Figure S1. Transcriptomic profiling and validation of the NOX1-mediated mechanism. (A) Efficiency of NOX1 knockdown using two independent siRNAs. Western blot analysis showing NOX1 protein expression in AGS and HGC-27 cells transfected with si-NC or two different NOX1-specific siRNAs (si-NOX1#1 and si-NOX1#2). GAPDH served as the loading control. *P<0.05, **P<0.01 and ***P<0.001 vs. the si-NC group. (B) Genetic inhibition of NOX1 abrogates Hesperadin-induced ROS generation. DCFH-DA fluorescence staining (green fluorescence represents ROS; scale bar, 50 μm) and quantitative analysis showing ROS levels in AGS and HGC-27 cells transfected with si-NC or si-NOX1 (#1 or #2) and treated with or without Hesperadin. *P<0.05, **P<0.01 and ***P<0.001 vs. the si-NC + Hesperadin group; ns: not significant (P≥0.05) vs. the si-NC group. (C) Western blot analysis of NOX1 protein expression in AGS and HGC-27 cells following treatment with Hesperadin, cisplatin, or their combination for 48 h. GAPDH was used as a loading control. Data were compared among all groups. *P<0.05, **P<0.01 and ***P<0.001. siRNA, small interfering RNA; si-NC, negative control siRNA; ROS, ROS, reactive oxygen species; ns, not significant (P≥0.05).