Effects of urban traffic noise on the early growth and transcription of *Arabidopsis thaliana*

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SUMMARY

Man-made noise in cities has been recognized as one of the major contributors to a range of adverse health outcomes, including heart disease. Besides the detrimental effects on human health, noise can act as a potential threat to other organisms in the ecosystem. Despite the growing concern, the biological effects of noise pollution on plants, in particular, remain largely obscure. Therefore, this study aimed to investigate the impacts of urban traffic noise on the growth of *Arabidopsis thaliana* **(***A. thaliana***). We hypothesized that traffic noise could stimulate plant growth (seed germination and seedling growth) since noise treatment has been reported to improve immunity and drought tolerance in** *A. thaliana* **(1, 2). Seeds in the experimental group were exposed to 48 hours of traffic noise recorded in a highly congested area of Seoul, while the control group was exposed to background white noise. Compared with background white noise, traffic noise exposure had no effects on seed germination but significantly increased the growth of seedlings by 39%. We further investigated the traffic noise-induced molecular changes in the** *A. thaliana* **seedlings by employing RNA sequencing. We detected aberrant expression of 690 genes in traffic noise-exposed seedlings. The differentially expressed genes were enriched for various biological pathways, such as stress response and auxin signaling. The observed molecular changes suggest profound impacts of traffic noise on the physiology of** *A. thaliana***. The results of our research can help shed light on the fundamental principles underlying the noise-plant interaction as well as provide a further basis for conserving our ecosystem.**

INTRODUCTION

With rapid urbanization, the emergence of vast transportation systems and industries gave rise to new acoustic conditions. Noise affects animal behavior, including vigilance, movement, foraging, and acoustic communication, and induces changes in animal physiology, population dynamics, fitness, and the ecosystem (3, 4). For example, a previous study that examined the effect of noise on ecological services found that noise increased flower pollination by hummingbirds but decreased seed dispersal, thereby influencing ecosystem structure and diversity (5). Considering its varied impacts on ecosystems, environmental noise is receiving substantial attention from not only biologists but also policymakers and resource managers, who all strive to grasp a more comprehensive understanding of the field.

Sound is physical energy in which acoustic, oscillatory pressure waves are transmitted. Numerous organisms, specifically plants, have evolved the ability to react to different ambient sounds and sound waves that have specific intensities and frequencies. Such responses trigger a range of molecular, cellular, and physiological responses in plants (6). In cities, plants are frequently exposed to white noise and noise pollution. White noise is a complex sound that covers a wide range of audible frequencies, while noise pollution is unfavorable noise caused by human activities such as road traffic and construction, as defined by the World Health Organization (WHO) (7, 8).

Sound can have multiple potential health impacts on plants. Previous studies on the physiological impacts of noise on plants have revealed mixed results. On one hand, a previous study reported the harmful effects of traffic noise on *Tagetes patula* and *Salvia splendens* (9). Compared with the control group placed in complete silence, the experimental group, which was exposed to 16 h of road traffic noise for 15 consecutive days, experienced a notable decrease in growth indices. This observation suggests that traffic noise may cause oxidative damage and disrupt the hormonal balance of plants (9).

On the other hand, more studies have indicated the beneficial effects of sound on plant physiology, resulting in enhanced growth, development, and disease resistance (1, 2, 10). For example, insects' chewing sound induced plant immunity-related chemicals such as glucosinolates in *Arabidopsis thaliana (A. thaliana)* (10). Exposure of 6-weekold *A. thaliana* plants to 100 dB of white noise for 10 h resulted in the upregulation of genes involved in pathogen response and promoting improved drought tolerance (1). The production of plant defense-related hormones, including salicylic acids, also increased when *A. thaliana* plants were exposed to 1000 Hz sound with 100 dB (2). Thus, it has been observed that noise treatment, when used as a physical stimulant, may bring key adaptive advantages to plant physiology.

Given the inconclusive findings in the literature, there is a need for more research on the effects of noise on the early growth of *A. thaliana*. Consequently, we investigated how traffic noise affects the growth and gene expression of this plant. *A. thaliana* has been a widely used experimental model organism since the 1980s, especially since researchers merged genetics with robust molecular biology methods. In 2000, the *A. thaliana* reference genome became the first publicly available genome sequence of a flowering plant, allowing scientists to make significant progress in understanding molecular principles (11). This plant is also

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Figure 1. Urban traffic noise and control white noise waveforms. Waveforms of traffic noise (A) and background control noise (B) were analyzed using the Sound Analyzer software. X-axis indicates the range of sound frequency between 5 and 22 kHz. Y-axis represents the intensity of sound from 0 to 90 dB.

used in research on ecological and evolutionary contexts in natural environments (12).

Based on previous findings that noise exposure boosted *A. thaliana* immunity and improved drought tolerance, we hypothesized that traffic noise treatment would positively impact both seed germination and seedling growth (1, 2, 10). Although the seed germination events did not show a significant difference between the control and experimental groups, noise-treated seedlings were significantly longer than control plants. After 48 h of noise treatment, bulk RNAsequencing (RNA-Seq) analysis was conducted to detect differences in the gene expression profiles of the whole seedlings. Intriguingly, we detected significant transcriptional changes responsible for stress response, auxin signaling pathway, as well as growth control, thus providing insights into the biological responses to noise pollution. Taken together, our findings demonstrate the stimulatory effects of traffic noise on the early growth of *A. thaliana.* Hence, our findings underscore the need for further investigation into the functional consequences of traffic noise on plant physiology and related ecological impacts.

RESULTS

To test whether environmental noise can stimulate seed germination and seedling growth, we first recorded traffic noise in several areas with high traffic density in Gangnam, Seoul. A total of 10 minutes of traffic noise contained at least three types of sources, such as buses & motorcycles, ambulances, and subway stations (**Figure 1A**). The treatment group of *A. thaliana* seeds was subjected to traffic noise at an average of 75 dB, with a minimum of 57 dB and a maximum of 90 dB, for 48 h. The control group was placed outside the sound-proof facility and exposed to background white noise at approximately 10 dB, ranging from a minimum of 5 dB to a maximum of 15 dB (**Figure 1B**).

After 48 h of continuous noise exposure, seed germination was determined (**Figure 2**). The criterion for germination was the presence of seed harboring protrusions of radicle, which is the first part of a seedling to emerge from the seed during the germination process. The percentage of germinated seeds was not significantly different between control (71.3% ± 12.22) and noise-treated (79.33% ± 3.05) seeds (*p* = 0.33, **Figure 3**).

Thus, 48 h of traffic noise did not affect the germination of seeds.

Next, we assessed the effects of noise exposure on seedling growth by measuring the length of seedlings from the beginning of the hypocotyl to the tip of the roots. Seedlings in the traffic noise treatment group had significantly longer seedling lengths (10.95 \pm 2.02 mm) than those in the control group (7.89 \pm 1.99 mm), corresponding to noise-triggered growth stimulation of up to 39% ($p=4.6 \times 10^{-14}$, **Figure 4**). These results thus indicate that 48 h of acute traffic noise treatment promoted early growth of *A. thaliana* seedlings.

To gain a deeper understanding of the molecular mechanisms occurring in the traffic noise-exposed seedlings, we performed RNA-seq of seedlings from traffic noise treatment and from control samples. A total of 690 differentially expressed genes (DEGs) (401 upregulated and 289 downregulated) were identified between the control and noise-exposed seedlings based on a fold change threshold of ≥ 1.5 for upregulation and ≤ −1.5 for downregulation (13) (**Figure 5**, **Table 1**).

To further clarify the biological pathways and functions in the *A. thaliana* seedlings affected by noise treatment, we classified DEGs based on biological processes (**Figure 6**, **Table 2**). Significantly enriched gene ontology categories were identified in the noise-exposed seedlings, including "stress response", "auxin signaling pathway", "growth regulation", "protein transport", "tricarboxylic acid cycle", "autophagy", and "hydrogen ion transport". Taken together, genome-wide transcriptomic analysis strongly suggests pleiotropic effects of traffic noise stimuli on various physiological events in *A. thaliana* seedlings.

DISCUSSION

In the present study, we aimed to determine the effects of urban traffic noise on the seed germination and early growth of *A. thaliana* as a model for noise pollution-plant interaction. Seeds exposed to traffic noise did not exhibit altered germination compared to those grown in control conditions, indicating that traffic noise had no impact on seed germination. In seedling growth analysis, the results show that seedlings treated with traffic noise for 48 h exhibited a significant elongation of up to 39% compared to control,

Figure 2. Schematic diagram of the experimental setup. To prevent external noise interference, the traffic noise treatments were performed in a sound-proof facility. Experimental groups were continuously treated with traffic noise at an average of 75 dB for 48 h. For the control group, the seeds were exposed to background white noise outside the sound-proof facility at an average of 10 dB. Both the sound-proof facility and the outside area were maintained at a constant temperature (22 °C) and humidity (60-70%).

suggesting a stimulatory effect of traffic noise on the early growth of seedlings. In transcriptomic RNA-seq analysis, we identified 690 DEGs in seedlings in response to noise exposure, which are related to stress response, auxin signaling pathway, growth regulation, autophagy, TCA cycle, protein transport, and hydrogen ion transport. Taken together, increased seedling length and altered gene expression profiles in traffic noise-treated *A. thaliana* with no sign of accelerated germination rate partially support the hypothesis that plants exposed to traffic noise pollution exhibit increased seed germination rate and enhanced early growth.

Further investigations will be needed to examine whether this phenotypic change represents a physiological response perturbed by artificial traffic noise or reflects the fitness response of *A. thaliana* to noise stress. To address this challenging question, we should first uncover fundamental principles of sound-sensing mechanisms in plants. Integrative analysis of the functional relationships between noise-evoked signaling networks and plant growth response pathways will ultimately allow us to understand the modes of noisemediated signaling actions in the control of physiological alterations in plants.

Different types of sound (e.g., varying frequency) and treatment conditions (e.g., varying intensity or time) have various effects on different plants (14). In a previous study, traffic noise collected from high-density areas in Iran was used to treat two urban plant species, *Tagetes patula* and *Salvia splendens*, for 16 h per day for 15 days (9). Two-monthold plants subjected to this long-term treatment showed 27% and 17% reductions in fresh weight of *S. splendens* and *T. patula*, respectively, suggesting the negative impact of noise on the growth of those urban plants. In contrast, our data indicate that traffic noise led to significantly increased growth of *A. thaliana* seedlings, suggesting the stimulatory effects of traffic noise pollution on the early growth of *A. thaliana.* By testing the effects of long-term noise treatment on mature *A. thaliana* plants, we can further determine whether noise pollution differentially changes growth response depending on the developmental stage. In addition, the difference in length of noise exposure time compared to previous studies could have played a major part in the growth of *A. thaliana* in our study. It will also be critical to investigate whether anatomical and physiological traits in different plant species from *A. thaliana* could lead to contrasting responses to traffic noise.

The increased seedling growth rate observed in the present study is consistent with the earlier reported promoted growth in *A. thaliana* under defined frequency sound treatments. For example, the exposure to the sound treatment condition (200 Hz, 80 dB continuously for 2 weeks) increased the root growth of *A. thaliana* seedlings with positive phototropism (15). In another study, *A. thaliana* seeds showed significantly longer root growth than the control group under two experimental conditions (single 100 kHz or dual $100 + 9$ kHz, 80 dB for 15 h per day for 3 days) (16). However, we should be cautious about generalizing these positive effects of sound on plant growth. A previous study with paddy rice showed positive effects on germination rate and growth of the sound treatment condition (400 Hz, 106 dB for 60 min per day for 2 days) (17). However, growth was severely inhibited by frequencies higher than 4 kHz and intensities greater than 111 dB (17). In addition, under the sound treatment condition (1 kHz, 100 dB for 60 min per day for 20 days), soluble protein content increased in *Actinidia chinensis*, but higher sound levels led to decreased content (18). Therefore, it is more likely that sound, including traffic noise pollution, elicits complex changes in plant growth depending on treatment conditions and species. Systematic investigations of additional experimental conditions (e.g., high loudness, different urban noise types, frequencies, noise exposure time) using *A. thaliana* will provide more comprehensive information that can be applied to interpret the physiological response of other plant species. More studies will be needed to examine whether *A. thaliana* and similar plants sharing the anatomical and ecological features

Figure 3: Effects of traffic noise exposure on the seed germination of *Arabidopsis thaliana.* **(A) Seeds were exposed to either traffic** noise or control white noise for 48 h. (B) Seed germination percentages (%) are presented as mean ± standard deviation (*n* = 3). Student's *t* test, n.s., not significant. Seed germination assays were performed independently three times, and similar results were obtained. Data are from one representative experiment out of three independent experiments.

elicit the same biological reactions to noise stimuli.

Further studies are required to uncover the molecular mechanisms underlying the traffic noise-induced stimulation of seedling growth. Based on our pathway analysis of the differentially expressed genes, a major pathway that was strongly influenced by traffic noise exposure was the stress pathway. Top-ranked genes that were robustly induced include heat shock protein (HSP) genes such as *HSP17.6II*, *AT1G53540 (HSP17.6C),* and *HSP17.4*. Heat stress response factors (HSFs) and HSPs are central players in the heat stress pathway and related biological events such as growth promotion and thermotolerance (19). Therefore, our results suggest that mechanical energy from the traffic noise is absorbed by the seedling, thereby leading to local heat production and the corresponding protective heat stress response. Further experiments to measure local temperature changes in noise-exposed seedlings will be needed to test whether noise-induced heat stress response is a key event in driving stress response and plant growth. Since HSPs are induced as molecular chaperones by a variety of stresses, such as dehydration, salinity, reactive oxidative species, heavy metals, and high-intensity irradiations, we should also consider the possibility that noise-dependent HSP induction could be mediated by other types of stress independent of heat (20, 21).

Vibrational energy from sound can mechanically stimulate cells, as touch produces a physiological response. Thigmomorphogenetic responses that are triggered by physical touches also include stress resistance (22). Two major mechanosensitive genes are TOUCH genes (TCH) that mainly encode calmodulins or calmodulin-like proteins and xyloglucan endo-transglycosylase/hydrolase (XTH) (23, 24). In our study, TCH and XTH genes were also upregulated by traffic noise treatment, which strongly suggests that noise and touch induce similar mechanical force-dependent gene expression changes.

Traffic noise also significantly stimulated the auxin signaling pathway. In the growth regulation pathway, expression of many small auxin upregulated RNA (SAUR) genes was indeed markedly elevated after traffic noise exposure. Auxin is one of the most important plant hormones essential for promoting cellular growth and increasing the rate of cell division (25). The SAUR family is a group of auxinresponsive genes conserved in many higher plant species, highlighting the importance of these genes in the regulation of auxin-induced dynamic and adaptive growth in response to noise (26). Hence, enhanced seedling growth can partly be explained by the activation of auxin-dependent signaling events.

Our transcriptomic analysis revealed unique changes in other biological pathways. Some genes in the TCA cycle and proton transport were downregulated by noise exposure, which indicates metabolic changes in noise-stimulated seedlings. One of the most dramatic reductions in expression by traffic noise was observed in the MLS gene, which encodes malate synthase, a key enzyme for the glyoxylate cycle (27). In germinating seeds, the glyoxylate cycle uses acetate from lipid breakdown and converts it to four-carbon gluconeogenic substrates to support seedling growth (28). Malate synthase-null seedlings exhibited enhanced growth, probably owing to accumulated glyoxylate serving as a substrate for biosynthesis (e.g., serine and glycerate) (29). By measuring glyoxylates and other energy metabolites, we can further validate the contribution of altered glyoxylate and energy metabolism to seedling outgrowth in response to traffic noise treatment.

The growth-defense trade-off is critical for plant homeostasis. Therefore, enhanced seedling growth in our study may have resulted in reduced immune response. From an in-depth analysis of DEGs, we identified significantly reduced expression of the RPS2 gene, which encodes a protein representative of the nucleotide-binding site-leucinerich repeat (NBS-LRR) class of plant resistance proteins (30). RPS2 specifically recognizes *Pseudomonas syringae* strains expressing the avrRpt2 gene and initiates defense responses to bacteria carrying avrRpt2 (31). In addition, noise treatment led to marked upregulation of the homolog of BEE2 interacting with IBH 1 (HBI1), which promotes growth but suppresses immunity (32). Furthermore, traffic noise-exposed seedlings showed elevated expression of constitutive expresser of pathogenesis-related genes-5 (CPR5). CPR5 is known as a master regulator of growth and immunity, as cpr5-null mutants showed compromised growth but increased resistance against pathogens (33). Taken all together, while seedling outgrowth was promoted, changes in gene expression (e.g., RPS2, HBI1, and CPR5) in traffic noise-exposed seedlings

Figure 4. Effects of traffic noise exposure on the seedling growth of *A. thaliana.* (A) Seeds were vertically grown under the treatment of either traffic noise or control white noise for 48 h. (B) Length of seedlings from traffic noise (*n* = 66) or control white noise (*n* = 59) groups. Student's *t* test, ****p*<0.001. Seed length measurement assays were performed independently twice, and similar results were obtained. Data are from one representative experiment out of two independent experiments. Scale bar, 5 mm.

may elicit a reduced immune response. More experiments are needed to validate the possibility that traffic noise negatively impacts immune defense upon pathogen infection.

Our study has a limitation associated with the application of background noise to control group seeds. Due to limited technical resources, control seeds were placed outside the sound-proof facility that housed the seeds exposed to traffic noise. Notwithstanding, the main observations drawn from this study are likely biologically relevant, as plants in nature have no chance to grow in a completely soundless condition, and the experimental groups and control groups were protected from light and grown under the same conditions (e.g., temperature, humidity). Further analysis of A. thaliana seeds in the same sound-proof facility in response to background noise or no sound treatment will be helpful to validate and strengthen our findings and related implications.

The results of this study demonstrate the stimulatory role of urban traffic noise on *A. thaliana* seedling growth, which partially supports our initial hypothesis that traffic noise could stimulate both seed germination and seedling growth. Accompanying changes in gene expression suggest that traffic noise reprograms various cellular processes related to stress, auxin signaling, metabolism, and immunity. More biological information using single-cell transcriptomics and other omics technology (e.g., proteomics, metabolomics) will be needed to better understand the integrative, complex actions of traffic noise on various plants. It will also be important to systematically investigate the biological effects of different frequencies or variable sound intensities on diverse plants. As noise pollution emerges as a serious environmental factor, its direct effects on plant growth and life cycle can make considerable ecological changes by indirectly affecting plant-animal (e.g., insects) relationships. Ultimately, further research on the biological impacts of noise on plants can make significant contributions to developing technologies to protect ecological health from the detrimental effects of environmental noise pollution.

MATERIALS AND METHODS Plant Material and Growth Condition

Wild-type seeds of *A. thaliana* obtained from the KAIST (Korea Advanced Institute of Science & Technology) plant biology laboratory were seeded on half-strength Murashige and Skoog (MS)-agar plates (MS 2.2 g, 2-morpholinoethanesulfonic acid 0.5 g, phytoAgar 8 g, distilled water 1L, pH 5.7) (Duchefa #M0222.0050, #M1503.0250, #P1003.1000). Seeds on the plates were irradiated with a pulse of far-red light (724 nm, 5 μ mol m⁻² s⁻¹) for 5 min, followed by a pulse of red light (656 nm, 25 µmol m^{-2} s⁻¹) for 5 min. After that, plates were sealed with 3M micropore tape to prevent dehydration and wrapped in aluminum foil to block light. No further light treatment was applied for both noise exposure and germination assays.

Exposure of Seeds to Urban Traffic Noise

To collect urban traffic noise, sound recording was performed in Gangnam, Seoul area with high traffic density by using iPhone 13 (Apple, Inc). Patterns of noise frequency and waveforms from three different source types (bus & motorcycle, ambulance, subway train) were analyzed by the Sound Analyzer 1.12.2 application (**Figure 1**). A total of 10 min recording of traffic noise was structured in the sequence of buses and motorcycles, succeeded by ambulances, and concluding with subway stations (**Figure 1A**). Sound analysis was also performed on the background white noise collected outside the sound-proof facility for control groups (**Figure 1B**). For the experimental groups, the traffic noise treatments were performed in a sound-proof facility by using the inote BT-V3 speaker system (Fusionfnc, Inc). Experimental groups were continuously treated with traffic noise at an average of 75 dB with a range of 57 to 90 dB for 48 h. Noise volume was measured using the Sound Meter application (Splend Apps, Inc). A sound-proof facility was custom-made to block external noise interference using polyester fiber acoustic panels and wood wool acoustic boards composed of wood fiber and inorganic cement binders (Rachmaninov Co. Korea). The facility measures 4 m in length, 3 m in width, and 2 m in height. As for the untreated control groups, the seeds were placed outside

Figure 5. Volcano plot of the differentially expressed genes (DEGs) due to 48 h of noise exposure. The green dots indicate genes that reduced 1.5-fold below, and the red dots indicate genes that increased 1.5-fold above the control. The dotted black line shows the significance threshold ($p = 0.05$) with points above the line having $p < 0.05$ and points below the line having $p > 0.05$. Among significantly altered genes, several genes of top ranked and interesting pathways are indicated.

the sound-proof facility and exposed to background white noise at an average of 10 dB with a range of 5 to 15 dB. Both the soundproof facility and the outside area were maintained at a constant temperature (22 °C) and humidity (60-70%).

Germination Assay

The experimental group of *A. thaliana* seeds was subjected to traffic noise at an average of 75 dB. *A. thaliana* seeds were placed in a sound-proof facility to prevent the transfer of vibrations between samples during the sound wave treatments. After 48 h of traffic noise exposure, the sound was turned off, and the seed germination was determined by counting all seeds harboring at least one radicle protrusion. The control group was exposed **https://doi.org/10.59720/23-199**

to a background noise of approximately 10 dB on average. Germination assays were independently performed three times.

Seedling Length Measurement

For seedling growth assays, seeds were stratified by immersion in water and incubation under dark conditions at 4°C for 3 days, followed by red pulses (10 min, 25 μ mol m⁻² s⁻¹). The light irradiation chamber was maintained at 22 °C. Seeds were vertically grown and exposed to traffic noise or control white noise for 48 h. Seedling growth was assessed by using ImageJ software. The length of seedlings was measured from the beginning of the hypocotyl to the tip of the roots. Seedling length measurement assays were independently performed twice.

RNA-seq Analysis & Gene Ontology (GO) Analysis

Three sets of seedling samples (8-10 seedlings per set) from 48 h noise exposure and control groups were collected and processed for RNA-Seq analysis. Total RNA was isolated using Trizol reagent (Invitrogen, Waltham, MA, USA). RNA quality was assessed with an Agilent 2100 bioanalyzer using the RNA 6000 Nano Chip (Agilent Technologies, Santa Clara, CA, USA). RNA quantification was performed using an ND-2000 Spectrophotometer (Thermo Fisher Scientific, Inc., Waltham, MA, USA). QuantSeq 3′ mRNA-Seq was performed by Ebiogen (Ebiogen, Inc., Seoul, South Korea). The libraries for control and traffic nose-treated seedlings (*n* = 3 each) were constructed using the QuantSeq 3′ mRNA-Seq Library Prep Kit (Lexogen GmbH, Vienna, Austria) according to the manufacturer's instructions. High-throughput sequencing was performed as single-end 75 bp sequencing using NextSeq 500 (Illumina, Inc., San Diego, CA, USA). Sequenced reads were trimmed for adaptor sequence, masked for low-complexity or low-quality sequence, and then mapped to TAIR10.1 (NCBI) whole genome using Bowtie2. Differentially expressed genes were identified through counts derived from unique and multiple alignments, utilizing Bedtools coverage. The read count data underwent processing via the quantile normalization method using EdgeR within R (R Development Core Team, 2016) through Bioconductor.

Classification of DEGs based on biological processes was conducted using the Database for Annotation Visualization and Integrated Discovery (DAVID) software (34). Data mining and

Figure 6. Gene Ontology (GO) enrichment analysis of DEGs between the noise-treated and control groups. (A) Gene classification was based on searches conducted by the DAVID software tool (34). Count of upregulated (red bars) and downregulated (blue bars) genes are represented. The black dotted line of the graphs represents the −log₁₀(*p* value). (B) The x-axis represents the fold enrichment of the GO terms. The color intensity and the size of the bubbles increase with the −log₁₀(*p* value).

graphic visualization were performed using ExDEGA (Ebiogen Inc., Seoul, South Korea). Sequencing data were deposited in the NCBI GEO (accession number GSE240794).

Statistical Analysis

Differences between averages of germinated seed numbers and measured seedling length in control and noise exposure groups were analyzed using a two-tailed Student's *t*-test. Statistical significance was set at *p* < 0.05. Statistical calculations were performed in Microsoft Excel (**Figure 3**) and GraphPad Prism 7 software (**Figure 4**).

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APPENDIX

Table 1. List of DEGs in *Arabidopsis thaliana* **seedlings exposed to traffic noise**

Table 2. GO enrichment analysis

The threshold of minimum gene counts belonging to an annotation term is expressed as count (%). The false discovery rate (FDR) is the ratio of the number of false positive classifications to the total number of positive classifications.

