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Transcriptional regulation of heat stress response in plants

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DOCTORAL (PhD) DISSERTATION

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1. INTRODUCTION

Temperature stress is one of the major abiotic stresses that negatively affects the survival and reproductive fitness of living organisms worldwide. Cells exposed to heat stress activate a series of biochemical and physiological changes that result in a reprioritisation of cellular physiology to aid survival; these processes are collectively called heat stress response (HSR). A central layer of HSR is the transcriptional regulation. While significant efforts have been put into understanding the activity of specific transcription factors that coordinate HSR, and the differentially expressed gene/transcript sets they regulate, *the understanding of general transcriptional machinery regulation and its specific functions during HSR remains scarce*.

In the present work, we aimed to unravel the regulation of RNA polymerase II (RNAPII) core machinery and its associated co-factors. To achieve this, we conducted a heat stress phenotyping screen on selected RNAPII and co-factor mutants and identified the Transcription Factor IIS (TFIIS) as a key player of HSR.

TFIIS is a biochemically and structurally conserved transcription elongation cofactor of RNAPII. While it has been extensively studied in yeast and metazoan systems, *its regulation and functions in plants, specifically during heat stress responses, have not been explored*. Therefore, we characterised the conservation, regulation, functions and molecular mechanisms of TFIIS in plants and its requirements under high-temperature conditions. We demonstrated that TFIIS *enable plants to conduct a timely, qualitative and quantitative reprogramming of the transcriptome*, which is vital for heat stress survival. Besides, we have *described* TFIIS *molecular actions on transcriptional fidelity processes at the single-nucleotide level* for the first time in plants.

In summary, we believe that our study on transcriptional elongation and the roles of TFIIS will contribute to a basic understanding of HSR and will accelerate breeding of heat-tolerant crops crucial for minimising the threats posed by global warming and climate change.

2. OBJECTIVES

In the present work, we aimed to study the functions of RNAPII, especially focusing on the transcriptional elongation cycle and the roles of TFIIS elongation cofactor during HSR in the model plant *Arabidopsis thaliana* and *Hordeum vulgare* monocot crop plant species.

Specific sub-tasks of the work are:

a) Understanding the regulation of transcriptional elongation during HSR.
b) Validation of the molecular changes coordinated by TFIIS activity, based on RNAseq data showing transcriptional reprogramming from a developmental transcriptome to a heat-stress transcriptome.
c) Understanding the genetic interaction between transcriptional and post-transcriptional RNA quality control pathways.
d) Analyse the consequences of transcriptional errors in plants under ambient and high-temperature conditions.

3. MATERIALS AND METHODS

3.1. Plant materials

Experiments were performed on *Arabidopsis thaliana, Brassica napus, Hordeum vulgare* and *Chlamydomonas reinhardtii*, as described below: *A thaliana* seeds were bleach sterilized for 15 minutes and then plated on Murashige and Skoog (Duchefa M0222, https://www.duchefa-biochemie.com) medium agar plates (0.5× Murashige and Skoog salts, 1% agar, pH 5.7). Plants were routinely grown in a Sanyo MLR-350 growth cabinet under cool white light at 21°C long-day condition (16 h light/8 h dark). *Chlamydomonas reinhardtii* (cc-4533) was grown on Murashige and Skoog medium agar plates. *Brassica napus* (RV31) and *Hordeum vulgare* (Golden Promise) were grown on soil. Mutant seeds *of A. thaliana* plants were ordered from NASC (https://arabidopsis.info): *tfIIs-1* (SALK_056755), *tfIIs-2* (SALK_027259), *upf1-5* (SALK_081178), *upf3-1* (SALK_025175, seeds were donated by K. Riha). Double mutants were generated by crossing these single mutants. For mutant complementation the *pTFIIS::GSy-TFIIS;tfIIs-1* constructs were generated by Csorba lab. All the plant materials (*wild type (Col-0), tfIIs-1, upf1-5, upf3-1 and tfIIs-1;upf1-5*) were grown at 21°, except for *tfIIs-1;upf3-1,* which is sterile at this temperature, therefore grown at 25°.

3.2. Genotyping

For genotyping, genomic DNA was extracted from 30 mg fresh plant material using 100 ul of Extraction buffer (E7526, Sigma-Aldrich) along with a stainless-steel bead (3 mm, Qiagen Sciences). Homogenisation was done using a mixer mill (Bullet Blender Storm Pro, Next Advance,) at speed grade 8 for 1 min; subsequently, the mixture was incubated at 95 °C for 12 min in a dry heat block and placed on ice for 1 min. Finally, 100 ul of Dilution solution (D5688, Sigma- Aldrich) was added, and aliquoted the supernatant after centrifugation (13000
rpm) for 10 mins. Genotyping PCR was done using DNA Taq polymerase (NEB, M0273S) based on the manufacturer's instructions.

3.3. Stress treatments

We performed 4 types of heat treatments, namely basal thermotolerance (BT), short-acquired thermotolerance (SAT), long-acquired thermotolerance (LAT) and thermotolerance to moderately high temperatures (TMHT). For BT, seedlings were grown on 0.5xMS, 1% agar plates; naïve 7-day-old seedlings were exposed to 45°C HS in a water bath for 10–30 min. For SAT, seedlings

were pre-grown for 6 days; following these seedlings were heat-treated by a sublethal temperature (37°C for 1 h, acclimation phase), then placed back at 21°C for 2 h for recovery; After the recovery period, the seedlings were challenged by lethal stress temperatures (45°C for 1–3 h, lethal HS). For LAT, 5-day-old seedlings were first acclimated at 37°C for 1h and then recovered for 2 days at 21°C. Lethal HS treatment was applied afterwards in a water bath (45°C, for 20–100 min). For TMHT, 7-day-old seedlings were placed in a growing cabinet pre-heated to 37°C and kept for 1–5 days, at long-day conditions (16 h light/8 h dark). All treatments were started at midday (Zeitgeber time, ZT8). Plants were cooled back to 21°C following each treatment.

3.3.1. Heat stress treatments:

For RNA and/or protein sample collections, plant materials were taken immediately after each treatment (after 1 hour, 1h, 4h or one day, 1d, alongside non-treated controls, NT); for recovery samples, materials were collected 2 days following TMHT treatment at ZT8).
For heat stress phenotyping, seedlings were kept and grown on plates for 1 or 2 weeks back to at 21◦C, LD conditions, and then photographed.
For analysing TFIIS expression in *C reinhardtii* green algae: agar plates containing the C reinhardtii culture were incubated in a water bath for 1 h at 37 °C.
For analysing TFIIS expression in *B napus* and *H vulgare* plants: leaf discs (of 1 cm diameter) of 1-week-old soil-grown plants were placed into hydroponic culture (0.5× MS) and heat stress treated in a water bath for 1 h at 37 °C.

3.3.2. Salt stress treatments:

We prepared salt (0,5xMS) media agar plates containing NaCl concentrations ranging from 0 mM to 200 mM. Seeds were surface sterilized and placed on the agar surface, grown for 3 weeks and then photographed.

3.4. RNA extraction and qRT-PCR

Total RNA was isolated from ~30 mg seedlings in 700 µl extraction buffer (0.1 M glycine–NaOH, pH 9.0, 100 mM NaCl, 10 mM EDTA, 2% SDS) using the phenol–chloroform (pH 4.3) method; the RNA was precipitated in ethanol and resuspended in sterile water. DNase treatment was performed on 5 µg of total RNA (Ambion AM2222, www.thermofisher.com). One microgram of DNase-treated RNA and random primer was used for the first-strand cDNA reaction (NEB, E6300S, www.neb.com). qPCRs were done using the qPCR

Master Mix (NEB, M3003S, www.neb.com), in a Light Cycler 96 real-time PCR machine (Roche). At least three independent biological replicas were analysed in each experiment. qRT-PCR primer sequences.

3.5. RNA transcriptome analysis

The HS treatment, RNA extractions for RNA transcriptome and alternative splicing analysis from wild type (Col-0) and *tfIIs-1* mutant at non-treated, one hour (1h) and 1 day was done as described above. Bioinformatic work was done by HM Szaker and published previously.

3.6. Protein extraction and western blotting

For protein isolation, we took ~30mg of 7d old seedlings (non-treated, NT; heat-treated for 1h, 1d and rec), homogenised them in 100 ul of extraction buffer (150 mM Tris–HCl, pH 7.5, 6 M urea, 2% SDS and 5% -mercapto-ethanol) and denatured the extracts at 95˚C for 5 mins. The cell debris was removed by centrifugation at 13000 rpm at 4˚C for 10 mins. The supernatants were resolved on 10% SDS-PAGE, transferred to Hybond PVDF membranes (GE Healthcare), and subjected to western blot analysis. Antibodies used for detection: anti-sHSP-CI antibody (AS07 254, Agrisera), anti-HSP90-1 antibody (AS08 346, Agrisera), and anti-HSP101 (AS07 253, Agrisera); as secondary antibody we used monoclonal HRP-conjugated anti-rabbit (A6154, Sigma-Aldrich). The proteins were visualised by chemiluminescence (ECL kit; GE Healthcare), and quantified by Image Lab 5.1 (Bio-Rad). For quantifications, protein signals have been normalised to Rubisco large subunit (RbcL).

3.7. Protein aggregate purification and detection

Protein aggregates were purified as described before (*76*). Briefly, 0.1 g fresh seedling material of non-treated and heat-treated (1d) in 2.4 ml of isolation buffer [25 mM HEPES, pH 7.5, 200 mM NaCl, 0.5 mM, Na_2EDTA, 0.1% (v/v), Triton X-100, 5 mM ε-amino-N-caproic acid, 1 mM benzamidine] by using a mortar and pestle and then a Cole-Parmer PTFE glass tissue for the grinder. The soluble and insoluble fractions were separated from 2 ml of total extract by centrifugation at 14000 rpm for 15 min at 4˚C. The soluble fraction was denatured by adding 0.5 volume of 2x SDS-PAGE buffer and heating for 5 min at 95℃. The insoluble pellet was washed six times repeatedly by resuspension in the isolation buffer containing 0.1 g of quartz sand (Sigma-Aldrich) and vortex. Later, the insoluble pellet was resuspended in 400 ml 2× SDS-PAGE sample buffer and clarified by centrifugation at 1500 rpm for 1

min (insoluble fraction). Samples were separated by SDS-PAGE and stained with Coomassie Blue Staining method. The whole lanes of insoluble fractions have been quantified by Image Lab 5.1 (Bio-Rad) and ratios to Rubisco large subunit (RbcL) stain free signals were calculated.

3.8. CirSeq library preparation and bioinformatic analysis

For Circle-sequencing (CirSeq) sample preparation, we have used non-treated (NT) and heat-treated (1d) samples of wild type (Col-0), *tfIIs-1* or *upf1-5* single and double mutant plants. CirSeq libraries' preparation and bioinformatic analysis were done as described previously in five biological replicates. Amplicon libraries were performed by Novogene Ltd. Sequencing service. Bioinformatic analysis was done by HM Szaker.

4. RESULTS AND DISCUSSION

4.1. TFIIS transcription elongation cofactor is needed for proper development and stress response

4.1.1. TFIIS is an RNAPII cofactor specifically needed for heat adaptation

The TFIIS protein, a transcription factor for RNA polymerase II, is identified as a vital and specific component of the heat shock response (HSR) in *A. thaliana*. Mutant plants lacking TFIIS (*tfIIs-1*) are highly sensitive to various heat stress regimes but not to salt stress. These mutants die under prolonged moderate heat (37°C) and show significantly reduced seed production, demonstrating that TFIIS is essential for survival and reproductive fitness under heat stress throughout the plant's lifecycle.

4.1.2. TFIIS transcriptional regulation in response to heat stress

4.1.2.1. *Cis* and *trans* factors of *TFIIS* locus regulation

The expression of the TFIIS gene is directly activated by heat shock transcription factors (HsfA1 family), which bind to heat shock elements in its promoter. The TFIIS protein itself is then required for its own efficient transcription, creating a positive feedback loop that ensures rapid accumulation of both TFIIS mRNA and protein during heat stress. This was confirmed by complementation assays, which showed that reintroducing a functional TFIIS gene into the mutant rescues its heat-sensitive phenotype.

4.1.2.2. TFIIS heat induction is conserved in the plant kingdom

The role of TFIIS in the heat shock response is highly conserved across plant species, including the green alga *Chlamydomonas reinhardtii*, the *A. thaliana* close relative, the dicot crop *Brassica napus*, and the monocot crop *Hordeum vulgare*. Experiments confirmed that their mRNA accumulates during heat stress. In barley, CRISPR mutants demonstrated that TFIIS is essential for heat tolerance, seed production, and germination, confirming its vital and conserved role in coordinating the heat shock response in both monocots and dicots.

4.1.3. TFIIS is needed for transcriptional reprogramming during HSR

4.1.3.1. TFIIS affects qualitative and quantitative aspects of HS transcriptome

TFIIS is essential for both the qualitative and quantitative reprogramming of the transcriptome during heat stress. Its absence causes delayed and inefficient expression of heat shock response genes (like HSFs and HSPs). Specifically, early expression of ATP-dependent chaperones is reduced, while ATP-independent chaperones accumulate excessively later on. This demonstrates that TFIIS ensures the precise timing and level of gene expression needed for an effective heat shock response.

4.1.3.2. Absence of TFIIS leads to enhanced proteotoxicity during HSR

The absence of TFIIS leads to increased cellular proteotoxicity. This is evidenced by a significant accumulation of ubiquitinated and sumoylated proteins, as well as a higher ratio of insoluble to soluble protein aggregates in the *tfIIs-1* mutant, both under normal conditions and during heat stress. This confirms that the transcriptional defects caused by the TFIIS mutation result in ineffective protein quality control.

Based on these findings, we proposed a model of TFIIS actions in plants. When TFIIS is present, RNAPII arrests are efficiently resolved under both normal and heat stress conditions. We postulate that elongation arrests could be less numerous under normal conditions, so the alternative rescue pathways may act efficiently to compensate for the absence of TFIIS. Under HS conditions, however, either (i) elongation arrests may be more frequent, (ii) alternative pathways less effective or (iii) a combination of these; therefore, the absence of TFIIS becomes vital (Fig 1).

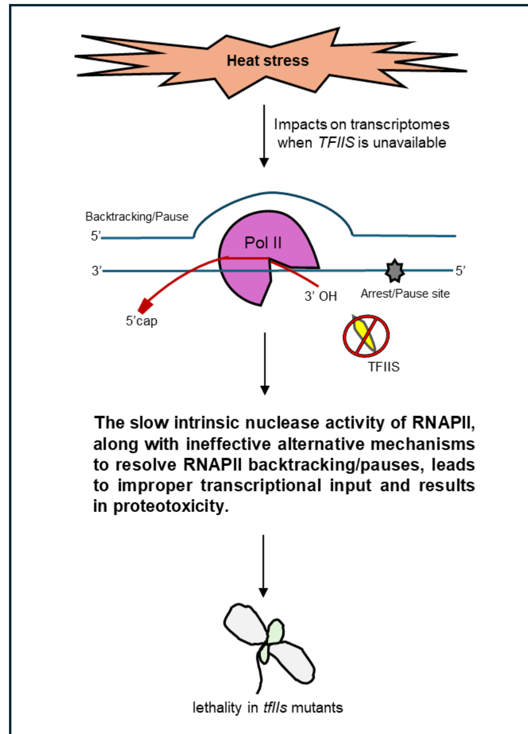

Figure 1: Absence of TFIIS protein results in inefficient transcriptional reprogramming and proteotoxicity under elevated temperature conditions. *Under HS, the likelihood of backtracking or pauses in RNAPII may be increased. Inefficient arrest resolutions in the absence of TFIIS cause qualitative and quantitative alterations of the transcriptome, indirectly resulting in proteotoxic stress that finally contributes to plant lethality (see text for details).*

4.2. Mechanistic actions of TFIIS roles during development and HSR

4.2.1. Elevated temperature promotes the accumulation of transcription fidelity errors

To study TFIIS's role as a transcription fidelity factor in plants, researchers combined the *tfIIs-1* mutant with mutants of the Nonsense-Mediated Decay (NMD) pathway (*upf1-5, upf3-1*). This was done because transcription errors (like frameshifts creating premature stop codons) are normally masked by NMD, which degrades the faulty mRNAs. By disabling this downstream RNA quality control, they aimed to stabilise the erroneous transcripts and directly reveal the transcription errors caused by the absence of TFIIS.

4.2.2. Interaction between nuclear and cytoplasmic mRNA fidelity pathways

4.2.2.1. NMD is needed for HSR

The Nonsense-Mediated Decay (NMD) pathway is active and required for heat stress adaptation. This was demonstrated by showing that NMD mutant lines (*upf1-5* and *upf3-1*) are sensitive to heat stress (TMHT), indicating that NMD plays a vital role in surviving high temperatures.

4.2.2.2. TFIIS and NMD factors interact genetically

We have generated double mutant plants *tfIIs-1;upf1-5* and *tfIIs-1;upf3-1* to investigate the interaction between the TFIIS and NMD pathways. The double mutants exhibited severe developmental defects, including altered leaf morphology, stunted growth, and sterility, which were more pronounced than in the single mutants. The *tfIIs-1;upf3-1* phenotype was linked to activated autoimmunity, as shown by elevated *PR1* gene expression. When exposed to heat stress (TMHT), the double mutants were significantly more sensitive than both single mutants, demonstrating that the TFIIS and NMD pathways function synergistically and are both essential for heat stress survival.

4.2.2.3. The impact of combined TFIIS and NMD absence on proteostasis

The combined loss of TFIIS and NMD leads to additive proteotoxicity during heat stress. This is evidenced by a significant, additive increase in insoluble protein aggregates and the persistent over-accumulation of protective sHSP chaperones in the double mutants (*tfIIs-1;upf1-5* and *tfIIs-1;upf3-1*) compared to single mutants. These findings suggest that the TFIIS and NMD pathways function through distinct but complementary mechanisms to maintain proteostasis by preventing the accumulation of faulty proteins. The heightened proteotoxicity in the double mutants is likely caused by error-containing mRNAs that escape quality control, leading to the production of defective proteins.

4.2.3. TFIIS is a fidelity factor

Besides the retarded and qualitatively altered expression of HS transcripts, faulty transcription may be another reason for imbalanced cellular proteostasis. As TFIIS was previously described as a transcriptional fidelity factor, we aimed to analyse transcriptional fidelity in its absence and in plants exposed to high temperature stress.

4.2.3.1. TFIIS is needed under imbalanced nucleotide concentrations

To test the importance of transcriptional accuracy, plants were treated with Mycophenolic Acid (MPA), a drug that induces transcription errors by reducing GTP levels. The *tfIIs-1* mutant was significantly more sensitive to MPA than wild-type plants, showing severe growth defects and death. This demonstrates that the error-correcting function of the TFIIS elongation factor is crucial for survival when transcription fidelity is compromised.

4.2.3.2. Circular Sequencing Methodology

To analyse the transcriptional fidelity impact at the nucleotide level as a potential underlying cause of HS and nucleotide imbalance sensitivity of *TFIIS* and *UPF* single or double mutants, we performed Circle-sequencing transcriptome analysis (CirSeq). Notably, CirSeq can differentiate between technical and *bona fide* biological mRNA errors, which is not enabled by a conventional RNAseq analysis. For CirSeq, total RNA was extracted from *A. thaliana* plants, then polyadenylated RNA was purified, fragmented, and circularised, resulting in concatemers through cDNA synthesis. Library construction and high-throughput sequencing, combined with bioinformatic analysis, enable differentiation of errors originating from reverse transcription (RT), library preparation, PCR amplification, or sequencing errors that cannot be filtered out in conventional RNA sequencing.

4.2.3.3. The error landscape of *Arabidopsis thaliana*

CirSeq analysis directly demonstrates that TFIIS is a crucial transcription fidelity factor in plants. The absence of TFIIS (*tfIIs-1*) caused a significant 2 to 3.4-fold increase in the RNA polymerase II error rate under normal and heat stress conditions, respectively. Heat stress itself compromises transcriptional fidelity. While the NMD mutants (*upf1-5*) alone did not alter the overall error rate, its combination with *tfIIs-1* revealed a potential specialised role for NMD in controlling insertions. TFIIS primarily prevents nucleotide substitutions and insertions, but not deletions. The spectrum of errors, particularly G→A substitutions, was influenced by both genotype and temperature.

4.3. A working model: Transcriptional fidelity regulation

Based on our findings, we propose a working model to explore the accuracy of transcription. In wt plants under ambient temperature, RNAPII may occasionally introduce errors; however, these are efficiently corrected by RNAPII intrinsic cleavage boosted by TFIIS activity. High temperatures do

not alter transcriptional error rates under these conditions, suggesting either the same error rate or an efficient transcriptional fidelity mechanism.

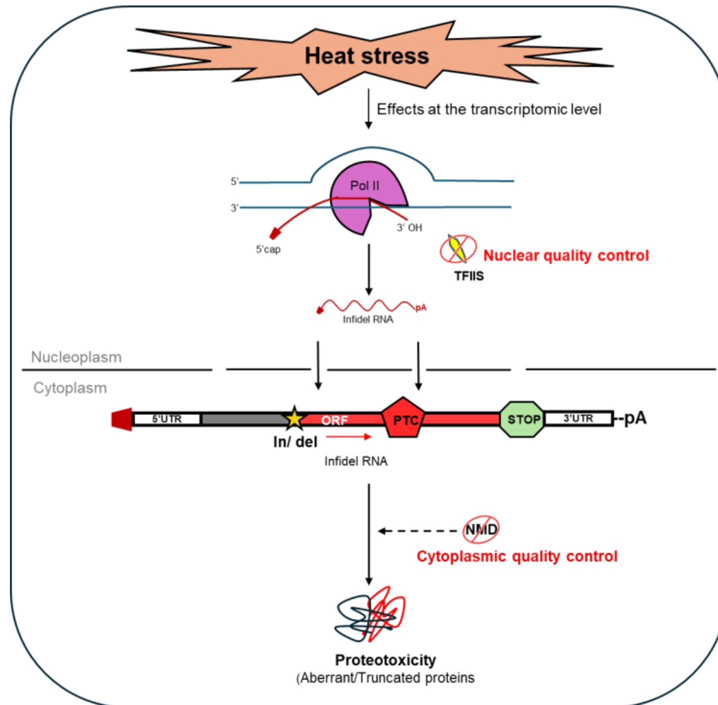

Figure 2: Transcriptional and post-transcriptional pathways interact to ensure transcript fidelity and prevent proteotoxicity, a working model. *During heat stress, the absence of TFIIS leads to the production of faulty/infidel RNAs causing truncated/ aberrant proteins that escape nuclear surveillance. The NMD pathway screens and eliminates the PTC-containing transcripts to prevent proteotoxic stress (see text for details).*

In the absence of TFIIS (*tfIIs-1*), however, errors are accumulating under NT conditions, showing that TFIIS acts as a fidelity factor. Under elevated conditions, error rates are further increased, showing that high temperatures cause a decrease in RNAPII fidelity. Some of the errors (mainly those consisting of small insertion or deletion mutations of 1–2 nucleotides) are detected and eliminated by NMD; in this respect, NMD emerges as a secondary layer of fidelity safeguarding. In the absence of TFIIS or NMD, the erroneous RNA transcripts (infidel RNA) that enter the cytoplasm will take part in translation, and the aberrant/truncated proteins potentially disrupt cellular processes through proteotoxicity (Fig 2).

5. CONCLUSIONS AND RECOMMENDATIONS

In summary, through a heat-stress phenotyping screen, we identified TFIIS as a transcription factor involved in the heat stress response (HSR). We have unravelled that TFIIS, despite being negligible under optimal conditions, its presence is vital for efficient HS adaptation in *Arabidopsis thaliana* plants. We demonstrated that TFIIS is positively regulated by HSFs and is also self-regulated. TFIIS protein accumulation during HS is conserved across evolutionarily distant species, including the unicellular alga *Chlamydomonas reinhardtii*, dicot *Brassica napus* and monocot *Hordeum vulgare* plants.
By creating CRISPR mutants in barley, we have shown that TFIIS functionality is necessary for HS survival in barley as well. Additionally, we investigated the downstream molecular changes regulated through TFIIS activity. TFIIS facilitates efficient transcriptional reprogramming from a developmental program to HS program, during which the timely expression, properly spliced and matured heat-stress transcript production is ensured, which is all needed to enable HS survival.

Finally, to uncover the mechanistic basis of TFIIS transcriptional regulation, we examined its role as transcriptional fidelity in conjunction with the cytoplasmic NMD RNA quality control pathway. We have proved that TFIIS acts as a nuclear fidelity factor; besides we demonstrated that NMD acts as a second layer to eliminate indel-containing transcripts and has a role under HS. Consequently, TFIIS in combination with NMD preserves transcriptome quality and prevents proteotoxic stress.
As a future direction for this research, we aim to identify and study alternative pathways that may resolve the arrested RNAPII complex in the absence of TFIIS.

6. **NEW SCIENTIFIC RESULTS**

i. We identified TFIIS, an RNAPII elongation cofactor, to be needed for heat stress adaptation.

ii. We suggest that TFIIS regulation and functions are likely widely conserved in the plant kingdom, based on sequence conservation and expression analysis experiment in *C. reinhardtii, B. napus, A. thaliana, H. vulgare* plants.

iii. Based on RNA transcriptome analysis, we validated the qualitative and quantitative differences in the absence of TFIIS and uncovered the consequences of these alterations at the proteome level.

iv. We have shown that the NMD pathway is required for heat stress adaptation.

v. We demonstrated the interaction between nuclear co-transcriptional and cytoplasmic post-transcriptional RNA fidelity pathways.

vi. We have provided evidence that TFIIS is a fidelity factor under both ambient and high temperature conditions; besides, we have described the error landscape and temperature-dependence in *A. thaliana* for the first time.

A2: LIST OF SCIENTIFIC ACTIVITIES

A2.1. PRESENTATIONS RELATED TO THE THESIS

- **Verma R**, Szádeczky-Kardoss I, Szaker MH, Pettkó-Szandtner A, Silhavy D, Csorba T: The role of transcriptional elongation during heat stress adaptation in plants, XX Genetikai Műhelyek Magyarországon minikonferencia, Szeged, September 24, 2021.
- **Verma R**, Szádeczky-Kardoss I, Szaker MH, Zoltán T, Csorba T: Efficient transcriptional elongation is needed for heat stress adaptation of plants. ETABS 2022, e-Conference, 14-15 January, 2022. (Best Oral Presentation)
- **Verma R**, Szádeczky-Kardoss István, Szaker MH, Zoltán T, Csorba T. The roles and regulation of TFIIS elongation factor during heat stress response in plants. (Poster) 16th Microsymposium on RNA Biology, Vienna BioCenter, 06 – 08 April, 2022.
- **Verma R**, Szádeczky-Kardoss I, Szaker MH; Silhavy D, Csorba T. Regulation of transcriptional elongation during heat stress response. RNA Mini-symposium, Gödöllő, 2023 June 29. (Best Presenter)
- **Verma R**, Szádeczky-Kardoss I, Szaker MH, Silhavy D, Csorba T: Transcriptional dynamics during heat stress adaptation in plants. GBI Napok Gödöllő, 30 November 2023. (Third price).
- Ahmad I, **Verma R**, Szadeczky-Kardoss I, Szaker MH, Syed Abbas H, Csorba T. Decoding the Role of TFIIS in Plant Resilience to Heat Stress, GBI Napok, 10-11 December 2024, Gödöllő, Hungary.
- **Verma R**, Szaker MH, Szadeczky-Kardoss I, Syed Abbas H, Pettkó-Szandtner A, Silhavy D, Csorba T. Transcriptome fidelity aids development and heat stress response in Arabidopsis. XXIII Genetikai Műhelyek Magyarországon Conference, 06 September 2024, Szeged, Hungary.

A2.2. PUBLICATIONS RELATED TO THE THESIS

- Szádeczky-Kardoss I*, Szaker MH*, **Verma R**, Darkó E, Pettkó-Szandtner A, Silhavy D, Csorba T, Elongation factor TFIIS is essential for heat stress adaptation in plants, Nucleic Acids Research, Volume 50, Issue 4, 28 February 2022, Pages 1927–1950.
- Ahmad I, Kis A, **Verma R**, Szádeczky-Kardoss I, Szaker MH, Pettkó-Szandtner A, Silhavy D, Havelda Z, Csorba T (2024). TFIIS is required for reproductive development and thermal adaptation in barley. Plant Cell Reports,43, 260.
- **Verma R***, Szaker MH*, Szádeczky-Kardoss István, Nóra Gál, Syed HA, Éva Darkó, Pettkó-Szandtner A, Silhavy D, Csorba T. (2025). Transcription fidelity and alternative splicing control contribute to heat stress survival in Arabidopsis (Accepted in Plant Cell) (*authors contributed equally to the work)