Arabidopsis bHLH Ib Transcription Factor Regulates the Post-transcriptional Imprint of FIT

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Abstract

FIT is a key regulator of iron homeostasis in Arabidopsis that is induced by iron deficiency at the transcriptional level, but the underlying regulatory mechanism remains poorly understood. This study used Arabidopsis single and double overexpression plants of bHLH38 and FIT, as well as bHLH Ib quadruple mutants as experimental materials, conducted iron deficiency treatment experiments, and analyzed FIT transcription levels via quantitative PCR. The results showed: (1) Under iron-sufficient conditions, the transcription level of FIT in bHLH38 overexpression plants was significantly higher than that in wild-type plants; (2) In bHLH Ib quadruple mutants, the transcription level of FIT was not induced by iron deficiency; (3) Single overexpression of FIT could not activate the transcription of endogenous FIT, but double overexpression of bHLH38 and FIT could activate the transcription of endogenous FIT; (4) Under iron-deficient conditions, the transcription levels of FIT in all plants showed no significant difference compared to that in wild-type plants. Based on these results, this study proposes that bHLH Ib transcription factors are a necessary but not sufficient condition for iron deficiency-induced transcription of FIT.

Full Text

Preamble

bHLH Ib Transcription Factors Regulate the Transcription of FIT in Arabidopsis thaliana

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Abstract

FIT (FER-LIKE IRON DEFICIENCY-INDUCED TRANSCRIPTION FACTOR) is a key regulator of iron homeostasis in Arabidopsis thaliana that is transcriptionally upregulated under iron deficiency, yet the underlying regulatory mechanisms remain poorly understood. This study investigated FIT transcript levels in single and double overexpression lines of bHLH38 and FIT, as well as in a bHLH Ib quadruple mutant, under iron-deficient conditions. The results demonstrate: (1) Under iron-sufficient conditions, FIT transcription in bHLH38 overexpression plants was significantly higher than in wild-type plants; (2) In the bHLH Ib quadruple mutant, FIT transcription was not induced by iron deficiency; (3) Single overexpression of FIT could not activate endogenous FIT transcription, whereas dual overexpression of bHLH38 and FIT could; and (4) Under iron-deficient conditions, FIT transcript levels in all transgenic and mutant plants showed no significant difference from wild-type levels. Based on these findings, we conclude that bHLH Ib transcription factors are necessary but not sufficient for iron deficiency-induced upregulation of FIT.

Keywords: Iron, transcriptional regulation, iron deficiency response, iron homeostasis

Iron is an essential micronutrient for plant growth and development, serving as a cofactor for numerous enzymes involved in critical physiological processes including photosynthesis, respiration, chlorophyll biosynthesis, nitrogen fixation, and phytohormone synthesis (Hänsch & Mendel, 2009; Balk & Schaedler, 2014). Although iron is the fourth most abundant element in Earth' s crust, its bioavailability to plants is limited due to its propensity to oxidize and form precipitates, particularly in high-pH soils where iron solubility is extremely low (Guerinot & Yi, 1994). Iron deficiency commonly manifests as interveinal chlorosis in leaves and, because plants constitute a major dietary source of iron for humans, plant iron deficiency consequently impacts human health.

To acquire sufficient iron from soil, plants have evolved two distinct strategies: Strategy I in non-graminaceous plants and Strategy II in graminaceous species (Marschner & Romheld, 1986; Romheld & Marschner, 1986; Grillet & Schmidt, 2019). The model plant Arabidopsis thaliana employs Strategy I, which comprises three sequential steps: rhizosphere acidification, reduction of Fe^{3+} to Fe^{2+}, and iron uptake. In Arabidopsis, rhizosphere acidification is primarily mediated by AHA2 (Santi & Schmidt, 2009), followed by reduction of Fe^{3+} to Fe^{2+} by FRO2 (FERRIC REDUCTION OXIDASE 2) (Robinson et al., 1999), and subsequent transport into root cells via IRT1 (IRON-REGULATED TRANSPORTER 1) (Varotto et al., 2002; Vert et al., 2002). Graminaceous plants such as barley (Hordeum vulgare), maize (Zea mays), and rice (Oryza sativa) secrete high-affinity mugineic acids (phytosiderophores) to directly chelate Fe^{3+}

(Walker & Connolly, 2008; Morrissey & Guerinot, 2009). Recent studies have revealed that Arabidopsis also secretes iron-chelating compounds (Rodriguez-Celma & Schmidt, 2013; Fourcroy et al., 2014; Schmid et al., 2014; Siwinska et al., 2018; Tsai et al., 2018).

FIT functions as a critical regulator within the Strategy I mechanism, and its loss of function leads to reduced expression of IRT1 and FRO2 and severe iron deficiency symptoms (Vert et al., 2002; Colangelo & Guerinot, 2004; Jakoby et al., 2004; Yuan et al., 2005). FIT interacts with four members of the bHLH Ib subfamily (bHLH38, bHLH39, bHLH100, and bHLH101) to regulate iron deficiency responses (Yuan et al., 2008; Wang et al., 2013). All four genes are induced by iron deficiency, and their protein functions are redundant (Wang et al., 2013).

FIT also serves as a central hub connecting various phytohormone signals and intracellular signaling pathways with iron deficiency signaling. For instance, FIT protein stability is regulated by ethylene and nitric oxide (NO) (Garcia et al., 2010; Lingam et al., 2011; Meiser et al., 2011). The ethylene signaling transcription factors EIN3 (ETHYLENE INSENSITIVE) and EIL1 (ETHYLENE INSENSITIVE 3-LIKE1) interact with FIT and enhance its stability (Lingam et al., 2011). NO inhibits FIT protein degradation and promotes its stability under iron deficiency (Meiser et al., 2011). Additionally, NO regulates FIT transcription through GRF11 (GENERAL REGULATORY FACTOR 11) (Yang et al., 2013). Gibberellin acts as another positive regulator of iron deficiency responses, with recent studies showing that DELLA proteins, negative regulators of gibberellin signaling, interact with FIT and inhibit its function (Wild et al., 2016). Furthermore, jasmonic acid negatively regulates iron deficiency responses by inducing expression of bHLH IVa subfamily genes (bHLH18, bHLH19, bHLH20, and bHLH25), whose products interact with FIT and promote its degradation (Matsuoka et al., 2014; Cui et al., 2018).

As a key transcription factor in iron deficiency signaling, FIT transcription is itself induced by iron deficiency. Our recent work demonstrated that bHLH121 directly targets the FIT promoter and positively regulates its transcription (Lei et al., 2020). As a crucial regulator of iron homeostasis signaling, FIT transcription is induced under iron deficiency conditions. Previous studies have shown that FIT and bHLH Ib can influence FIT transcription (Wang et al., 2007; Naranjo-Arcos et al., 2017), but the underlying regulatory mechanisms remain unclear. This study focused on how the bHLH Ib member bHLH38 and the FIT transcription factor regulate FIT transcription levels. Specifically, we investigated whether bHLH38 overexpression can activate FIT transcription, whether FIT transcription remains responsive to iron deficiency in the bHLH Ib quadruple mutant, whether FIT overexpression can activate endogenous FIT transcription, and how dual overexpression of bHLH38 and FIT affects FIT expression.

1.1 Plant Materials and Growth Conditions

All Arabidopsis thaliana plants used in this study were of the Columbia-0 ecotype. Seeds were surface-sterilized with 70% ethanol for 15 minutes and washed at least three times with distilled water before being plated on growth medium. After stratification at 4 °C for two days, seeds were transferred to a growth chamber (22 °C, 16 h light/8 h dark cycle). Iron-sufficient (+Fe) medium consisted of half-strength MS medium supplemented with 1% sucrose, 0.7% agar A, 0.1 mM Fe-EDTA, pH 5.8. Iron-deficient (-Fe) medium was identical except for the omission of Fe(II)-EDTA. The FIT overexpression plants used in this study were obtained from Dr. Hongqing Ling' s research group at the Institute of Genetics and Developmental Biology, Chinese Academy of Sciences (Cui et al., 2018).

1.2 Vector Construction and Plant Transformation

Total RNA was extracted from wild-type Arabidopsis roots and reverse-transcribed into cDNA. The full-length coding sequence of bHLH38 was amplified by PCR and cloned into the pOCA30 binary expression vector. The construct was transformed into Agrobacterium tumefaciens strain EHA105 and introduced into wild-type Arabidopsis via the floral dip method. T1 transgenic seeds were selected on half-strength MS medium containing 50 mg/L kanamycin.

1.3 Quantitative RT-PCR Analysis

Seven-day-old seedlings grown vertically on +Fe medium (0.1 mM Fe(II)-EDTA) were transferred to either +Fe or -Fe vertical plates for three days. Roots were then harvested and flash-frozen in liquid nitrogen. Total RNA was extracted from root tissues using a Trizol reagent kit and reverse-transcribed into cDNA using oligo(dT)18 primers from a reverse transcription kit (TaKaRa). Quantitative PCR was performed using the SYBR Premix Ex TaqTM kit (TaKaRa) on a Roche Light Cycler 480 real-time PCR system, with ACT2 serving as the internal reference gene.

2 Results and Analysis

2.1 bHLH38 Overexpression Promotes FIT Expression Under Iron-Sufficient Conditions

We selected bHLH38 as a representative member of the bHLH Ib transcription factor family for investigation. IRT1 and FRO2, which are target genes of FIT and bHLH Ib transcription factors, are induced by iron deficiency and served as positive marker genes in our experiments. Quantitative PCR analysis of iron deficiency-responsive genes revealed that under iron-sufficient conditions, expression levels of IRT1, FRO2, and FIT were significantly higher in bHLH38 overexpression plants compared to wild-type. However, under iron-deficient conditions, their expression in bHLH38 overexpression plants was similar to or slightly lower than that in wild-type plants [Figure 1: see original paper].

Data represent means ± SD (n = 3). Different letters above bars indicate statistically significant differences (ANOVA, $P < 0.01$); lowercase letters for +Fe conditions, uppercase letters for -Fe conditions. The same notation applies below.

2.2 FIT Transcription Is Not Induced by Iron Deficiency in the Quadruple Mutant

We subjected the bHLH Ib quadruple mutants bhlh4x-1 and bhlh4x-2 to iron deficiency treatment (Cai et al., 2021) and analyzed IRT1, FRO2, and FIT expression by quantitative PCR. Under iron-sufficient conditions, IRT1 and FRO2 expression levels in bhlh4x-1 and bhlh4x-2 were significantly lower than in wild-type, while FIT expression showed no significant change. In contrast, under iron-deficient conditions, expression of IRT1, FRO2, and FIT in both bhlh4x-1 and bhlh4x-2 was significantly lower than in wild-type [Figure 2: see original paper].

2.3 Exogenous FIT Overexpression Cannot Activate Endogenous FIT Transcription

We performed iron deficiency treatments on FIT overexpression plants and analyzed expression of IRT1, FRO2, and endogenous FIT by quantitative PCR [Figure 3: see original paper]. Endogenous FIT expression was quantified using a primer pair spanning the last exon and 3' UTR of the FIT gene. The results showed that IRT1 and FRO2 expression levels were slightly upregulated in FIT overexpression plants under both iron-sufficient and iron-deficient conditions, whereas endogenous FIT expression did not differ significantly between FIT overexpression and wild-type plants.

2.4 Dual Overexpression of bHLH38 and FIT Promotes Endogenous FIT Expression Under Iron-Sufficient Conditions

We generated plants with dual overexpression of bHLH38 and FIT, subjected them to iron deficiency treatments, and analyzed transcriptional changes in IRT1, FRO2, and endogenous FIT by quantitative PCR. The results demonstrated that under both iron-sufficient and iron-deficient conditions, IRT1 and FRO2 expression levels were higher in dual overexpression plants than in wild-type. Notably, endogenous FIT levels were elevated above wild-type only under iron-sufficient conditions, while under iron-deficient conditions they were comparable to wild-type levels [Figure 4: see original paper].

3 Discussion and Conclusion

Iron is an essential mineral element for plant growth and development, with its bioavailability largely dependent on soil pH. In acidic soils, some iron exists in ionic forms accessible to plants, whereas in alkaline soils iron predominantly occurs as insoluble oxidized compounds. As sessile organisms, plants growing in alkaline soils must cope with iron deficiency stress. Through long-term evolution, plants have developed specialized mechanisms to adapt to iron-limited environments, enabling them to sense changes in iron concentration and activate expression of iron uptake-related genes through a sophisticated iron signaling transduction system.

In the Arabidopsis iron deficiency response system, FIT occupies a central position as a transcription factor that directly controls expression of the iron uptake genes IRT1 and FRO2 (Schwarz & Bauer, 2020). However, FIT transcription is itself induced by iron deficiency (Colangelo & Guerinot, 2004), making the mechanism of FIT activation under iron-limited conditions a focal point in iron signaling research.

Overexpression of bHLH39, another member of the bHLH Ib subfamily, has been shown to activate FIT expression under iron-sufficient conditions (Naranjo-Arcos et al., 2017), demonstrating that bHLH39 positively regulates FIT transcription when iron is abundant. Our analysis of bHLH38 overexpression plants revealed a similar trend in FIT expression, further supporting the functional redundancy among bHLH Ib family members. Due to this strong redundancy, single and double mutants of the four bHLH Ib members exhibit no obvious iron deficiency phenotypes, triple mutants show mild symptoms (Sivitz et al., 2012; Wang et al., 2013; Maurer et al., 2014), while the quadruple mutant displays severe iron deficiency symptoms (Cai et al., 2021). Our study found that FIT expression in the quadruple mutant was not significantly altered under iron-sufficient conditions but was substantially reduced compared to wild-type under iron deficiency, indicating that bHLH Ib factors are essential for FIT upregulation during iron deficiency.

Previous work showed that FIT promoter activity is significantly lower in fit mutants than in wild-type (Wang et al., 2007), suggesting that FIT is required for its own transcription. Our results demonstrate that endogenous FIT expression does not differ between FIT overexpression and wild-type plants, indicating that exogenous FIT overexpression alone is insufficient to promote endogenous FIT transcription. Therefore, FIT is necessary but not sufficient for its own transcriptional activation. In the iron homeostasis signaling pathway, FIT forms heterodimers with bHLH Ib members to co-activate downstream genes IRT1 and FRO2 (Yuan et al., 2018; Wang et al., 2013). While bHLH39 overexpression can promote IRT1 and FRO2 expression in wild-type plants, it fails to activate these target genes in the fit mutant background (Naranjo-Arcos et al., 2017), demonstrating that bHLH39-mediated activation of IRT1 and FRO2 requires FIT participation. Under iron-sufficient conditions, both bHLH38 overexpression and bHLH38/FIT dual overexpression promote transcription of IRT1, FRO2, and endogenous FIT. In contrast, under iron-deficient conditions, dual overexpression enhances IRT1 and FRO2 expression but not FIT expression, while bHLH38 single overexpression has minimal effects on IRT1, FRO2, and FIT. We therefore conclude that bHLH Ib factors are necessary but not sufficient for iron deficiency-induced activation of FIT.

When exogenous FIT protein is overexpressed, Arabidopsis activates the 26S proteasome degradation system to promote FIT turnover, thereby maintaining relatively stable FIT protein levels (Meiser et al., 2011; Sivitz et al., 2011). Researchers propose that under iron-deficient conditions, plants must maintain stable levels of active FIT protein to ensure adequate iron uptake while preventing iron toxicity from excessive accumulation (Sivitz et al., 2011). Our data indicate that FIT transcript levels reach a maximum under iron deficiency, and even additional positive regulatory transcription factors cannot further increase FIT transcription, suggesting that plants either do not require or cannot sustain excessively high endogenous FIT transcript levels. This threshold phenomenon for endogenous FIT transcripts mirrors the previously observed threshold for FIT protein levels. We hypothesize that plants have evolved multiple mechanisms to maintain iron homeostasis, and controlling FIT levels at both the RNA and protein levels may be a critical component of this regulatory network. Beyond transcriptional and post-translational regulation, Arabidopsis can also adjust the nuclear-to-cytoplasmic ratio of FIT protein and modulate its phosphorylation status in response to iron availability (Gratz et al., 2019, 2020), ultimately maintaining iron homeostasis. This study provides new insights into how plants coordinate multiple regulatory pathways to maintain iron homeostasis by analyzing FIT transcriptional changes from an RNA-level perspective.

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