybean line in (a). The numbers in (b-d) denote the  
844 respective percentages. (e) Diagram illustrating the position of a TE (LTR/*Copia*) downstream of  
845 the *TNL* gene on chromosome 16. Both the TE and gene were up-regulated in Colfax upon  
846 pathogen inoculation. The dark blue boxes indicate the five exons in the *TNL* gene.

847

848 **Figure 5. LncRNA XLOC\_013220 is associated with the expression of its potential target**  
849 **genes following pathogen inoculation in the resistant lines.** (a) The proportion of identified  
850 lncRNAs in the soybean transcriptomes. (b) The number of up- and down-regulated lncRNAs  
851 upon inoculation in each line at different time points ( $P < 0.01$ ). (c-d) The number of up-  
852 regulated non-TE lncRNAs at 8 hpi (c) and 16 hpi (d), where shared lncRNAs were exclusively  
853 found in either the resistant or susceptible lines. The shared lncRNAs were only present in the  
854 resistant lines as up-regulated at 8 and 16 hpi (no shared down-regulated lncRNAs were  
855 identified). The red number represents the shared DE non-TE lncRNAs in the resistant lines. (e)  
856 The number of potential *trans-* and *cis*-target genes for each lncRNAs identified as shared DE  
857 non-TE lncRNAs in the resistant lines in (c) and (d). The red number indicates the gene  
858 *Glyma.03G040900*, which encodes a LURP-one-related (LOR) protein and is recognized as both  
859 the potential *trans-* and *cis*-target of XLOC\_013220. (f) Transcript level change of  
860 XLOC\_013220 at different time points for each line and condition.

861

862 **Figure 6. CHH methylation levels in lncRNAs increase upon pathogen inoculation at the**  
863 **later time point in the resistant line compared to the susceptible line.** (a) Mean proportions of  
864 CHH methylation within non-TE lncRNA transcript bodies, and 2 kb up or downstream of these  
865 bodies for each line and condition. The error bars depict  $\pm$  SE. Asterisks denote statistically

866 significant differences in means between inoculated plants and controls (Student's *t* test; \*\*\*,  
867  $P \square$  value < 0.001; \*\*,  $P < 0.01$ ; \*,  $P < 0.05$ ; ns, not significant,  $P \geq 0.05$ ). (b) Distribution of  
868 CHH methylation within 2 kb upstream and downstream of non-TE lincRNA transcript bodies.  
869 The mean methylation proportion was calculated in 40 windows for each of upstream, body, and  
870 downstream of transcripts. The lines were smoothed using locally estimated scatterplot  
871 smoothing (LOESS).

872

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