The CXXC domain of DEMETER Is critical for DME's Functional Robustness *in vivo*.

DNA methylation maintains TE silencing is important for genome stability, and for the stable inheritance of epigenetic information. The homeostasis of DNA methylation is achieved by maintenance and de novo DNA methylation, as well as by active DNA demethylation, catalyzed by the DEMETER (DME)-like family of DNA glycosylases. DME is a plant-specific DNA glycosylase/demethylase essential for seed viability. In addition to the catalytic glycosylase domain, DME also contains a CXXC domain whose function has not been elucidated in plant. The mammalian CXXC domain is known to bind unmethylated cytosines, mediate protein-protein interactions, and is required for localization to target sites in certain cases. In this study, we found that the CXXC domain is dispensable for DME's targeting to specific genomic regions, but it is critical for maintaining DME's functional robustness in vivo. Plants expressing a CXXC-impaired version of DME (CTD-AXXA, where the conserved cysteines were mutated to alanines) only partially rescuing the *dme* seed abortion phenotype. Methylome analysis revealed that in the aborted seeds expressing CTD-AXXA, DME was able to fully demethylate a small subset of loci—primarily within gene body regions—whereas most targets remained partially demethylated. Notably, the CTD-AXXA performance improved under elevated temperature but declined when plants were grown in cold conditions. Based on these findings, we propose a model that the CXXC domain enhances DME's in vivo efficiency by stabilizing its association with an unidentified demethylation complex, thereby ensuring complete and robust demethylation of target loci under variable environmental conditions.