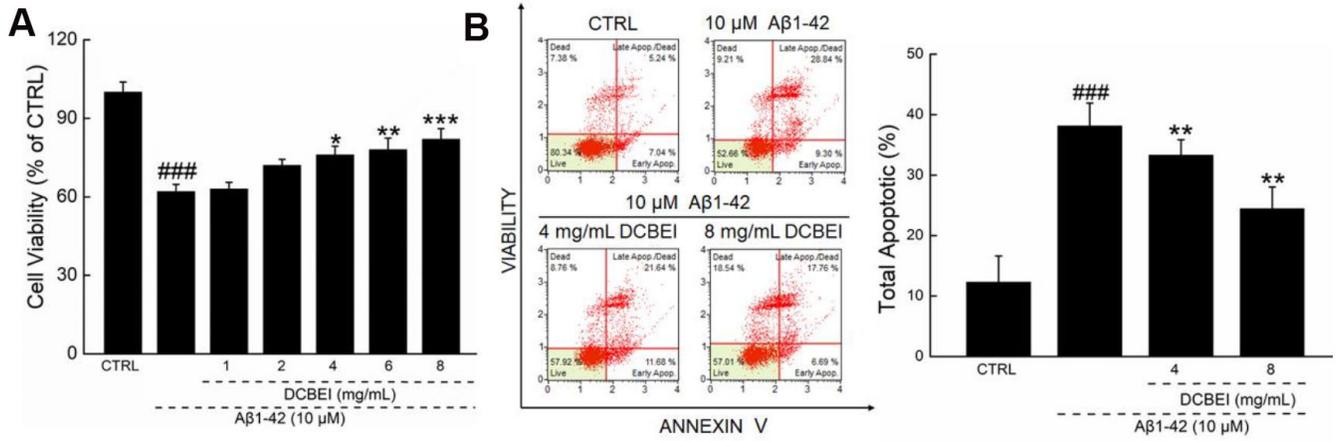
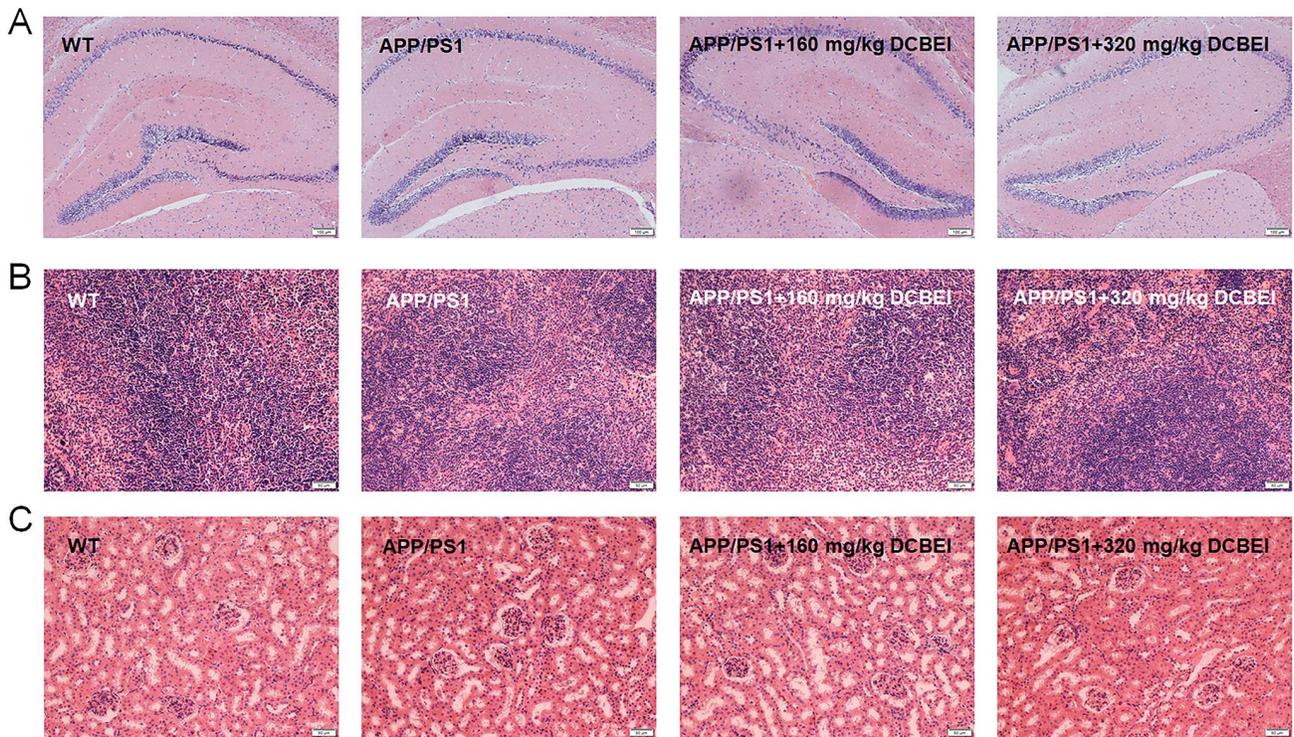


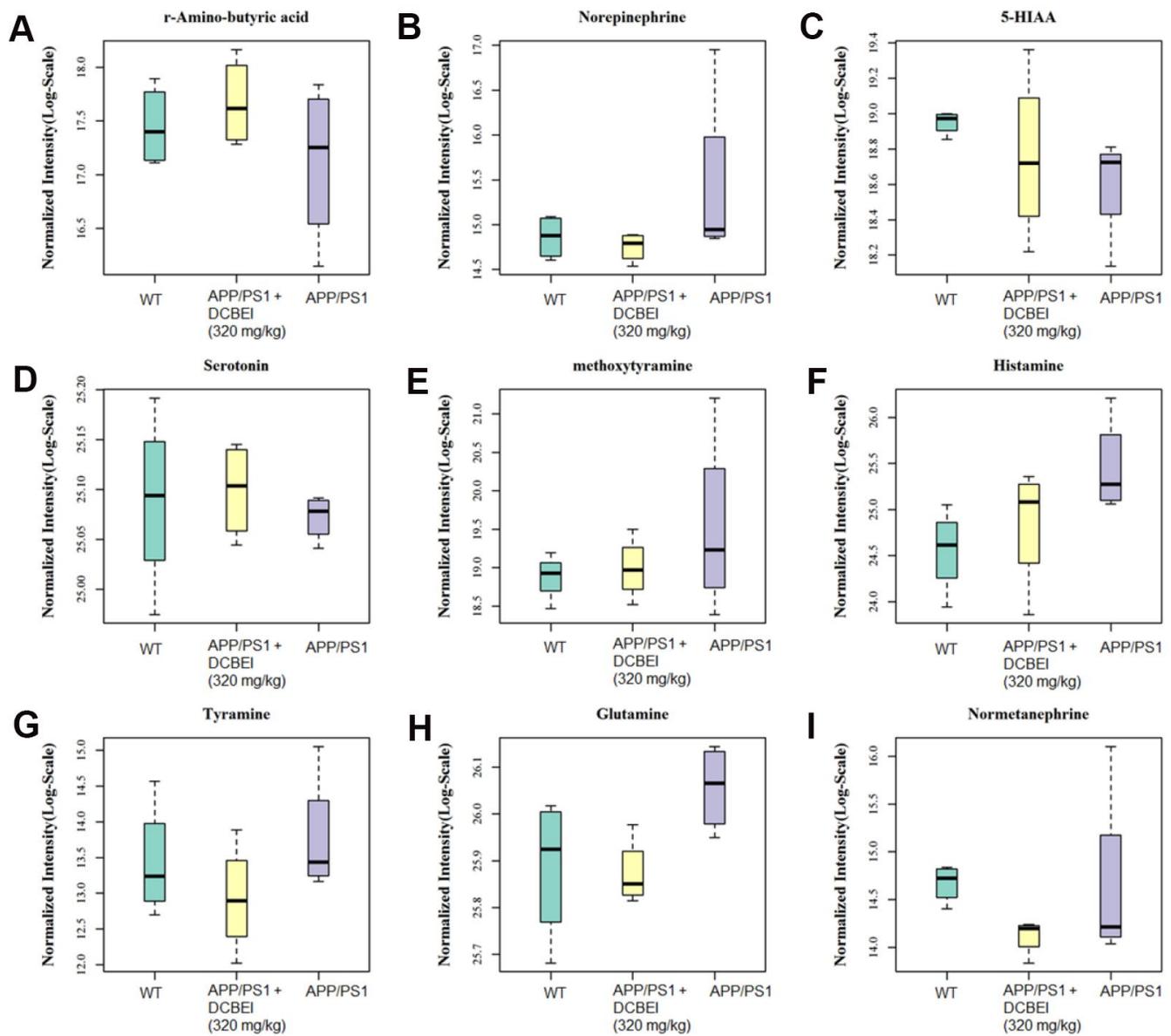
SUPPLEMENTARY FIGURES



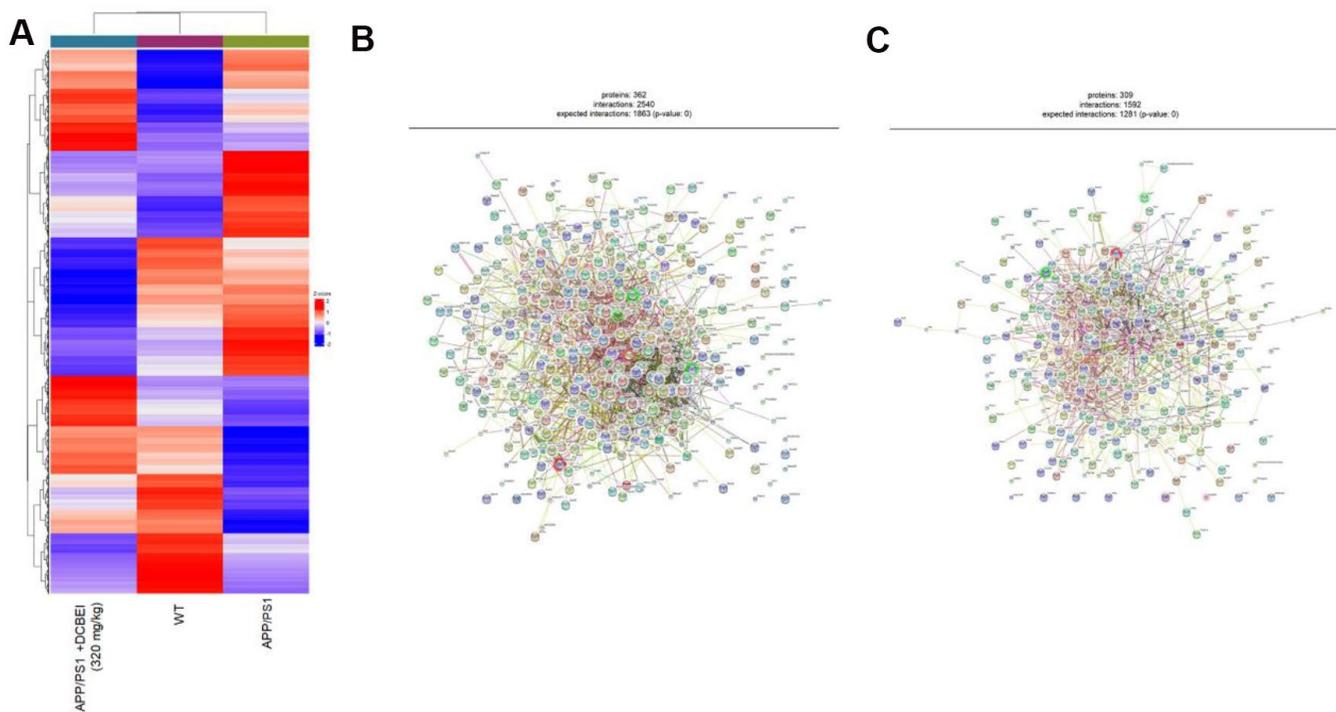
Supplementary Figure 1. DCBEI protects U251 cells against Aβ₁₋₄₂-induced damage. U251 cells were pre-incubated with DCBEI (4 mg/mL to 8 mg/mL) for 3 h, and co-treated with Aβ₁₋₄₂ for a further 24 h. **(A)** DCBEI increased cell viability. **(B)** DCBEI inhibited apoptosis. Data are expressed as a percentage of corresponding control cells and means ± S.D. (n = 6). ### *P* < 0.001 vs. CTRL, * *P* < 0.05, ** *P* < 0.01 and *** *P* < 0.001 vs. Aβ₁₋₄₂-treated cells.



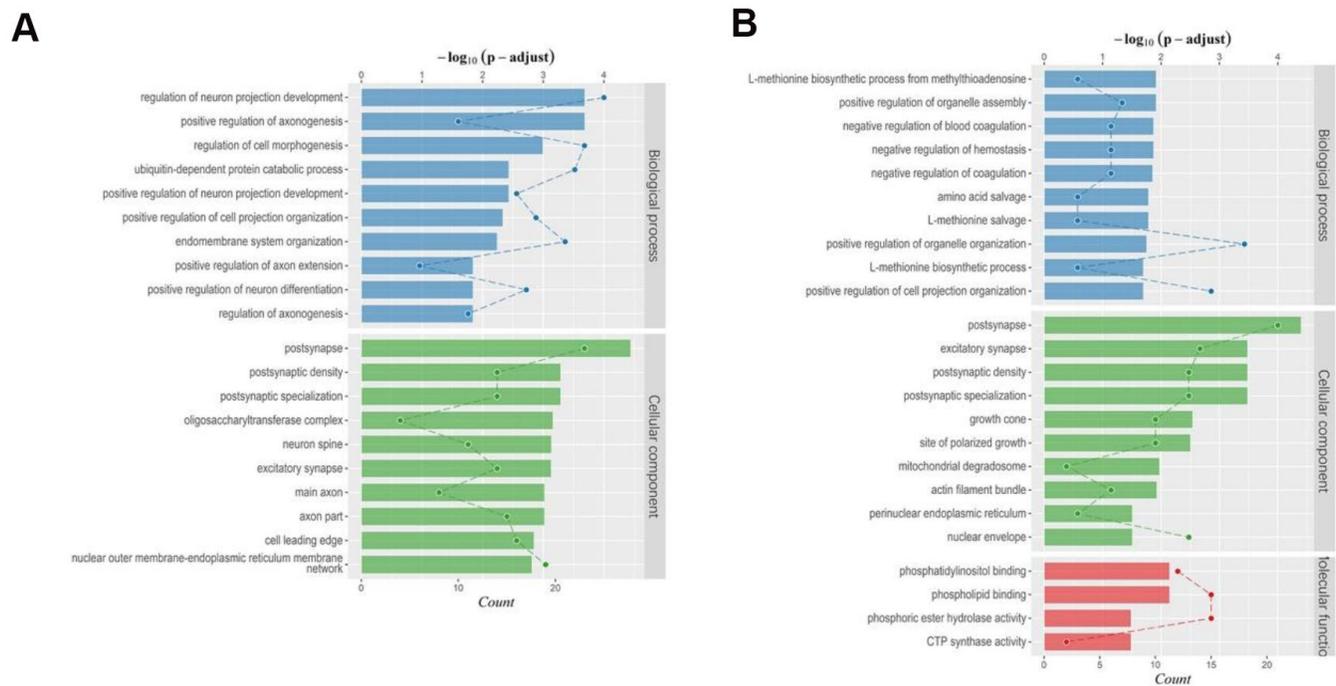
Supplementary Figure 2. Histopathological analysis via hematoxylin and eosin (H&E) staining. No significant changes on **(A)** brain tissues (100×) (Scale bar: 100 μm), **(B)** spleen tissues (200×) (Scale bar: 50 μm) and **(C)** kidney tissues (200×) (scale bar: 50 μm) were noted among all experimental groups including WT, APP/PS1, and DCBEI-treated APP/PS1 mice (n=3).



Supplementary Figure 3. DCBEI regulates neurotransmitter levels in serum analyzing by metabonomics via LC-MS/MS in APP/PS1 mice (n=3) including (A) r-amino-butyric acid, (B) norepinephrine, (C) 5-HIAA, (D) serotonin, (E) methoxytyramine, (F) histamine, (G) tyramine, (H) glutamine, and (I) normetanephrine.



Supplementary Figure 4. DCBEI broadly regulates protein expression analyzing by proteomics. (A) The list of differentially expressed proteins among each experimental group. Protein–protein interaction network of (B) WT vs. APP/PS1 group and (C) APP/PS1 vs. DCBEI group analysis via STRINGdb.



Supplementary Figure 5. GO enrichment analysis of (A) WT vs. APP/PS1 group and (B) APP/PS1 vs. DCBEI group.