**Figure legends**



**Fig. 1. Growth of pakchoi seedlings exposed to Cd concentrations of 2 mg/L, 4 mg/L, 6 mg/L, and 8 mg/L, respectively, for 32 days.** (A) The phenotypic identification of pakchoi seedlings. (B) ROS accumulation and relevant physiological indicators, including O2·**-** levels (a), H2O2 levels (b), MDA content (c), and enzyme activities of POD (d), SOD (e), CAT (f) into leaves. (C) DAB and NBT staining in the large leaves. Data are presented as mean ± SD (n=3). \*p<0.05, \*\*\*p<0.01, means a significant difference compared to the control.



**Fig. 2. Observation of chromatin decondensation levels induced by different Cd concentrations in pakchoi.** (A) Chromatin decondensation of representative interphase nuclei from different Cd concentration treatments. (B) Quantitative analysis of immunostaining results in (A) using Metamorph software. (C) Nucleolus size increased during Cd treatment. (D) Statistical analysis of nucleolus size. Nuclei were stained with DAPI (blue). More than 400 nuclei were analysed for each antibody. Data are presented as mean ± SD (n=3). \*p<0.05, \*\*\*p<0.01 compared to the control group according to the t-test. a-e represent the groups Cont, Cd2, Cd4, Cd6, and Cd8, respectively.

**Fig. 3. Growth of pakchoi seedlings exposed to Cd concentrations of 6 mg/L plus six low concentrations of epi-modification inhibitors.** (A) The phenotypic identification of pakchoi seedlings exposed to Cd concentrations of 6 mg/L plus six low concentrations of epi-modfication inhibitors 5-AC, RG108, TSA, CUDC101, AT13148, and H89 for 32 days. (B) Cd accumulation. (C) Antibodies specific for H3, H3K9ac, and H4K5ac were used to analyse histone extracts from pakchoi seedlings. (b)–(c) Quantitative analysis of H3K9ac and H4K5ac in (a) Image J. Data are presented as mean ± SD (n=3). \*p<0.05, \*\*\*p<0.01, means a significant difference compared to the control.

**Fig. 4. Effect of low concentrations of epi-modification inhibitors on the response mechanism of the antioxidant system under Cd stress.** (A) Concentration of O2·- (a) and H2O2 (b). (B) Enzyme activities of POD (a), SOD (b), and CAT (c) in leaves. Data are presented as mean ± SD (n=3). \*p<0.05, \*\*\*p<0.01, means a significant difference compared to the control.

**Fig. 5. Effect of low concentrations of epi-modification inhibitors on Cd stress-induced cell cycle arrest.** (A) Flow cytometry results for ploidy levels treated with six low concentrations of epi-modification inhibitors 5-AC, RG108, TSA, CUDC101, AT13148, and H89 plus additional Thi for 32 days under Cd stress. (B) Percentage of cells in G1, S, and G2 phase detected by flow cytometry. Data are presented as mean ± SD (n=3). \*p<0.05, \*\*\*p<0.01, means a significant difference compared to the control.

**Fig. 6. Low concentrations of epi-modification inhibitors attenuated Cd-induced DNA damage and Thi acted synergistically.** (A) Detection by TUNEL assay. (B) Detection of the γ-H2AX signal. Cell nuclei were stained with DAPE (blue). Bar=5 µm.

**Fig. 7. Effect of low concentrations of epi-modification inhibitors on the expression levels of genes related to histone acetylation**. (A) *GCN5*. (B) *HADC101*. (C) *HAT-B*. The expression levels of all genes in the root were measured by qPCR of mRNA prepared from the control group, the Cd6 group, and different treatment groups with epi-modification inhibitors. *β-ACTIN* was used as an internal control. Data are presented as mean ± SD (n=3). \*p<0.05, \*\*\*p<0.01, means a significant difference compared to the control.

**Fig. 8. Variations of ROS-related molecules and enzyme activities under Cd concentration of 6 mg/L plus high TSA concentration.** (A) The phenotypic identification of pakchoi seedlings exposed to Cd concentrations of 6 mg/L plus high TSA concentration. (B) Concentration of O2·- (a) and H2O2 (b). (C) Enzyme activities of POD (a), SOD (b), and CAT (c) in leaves. Data are presented as mean ± SD (n=3). \*p<0.05, \*\*\*p<0.01, means a significant difference compared to the control.