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Article

Carbon Monoxide Signal Breaks Primary Seed Dormancy by Transcriptional Silence of *DOG1* in *Arabidopsis thaliana*

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Abstract: Primary seed dormancy is an adaptive strategy that prevents germination for viable seeds in harsh environment, ensuring seeds germination under favorable condition. Accurately inducing seeds germination in a controllable manner is important for crop production. Thus searching the chemicals that efficiently breaks seed dormancy is valuable. DOG1 protein abundance in the freshly harvested seed is high, and its level is correlated to seed dormancy intensity, thus DOG1 is regarded as the timer to evaluate the seed dormancy degree. In this study, we found the carbon monoxide (CO) donor treatment, the transgenic line with high CO content, showed lower seed dormancy, while scavenging CO, or the mutant with lower CO level, presented strong primary seed dormancy, genetic analysis showed that DOG1 was targeted by CO signal and was prerequisite for CO-dependent seed dormancy release. Furthermore, we found CO signal activated the expression of ERF/AP2 transcriptional factor *ERF12*, as well as enhanced the binding of ERF12 to the promoter of *DOG1*, ultimately transcriptional silence of *DOG1* expression to break primary seed dormancy. Meanwhile CO signal reduced the histone acetylation level at the chromatin of *DOG1* locus to suppress its expression. Together, our results revealed that CO acts as the novel regulator to suppress *DOG1* expression and efficiently break primary seed dormancy through activating the negative factor ERF12.

Keywords: Arabidopsis; DOG1; carbon monoxide; primary seeds germination

1 Introduction

A critical characteristic of plant adaptive fitness is the ability to precisely coordinate the shift between vegetative and reproductive development in response to seasonal change or environment stress. Seeds germination is a crucial period for plant life cycle. The mature seeds sustain the dormancy status, and germinate immediately once entering into the favorable environment. In the field, the seed dormancy can be interrupted after a period of after-ripening storage or seeds stratification under cold and moisture condition [1–4]. During agricultural application, uniform and fast seeds germination shows necessary for high-efficient management, while lower seed dormancy results into preharvest sprouting in crops, such as wheat, rice and barley, that seriously reducing grain yield and quality, thus investigating the regulatory

mechanism for seeds germination or dormancy show vital for us. Seeds germination and dormancy status is severely restrained by endogenous phytohormone level and external environment factors. The balance of gibberellins (GA) and abscisic acid (ABA) determines the conversation between seeds germination and dormancy, ABA plays the central role to establish and maintain seed dormancy, whereas GA antagonizes ABA to promote seeds germination. Besides GA/ABA biosynthesis, the components associated with GA/ABA signal transduction also affect seeds germination or dormancy status. For example, a series of ABSCISIC ACID INSENSITIVE (ABI) factors, such as ABI3, ABI4 and ABI5, are functional in ABA-dependent inhibition of seeds germination. On the contrary, DELLA proteins, including REPRESSOR OF GA (RGA), GIBBERELLIC ACID INSENSITIVE (GAI) or RGA-LIKE2 (RGL2), as the negative factor of GA signal also suppresses seeds germination [1,3,5]. Apart from GA and ABA, other plant hormones, such as auxin, ethylene or BR are also reported to regulate seeds germination [6–9].

Besides endogenous phytohormone level, the natural genetic variation in Arabidopsis also affects the seed dormancy degree in a typical quantitative manner [10,11]. Several quantitative trait loci (QTL) for delay of germination (DOG) have been identified [4,12,13]. Among them, DOG1 is the major loci to control seed dormancy in Arabidopsis. DOG1 is mainly induced in seed maturation, and its expression is enhanced under cool seed maturation conditions to strengthen seed dormancy. DOG1 protein abundance in the freshly harvest seeds is proportional to seed dormancy degree, the mutant deficiency in DOG1 protein accumulation loss the dormancy feature, thus DOG1 is used as the timer to indicate seed dormancy release intensity. Though the DOG1 protein abundance remains unchangeable during after-ripening, the seeds gradually recovers the germination capability possibly for the inactivation of DOG1 though protein modification [14]. DOG1 expression is also regulated via alternative splicing and polyadenylation, histone modification, or *cis*-acting antisense noncoding transcript, suggesting the multifaceted regulatory mechanism underlying DOG1 [15–18]. Recently Le et al. reported the ERF/AP2 transcriptional factor ERF12 recruits the corepressor TPL to regulate DOG1 expression and break seed dormancy [9], other component such as bZIP67 activates DOG1 to establish the primary seed dormancy. DOG1 to control seed dormancy [19], but the regulatory mechanism of DOG1 in controlling primary seed dormancy during environment change needs more investigation.

Carbon monoxide (CO) is a small signal that plays multiple function in animal [20–22], its physiological role in plant is also reported [23–25]. Heme oxygenase (HO, EC 1.14.99.3) is responsible for CO production during catalyzing heme to biliverdin. In Arabidopsis, HY1 encodes HO, and is also the main enzyme for CO generation, it is an inducible enzyme in response to environment stress, and is associated with salt stress [26]. HY1-mediated CO signal regulates the expression of ABI4 to modulate stomata closure, and enhance Arabidopsis tolerance to drought stress [26], our previous result showed that CO acts as the antioxidant to scavenge ROS overaccumulation and enhance seeds germination after desiccation treatment [27,28]. In this study we reported CO is also involved in controlling primary seed dormancy. Our pharmacologic and genetic experiments showed that CO acts as the signal to efficiently break primary seed dormancy. ERF12 acts as the negative factor to suppress seed dormancy. We found that CO signal activates the expression of EFR12, as well as strengthening the binding of EFR12 to the promoter of *DOG1*, ultimately suppressed *DOG1* expression to break seed dormancy. Thus our work identifies CO as the novel regulator to break seeds primary dormancy by integrating EFR12 to suppressing *DOG1* expression, this finding broaden our knowledge on the physiological function of CO signal in plant.

2 Material and Method

2.1 Plant Materials and Growth

All *Arabidopsis* mutant and transgenic lines were generated from a Columbian (Col-0) background. Mutant seed of *erf12* was obtained from the ABRC (Ohio State University, Columbus, USA). Mutant seeds of *hy1-100* and *HY1-ox* were reported before [28]. The transgenic *p12S:ERF12-Flag* (abbreviated

as *ERF12-Flag*) was constructed as previous reported method [9]. After cold stratification at 4°C for 3 days, the seeds were sterilized and sown on 0.6% agar (pH 5.7) plates. Adult plants were grown under long-day conditions at 22°C in a growth chamber with a 16-h white light (50 μmol m^{-2} s^{-1})/8-h dark cycle. Homozygous seeds in each batch were harvested at the same time and dried in an incubator at 22°C for approximately 2 months prior to the germination assays.

2.2 Seed Germination Measurement

Seed *germination* percentage was measured as reported method [28]. The freshly harvested seeds were surface-sterilized and transferred to wet filter paper containing different concentration of CO donor or scavenger. At least 100 seeds were used for each experiment, and three biological replicates were used for statistical analysis.

2.3 RNA Extractions and Quantitative RT-PCR

After different treatments, seeds were collected and total RNA was extracted using TRIzol (Invitrogen) according the manufacturer's recommended method [28]. After DNAase I treatment to remove potential genomic contamination, first-strand cDNA was synthesized using 500 ng of total RNA in a 10-μL system with M-MuLV reverse transcriptase (Fermentas) and oligo (dT)18 primers. Transcription levels of target genes were measured via real-time PCR using SYBR Green I master mix and a Roche LightCycler 96 PCR machine (Roche). At least three different biological replicates were used to confirm gene expression patterns, and PP2A or Ubiquitin 10 was used as an internal gene expression control. Primers used are described before [28].

2.4 CO Content and HY1 Enzyme Activity Measurement

Carbon monoxide in the imbibition seeds was measured using a previously described method [27,28]. In brief, about 100 mg of *seeds* were ground into a fine powder and transferred into a 2 ml bottle. Samples were stored under vacuum conditions in an ultralow chiller prior to further processing. Each tube was added with 20 μl 1-octanol and 1 ml distilled water were sequentially, and then one milliliter of sulfuric acid was added into each bottle carefully. Bottles were shaken briefly, and then placed into a 70°C water bath for 3 h with occasional shakings. After each bottle was cooled down to room temperature, 1 ml of air from the headspace was taken to quantify the CO concentration using a GC/MS system. The HY1 enzyme activity was measured as previously described [28].

3 CHIP Analysis

CHIP analysis was performed as described method before. In brief, the collected seeds were cross-linked in 1% formaldehyde solution under a vacuum for 30 min, and the chromatin was extracted and shared with sonication to an average length of 300–500 bp, the sonicated chromatin was immunoprecipitated with anti-Flag (Catalog no 6795, Sigma) and anti-acetyl-histone H3 (Catalog no 06–599, Millipore). The amount of each immunoprecipitated DNA fragment was measured by quantitative PCR using the primes listed as our before paper.

4 Results

4.1 CO Signal Modulates Primary Seed Dormancy in Arabidopsis

We previously reported that CO signal play the important role in seeds germination under light irradiation [28], for example, the *hy1-100* mutant is defective in the biosynthesis for CO and displays the lower germination after light irradiation, whereas over-expressing *HY1* with high CO content shows higher germination, compared with the wild-type Col seeds. Since CO signal is involved in regulating seeds germination, we hypothesized that CO signal may also be functional as a key positive regulator for enhancing seeds germination tolerance. To test it, we grew the wild-type Col seed in standard conditions and scored the germination percentage of seeds after 3 day of imbibition. The germination of freshly

harvested Col seeds showed was less than 10%, and the seeds germination percentage gradually increased after prolonging the after-ripening period, the seeds germination achieved over 90% after three weeks of after-ripening storage. However, incubating the freshly harvested Col seed with CO donor CORM-2 obviously increased the seeds germination in contrast to the seeds incubating with water as the control. Similarly, CORM-2 treatment also increased the seeds germination for those seeds after 1 or 2 weeks of after-ripening storage (Fig. 1A). ZnPPIX is the scavenger for CO and widely used to suppress the accumulation of CO in plant. We found ZnPPIX treatment distinctly repressed the germination capability for those seed with various period of after-ripening storage (Fig. 1A). Thus these data suggest that CO signal acts as the novel regulator to break primary seed dormancy.

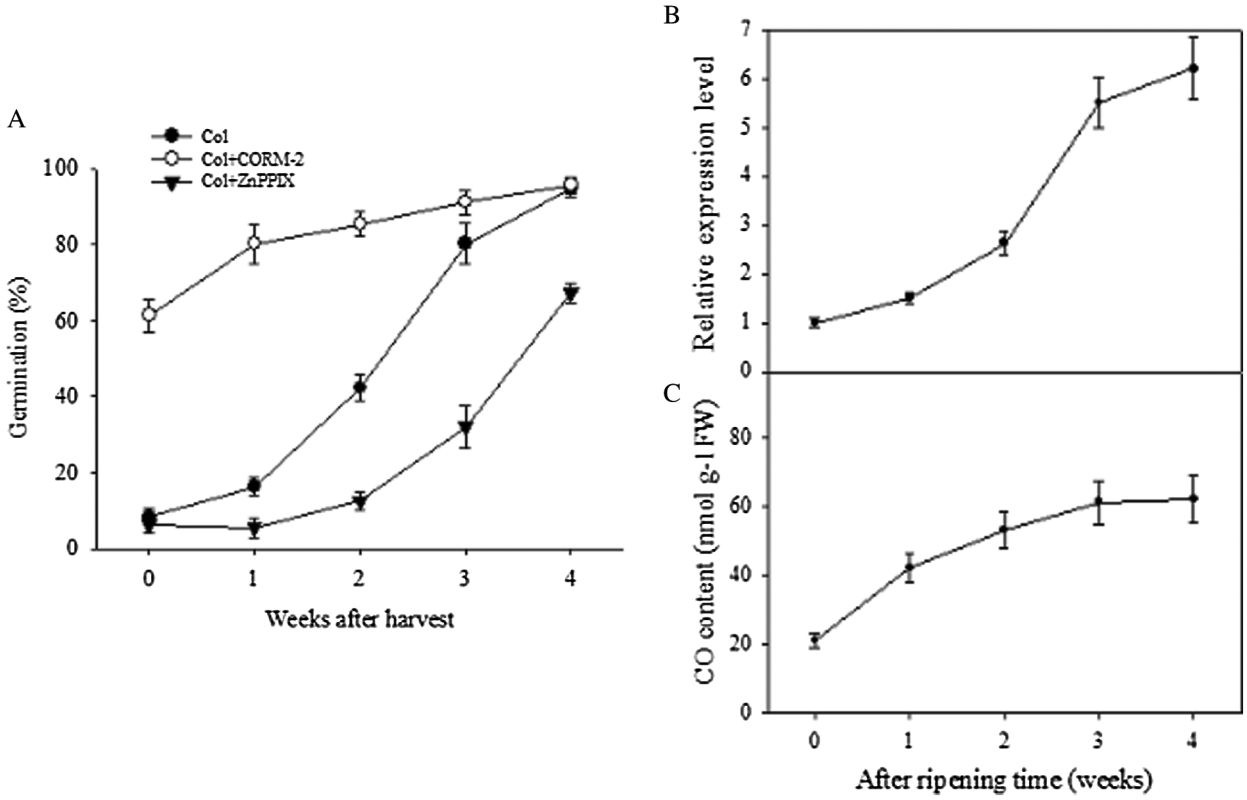

Figure 1: CO signal breaks the primary seed dormancy. A) Germination percentage of freshly harvest seeds or after-ripening seeds with different periods of dry storage. These seeds were sowed with the wet filter with or without the CO donor CORM-2 (1 μM) or CO scavenger ZnPPIX (100 nM) for 3 days, and the seeds germination percentage was recorded. The bars mean ±SE of three independent batches of seeds experiments. (B) & (C) The transcriptional level of *HY1* and CO content in freshly harvest seeds or after-ripening seeds. The freshly harvested or after-ripening seed for indicated storage time was germinated for 3 days, and the HY1 enzyme activity or CO content was measured. The bars mean ±SE of three independent batches of seeds experiments

In Arabidopsis, HY1 is the main enzyme responsible for CO biosynthesis [24,26], we also measured the activity of HY1 in the freshly harvest seeds or the after-ripening seeds. As shown Figs. 1B and 1C, the transcripts of *HY1* in freshly harvested seeds was lower, prolonging the storage time gradually increased its enzyme activity. Consistently, the CO content in the freshly harvested seeds was also lower than that in after-ripening seeds. These data also suggest the potential role of CO signal in control primary seed dormancy.

4.2 Disruption of HY1 Enhances, But Over-Expressing HY1 Breaks, the Seeds Primary Dormancy in Arabidopsis

As exogenous CO treatment breaks the primary seed dormancy, we want to know if endogenous CO signal is also functional to modulate primary seed dormancy. To test the function of endogenous CO in planta, we used the *hy1-100* mutant which is defective in CO biosynthesis and contained lower level of CO, and the transgenic *HY1-ox* seeds in which HY1 is strongly expressed under the control of 35S promoter, leading into high level of endogenous CO content. We then compared the primary seeds germination among fresh-harvested *hy1-100*, *HY1-ox* and Col. In contrast to Col, and the *hy1-100* seed showed deeper seed dormancy while *HY1-ox* seeds showed slighter seed dormancy (Fig. 2A). Additional CO donor CORM2 treatment added the seeds germination of *hy1-100*, but ZnPPIX treatment reduced the seeds germination of *HY1-ox*. Thus these seeds phenotype are consistent with the exogenous CO treatment, and suggest that CO signal negatively control primary seed dormancy, that is to say, high level CO signal reduces primary seed dormancy, while low level of CO signal increases primary seed dormancy.

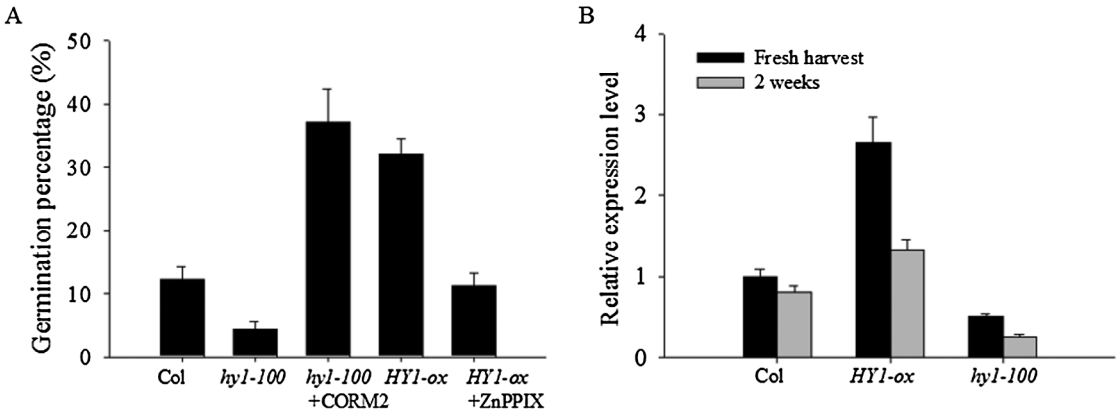

Figure 2: CO signal breaks primary seed dormancy and regulates the expression of *DOG1*. The bars mean ±SE of three independent batches of seeds experiments. A) Genetic analysis of the seeds germination in Col, *hy1-100* and *HY1-ox* seeds. These seed was incubated with wet filter paper containing CORM-2 (1 μM) or ZnPPIX (100 nM), and the seeds germination was recorded after 3-day of incubation. The wet paper without chemical was used as the control. CO signal regulates the expression of *DOG1*. The freshly harvested or two-week after-ripening seeds of Col, *hy1-100* or *HY1-ox* seeds were used, and the transcript of *DOG1* was measured by RT-qPCR analysis

4.3 CO Signal Negatively Regulates the Transcriptional Level of DOG1

DOG1 is the main loci to co ntrol primary seed dormancy [13,14], thus we reasoned that CO signal regulated the expression of *DOG1*. To test it, we performed quantitative RT-PCR to measure the transcripts of *DOG1* in freshly harvested Col, *hy1-100* and *HY1-ox* seeds, and found that the transcriptional level of *DOG1* was higher in the *HY1-ox* seeds, but lower in the *hy1-100* seed, in contrast to the wild-type Col seeds (Fig. 2B). Thus these data suggest that the CO content is correlated with the *DOG1* transcripts level that determine to the germination potential in freshly harvested seeds.

4.4 CO Signal Enhances the Binding of ERF12 to the Promoter of DOG1

Previously study demonstrated that CO signal regulates stomatal movement and drought response through the ERF/AP2 transcriptional factor ABI4 [26], another ERF/AP2 transcriptional factor ERF12 is also reported to bind the *DOG1* promoter to suppress *DOG1* expression [9]. It is possible that CO affects the expression of *DOG1* through ERF12. To this end, we first tested the effect of CO on the

transcriptional level of *ERF12*, and found incubating the freshly harvested Col seed with CORM-2 increased the transcriptional level of *ERF12*, while incubation the seeds with ZnPPIX reduced the transcriptional level of ERF12 (Fig. 3A). As the expression pattern of *DOG1*, the Col, *hy1-100, erf12* showed lower seeds germination, while *HY1-ox* and *dog1-3* mutant showed higher seeds germination (Fig. 3B). We crossed *hy1-100* with *dog1-3*, and found *hy1-100/dog1-3* showed high seeds germination, suggesting HY1 regulates seeds germination through DOG1.We also crossed *HY1-ox* with *erf12*, and found *HY1-ox/erf12* showed relative lower seeds germination than *HY1-ox* (Fig. 3B), suggesting the promoting function of HY1 requires ERF12.

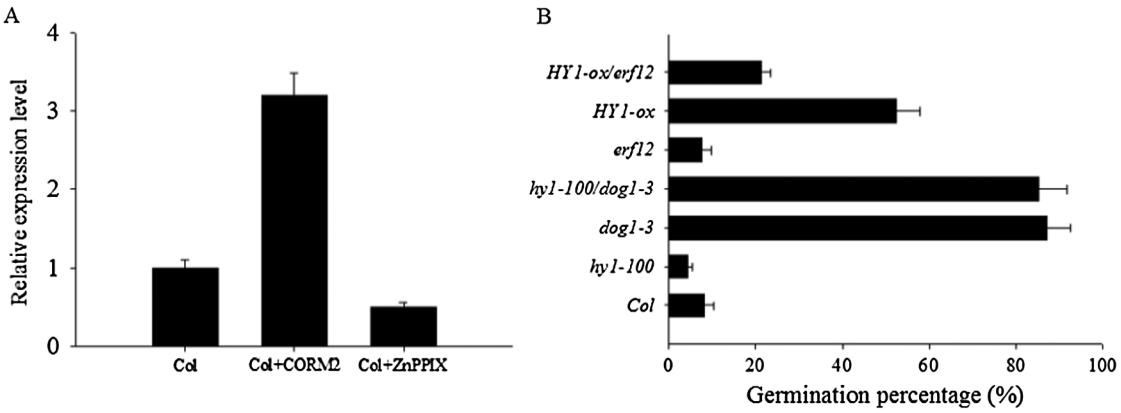

Figure 3: ERF12 is required for breaking primary seed dormancy by CO signal. The bars means ± SE of three independent batches of seeds experiments. A) Effect of CO signal on the expression of *ERF12*. The fresh harvested Col seeds was treated with CORM-2 or ZnPPIX for 24 h, and the transcript of ERF12 was measured by RT-qPCR analysis. Genetic analysis of seeds germination percentage among Col, *hy1-100, HY1-ox, dog1-3* and *erf12* parent mutant and their crossed seeds. All of these fresh harvested seed was imbibed for 3 day and the seeds germination percentage was calculated

ERF12 recognizes the DRE/CRT motif within the promoter of *DOG1* to suppress its expression. Meanwhile, we also checked whether CO treatment enhances the binding of ERF12 to the promoter of *DOG1*. To this aim, we generated the transgenic line expressing *ERF12-Flag* fusion under the control of seeds specific *12S* promoter (*p12S:ERF12-Flag*, abbreviated as *ERF12-Flag*), the CHIP analysis showed that CORM-2 treatment increased, while ZnPPIX treatment reduced, the binding of EFR12 to the fragment containing DRE/CRT motif within *DOG1* promoter (Fig. 4). We also introduced *p12S:ERF12-Flag* into *hy1-10* or *HY1-ox* line to generate *ERF12-Flag/hy1-100* and *ERF12-Flag/HY1-ox*, In contrast to *ERF12-Flag* under Col genetic background, CHIP analysis revealed that more P3 fragment was enriched in the imbibition seeds of *ERF12-Flag/HY1-ox, but* less P3 fragment was enriched in the imbibition seeds of *ERF12-Flag/hy1-100* (Fig. 4). Thus these data suggest that CO signal increases the transcriptional level of *ERF12*, as well as enhances the binding of negative factor EFR12 to the promoter of *DOG1* to block its expression.

4.5 CO Signal Reduces the H3 Acetylaion Level at DOG1 Promoter to Repress Its Expression

Our previous results showed that CO recruits HDA6 to epigenetic deacetylation of *SOM* loci and repress *SOM* expression [28]. As ERF12 recruits TPL complex to suppress *DOG1* expression, and TPL complex also interacts with HDA6 [9,29], thus we hypothesized that CO signal also modify the deacetylation level at the promoter *DOG1* to suppress *DOG1* expression. In order to check the effect of CO on the acetylation level within the *DOG1* promoter, we performed CHIP analysis to compare the different acetylation level at different fragment within the DOG1 promoter region or its first exon region. As

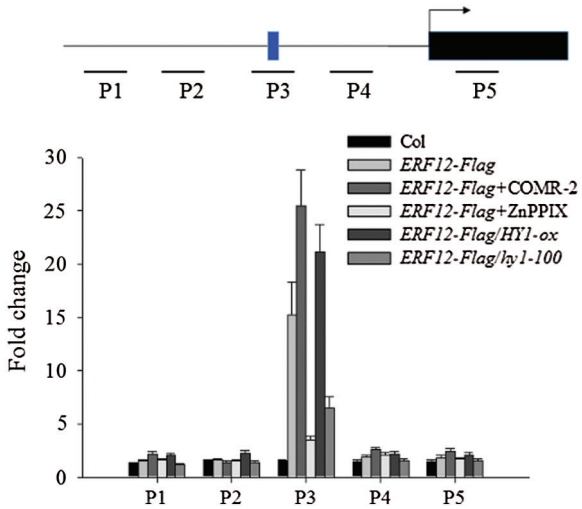

Figure 4: CO signal enhances the binding of EFR12 to the promoter of *DOG1* by CHIP-qPCR analysis. The freshly harvested Col and *ERF12-Flag* seeds were treated with CORM-2 or ZnPPIX for 24 h, and the binding capability of ERF12 on the different fragment within *DOG1* promoter or the first exon was measured by CHIP-qPCR analysis. The anti-Flag antibody was used for immunoprecipitation treatment. *ACTIN8* was used as the internal control. The experiment was repeated three times with independent sample. The bars mean ± SE of three independent batches of seeds experiments. The schematic diagram of the *DOG1* gene structure was shown at the upper panel. The relative position of the PCR amplified fragments (P1 to P5) for each tested region is depicted below the gene structure

shown in Fig. 5, the P3 fragment within the *DOG1* promoter presented high acetylation level in the imbibition *hy1-100* seeds, but lower in the *HY1-ox* seeds, compared with wild-type Col seeds. The H3ac level at P4 or P5 region in *hy1-100* was also a little higher, but not obviously than that in P3 region. In Arabidopsis, HDA6 is responsible for the histone deacetylation modification, we thus introduce *HY1-ox* into *hda6* mutant to get *HY1-ox/had6* seeds, and found the relative acetylation level of H3ac in *HY1-ox/had6* than *HY1-ox* seeds alone. These data suggest that CO signal reduces histone acetylation modification within the *DOG1* chromatin through HDA6 activity to negatively regulate *DOG1* expression.

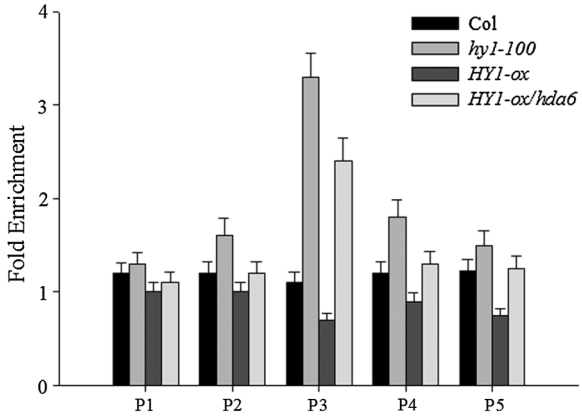

Figure 5: CHIP-qPCR analysis of the histone acetylation level at the chromatin of *DOG1* locus. The freshly harvested seed of Col, *hy1-100*, *HY1-ox* or *HY1-ox/hda6* was imbibed for 24 h, and the histone acetylation level within the promoter and first exon region of *DOG1* was measured by anti-Histone antibody. The bars mean ± SE of three independent batches of seeds experiments

5 Discussion

As we know, primary seed dormancy is an adaptation strategy ensuring plant germination at appropriate time and condition [1–3], but the deep seed dormancy also caused lower and inconsistent seeds germination in agriculture production, resulting into crop yield, thus it is valuable to discover a new seeds germination regulator to break the seed dormancy. Accumulated evidences demonstrated the diversity physiological function of CO in plant response to environment stress [24–26]. Our previous study showed that CO treatment enhances seeds enhances seeds germination tolerance to desiccation treatment in *Baccaurea ramiflora* [27]. CO signal also promotes light-irradiation induced seeds germination by inducing the biosynthesis of NO in Arabidopsis [28]. In this study we reported that CO signal act as the new regulator to break primary seed dormancy in Arabidopsis, as the CO donor treatment, or the transgenic *HY1-ox* seeds with high endogenous CO content appeared obviously higher seeds germination in the freshly harvested Col seed, while the CO scavenger treatment, or the CO mutant *hy1-100* seeds showed deep seed dormancy. Previous study showed that ROS generation is prerequisite for seeds germination [30], and CO signal is reported to inducing ROS signal to enhance plant tolerance, it is possible that CO triggers the ROS signal to break seed dormancy [27,31]. Another possibility is that CO interplay with NO to break seed dormancy, as NO triggers the s-nitrosylation of ABI5 for its degradation, ultimately promoting seeds germination [32]. Thus more work need to be more investigate to understand the cross talk role of CO with ROS or NO in regulating primary seed dormancy in future.

DOG1 is the main QTL to determine the primary seed dormancy, the transcriptional level of *DOG1* [12–14], as well its protein abundance is high in the freshly harvest seeds to ensure seed dormancy, the DOG1 activity gradually declines to loss its dormancy during after-ripening storage [14], hence DOG1 is regarded as the timer for indicate the seed dormancy intensity. In this study our RT-qPCR analysis showed that CO signal suppressed the expression of *DOG1*, the genetic analysis showed that *hy1-100/dog1-3* double mutant seeds showed similar lower seed dormancy, these data suggest the conclusion that the effect of CO signal on breaking seed dormancy depends on DOG1. Several evidences demonstrate that there exists the accurate regulatory mechanism for DOG1 homeostasis, such as alternative splicing or polyadenylation, or non-coding antisense regulating *et al.* [15–18]. Several *cis*-elements is identified in the promoter region within the *DOG1* promoter, and corresponding transcriptional factors, such as bZIP67 or ERF12 were reported to recognize these *cis*-element to positively or negatively modulate *DOG1* expression, respectively, and ultimately control the primary seed dormancy [9,19]. It is reported that ethylene signal induce the expression of ERF12 through ethylene receptor, EFR12 then recruits the transcriptional corepressor TPL to suppress *DOG1* expression and release seed dormancy [9]. Previous study showed that CO signal control Arabidopsis drought stress through the ERF12/AP2 factor ABI4 [26]. In this study we found that CO signal induced the expression of EFR12, which as the paralog of ABI4 also belongs to the ERF/AFP2 transcriptional factor. On the one hand, CO donor treatment directly, or the transgenic *HY1-ox* seeds, displayed high transcriptional level of ERF12, and vice versa for the wild-type Col seeds with CO scavenger treatment, or the *hy1-100* seeds. On the other hand, our CHIP analysis revealed that CO signal treatment enhances the binding capability of EFR12 to the DRE/CET motif within the *DOG1* promoter. Thus data suggest that CO signal activates the transcriptional level of ERF12, also enhances the binding of ERF12 to the promoter of DOG1, to suppress the expression of DOG1. Since EFR12 recruits TPL to suppress the expression of DOG1, and TPL interacts with HDA19 as the TPL-HDA complex to suppress target gene expression through HDA19-dependent histone deacetylation. In agreement with the result that CO signal suppress DOG1 expression, the CHIP analysis showed that the histone acetylation level at P3 fragment within DOG1 promoter was lower in the imbibition *HY1-ox* seeds, or by CO donor treatment. Conversely, the histone acetylation level at P3 fragment was lower in the *hy1-100* mutant seeds, or by CO scavenger treatment. Supporting our data,

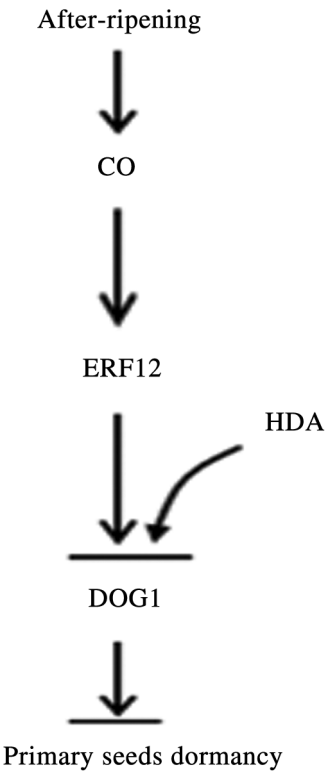

Figure 6: The proposed model illustrating the role of CO signal in breaking primary seeds germination through ERF12 and downstream DOG1. We propose that after-ripening storage induces the CO generation through HY1 enzyme activity, the accumulation of CO activates the expression of ERF12, also enhances the binding of ERF12 to the promoter of *DOG1*, the increased ERF12 then recruits HDA activity to reduce the histone acetylation level at the chromatin of *DOG1*, therefore silencing *DOG1* expression to break primary seed dormancy

previous study also showed that CO signals enhance the binding of HDA6 to the promoter of SOM to regulate seeds germination under light [28].

6 Conclusion

Our finding reveals the novel function of CO signal in breaking primary seed dormancy. Based on our data we propose a model to illustrate the role of CO in controlling primary seed dormancy. As shown Fig. 6, the freshly harvested seeds contain high level of *DOG1* to induce primary seed dormancy, the HY1-dependent CO biosynthesis gradually strengthens for after-ripening seeds, as a result, CO signal activates the expression of EFR12, as well as enhancing the binding of ERF12 to promoter of *DOG1*. Meanwhile, EFR12 recruits TPL/HDA complex to the promoter region of DOG1 and trigger the histone acetylation level at the *DOG1* locus, finally suppresses the expression of *DOG1* to break primary seed dormancy. Therefore, our finding highlights the new function of CO in breaking primary seed dormancy and uncovers the novel mechanism by which CO signal suppresses *DOG1* expressing through activating ERF12 and recruit TPL/HDA complex.

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Conflicts of Interest: The authors declare that they have no conflicts of interest to report regarding the present study.

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