1. Please translate the **first paragraph** (30%) in Chinese:

Plants defend themselves from pathogen attack by an array of mechanisms, including preformed and induced responses. The defenses may be induced throughout the plant and depend on the perception of the pathogen. Localized and systemic defenses rely on activation of one or more signaling pathways that lead to the induction of defense gene expression. The most studied of these pathways are regulated by salicylic acid (SA), jasmonic acid (JA), and ethylene (ET) or their derivatives (for review, see Thatcher et al., 2005). These pathways have been associated with resistance to different types of pathogens, with the SA-dependent pathway mainly providing resistance to biotrophic(營養上絕對寄生) pathogens while the JA and ET pathways provide resistance predominantly to necrotrophic(營養上破壞寄主細胞維生) pathogens (Thomma et al., 1998; Glazebrook, 2005). In many instances, the JA and ET pathways have been shown to regulate similar types of defense genes (Schenk et al., 2000; Lorenzo and Solano, 2004).

The regulation of plant defense responses is complex, with a number of transcription factor families playing important roles (Rushton and Somssich, 1998; Singh et al., 2002). There is considerable interest in identifying and utilizing key transcription factors in plant defense for engineering increased resistance to plant pathogens in agriculture (Gurr and Rushton, 2005). One transcription factor family that is being explored is the ET response factor (ERF) family, members of which are a point of integration of the JA and ET pathways (Lorenzo et al., 2003). In Arabidopsis (*Arabidopsis thaliana*), there are thought to be 147 members of the AP2/EREBP family of plant transcription factors (Feng et al., 2005; Nakano et al., 2006). The proteins encoded by the *AP2/EREBP* gene family have diverse functions throughout the plant life cycle, including regulation of development, responses to abiotic stresses such as drought and cold, as well as to biotic stresses such as fungal pathogen infections (Feng et al., 2005). The AP2/EREBP family is divided into the RAV, AP2, and EREBP subfamilies, with the EREBP subfamily being divided into DREB or A subgroup and the ERF or B subgroup. The ERF or B subgroup contains 65 *ERF* genes and contains all of the *AP2/EREBP* genes that have been linked to disease resistance responses (Gutterson and Reuber, 2004). *ERF* genes have been shown to be responsive to both JA and ET (Oñate-Sánchez and Singh, 2002; Lorenzo et al., 2003; Gutterson and Reuber, 2004; McGrath et al., 2005), while work in tomato (*Lycopersicon esculentum*) has revealed direct regulation of the ERFs *Pti4* and *Pti5* by the PTO R protein following recognition of *Pseudomonas syringae* pv *tomato* (Zhou et al., 1997). ERFs are known to bind to the GCC box and related elements in the promoters of JA/ET-inducible, pathogenesis-related (*PR*) genes, such as the defensin *PDF1.2*, basic chitinase (*ChiB*), and thionin (*Thi2.1*), and either induce or repress the expression of these genes (Menke et al., 1999; Fujimoto et al., 2000; Ohta et al., 2001; Tournier et al., 2003). Several members of the *ERF* gene family have been shown to be functionally involved in plant defense against pathogens, as overexpression leads to increased expression of *PDF1.2*, *ChiB*, and *Thi2.1* and increased resistance to a range of pathogens, both necrotrophic and biotrophic (Berrocal-Lobo et al., 2002; Gu et al., 2002; McGrath et al., 2005). Although most ERFs described so far are activators, 14 Arabidopsis ERF proteins contain an ERF-associated amphiphilic repression (EAR) motif (Nakano et al., 2006), which has been shown to function as a repression domain (Fujimoto et al., 2000; Ohta et al., 2001). Overexpression of *AtERF4*, an EAR-containing ERF, reduces *PDF1.2* induction by methyl jasmonate (MeJA) and plant resistance to *Fusarium oxysporum* (McGrath et al., 2005).

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Although overexpression of several *ERF*s has been shown to modify defense gene expression and resistance to pathogens, little has been reported on defense phenotypes caused by silencing, mutation, or knockout of *ERF*s (McGrath et al., 2005). Since the ERF family in Arabidopsis contains 65 members (Feng et al., 2005; Nakano et al., 2006), many of which are regulated by the same stimuli and potentially bind the same promoter element, it may be expected that a high level of functional redundancy exists and, thus, isolation of mutant phenotypes with knockout of a single *ERF* is uncommon. This notion is supported by the observation that few *AP2/EREBP* genes have been isolated through loss-of-function mutant screens. Exceptions are *BD1* (Chuck et al., 2002) and its ortholog *FZP* in maize (*Zea mays*; Komatsu et al., 2003), and the *DREB* or A subfamily genes *ABI4* (Finkelstein et al., 1998) and *CBF2* (Novillo et al., 2004) that control development or response to cold and drought conditions. To date, to our knowledge, no gene of the 65 member *ERF* or A subfamily that is associated with pathogen defense has been isolated through a mutant screen.

Previously, we identified Arabidopsis *ERF* genes whose expression was specifically induced by *P. syringae* pv *tomato* DC3000 (*avrRpt2*) infection with overlapping but distinct induction kinetics (Oñate-Sánchez and Singh, 2002). We chose *AtERF14* for further characterization since it was the only *ERF* whose induction started later than 6 h following *P. syringae* pv *tomato* DC3000 (*avrRpt2*) infection when potential downstream genes were also being induced. This unique expression pattern suggested that AtERF14 may play a different role than the other studied *ERF* genes that were induced prior to defense gene induction. We show that overexpression of *AtERF14* leads to increased *ERF* and defense gene expression and pleiotropic effects, including severe growth retardation and loss of seed set. Interestingly, loss-of-function mutations of *AtERF14* lead to loss of ET-mediated induction of defense genes and other *ERF*s. These results suggest a non redundant role for AtERF14 in the coordination of *ERF* and defense gene expression. Moreover, loss-of-function mutants showed increased susceptibility to *F. oxysporum*, confirming that AtERF14 plays a key role in defense against some pathogens. These results are the first report of a loss-of-function mutant phenotype for an ERF activator and show that the *AtERF14* gene is important for ET responses and pathogen resistance.

Based on the article above, please answer to the following questions in English:

2. Why is it important to study the function of transcription factors involved in plant defense mechanisms? (20%)

3. Ethylene response factors (ERFs) bind to what kind of promoter elements, which are known to be present in the promoter region of what genes? (15%)

4. Silencing ERFs through knockout mutation (disruption of the gene that will than block protein formation) was not very successful in identifying defense phenotypes. Do you have an explanation? (15%)

5. Why the authors choose to study *AtERF14* into details? (20%)

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