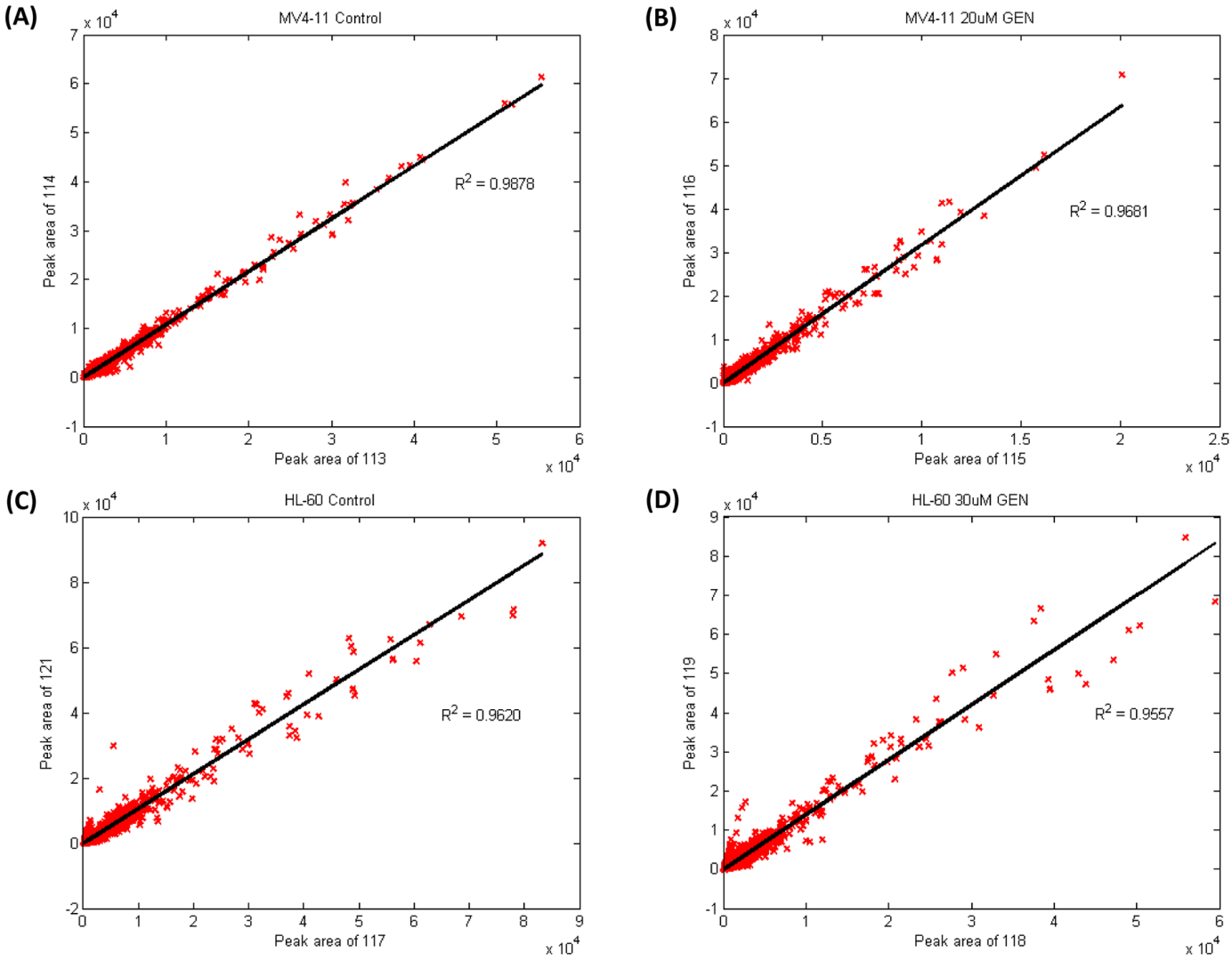
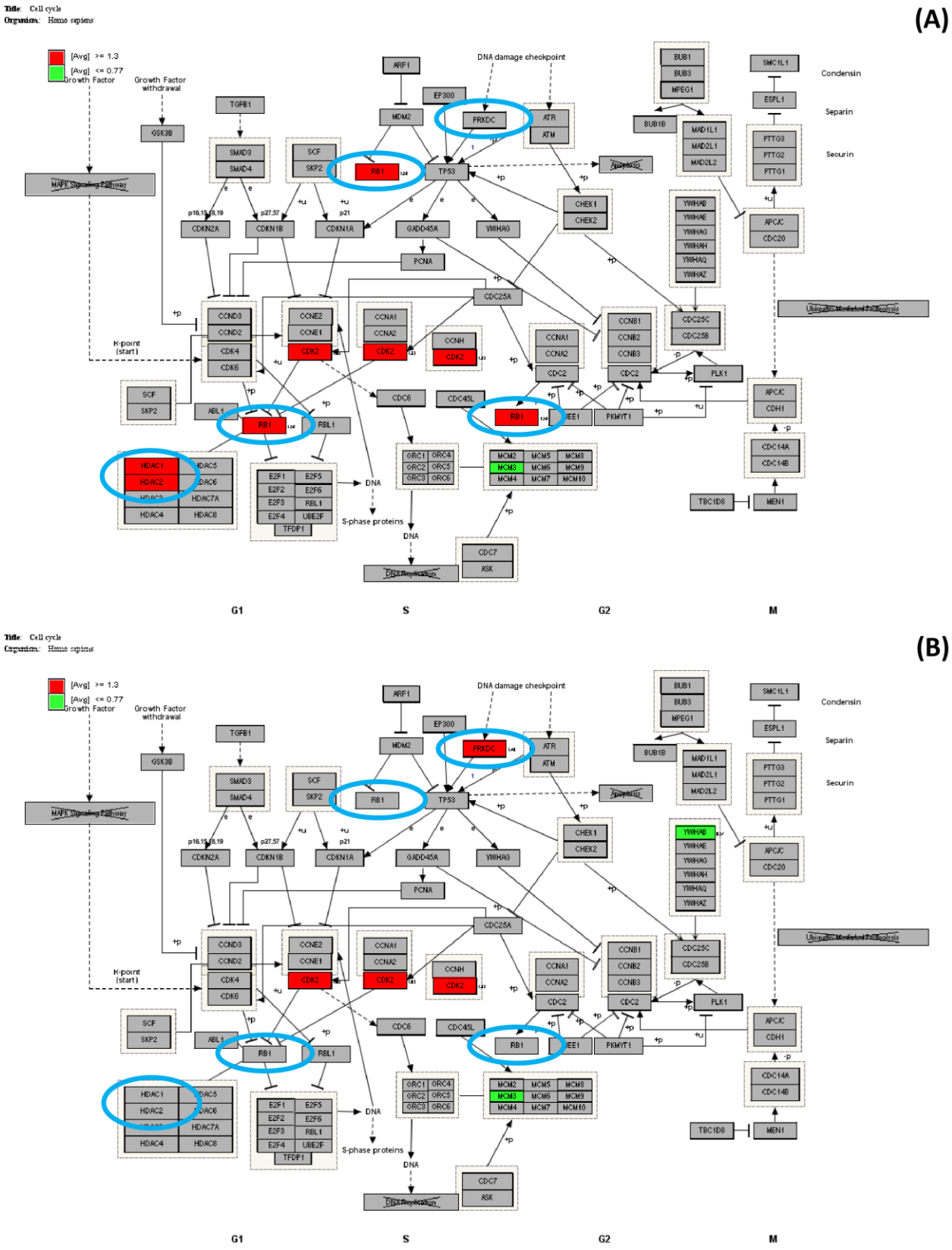
Genistein exerts anti-leukemic effects on genetically different acute myeloid leukemia cell lines by inhibiting protein synthesis and cell proliferation while inducing apoptosis – molecular insights from an iTRAQ™ quantitative proteomics study

Supplementary Material



Supplementary Figure 1: Regressional analysis of the scatter plots generated from the respective peptide peak areas from the various samples.
The peak areas of each peptides, obtained from the generated petide summary using ProteinPilot (AB SCIEX) software, for each labelled samples were ploted in a scatter plot before conducting a regressional analysis to determine the R^2 value for each set of treatment duplicates: (A)MV4-11 Control; (B)MV4-11 cells treated with 20uM of genistein (GEN); (C)HL-60 Control; (D) HL-60 cells treated with 20uM of GEN. R^2 values closed to 1 in a regressional analysis usually shows close correlation between the two sets of data points compared. The generally high R^2 values across all sets of treatment duplicates demonstrate the high conformity for each samples.



Supplementary Figure 2. Cell cycle pathway mapped with the iTRAQ™ data of MV4-11 (A) and HL-60 (B) cell lines. Blue circle highlights the difference in protein expression profile between MV4-11 and HL-60 cell lines after treatment with genistein. Retinoblastoma protein (RB1), Histone deacetylase 1 and 2 (HDAC1 and HDAC2) were up-regulated in MV4-11 cells but not in HL-60 cells. DNA-dependent protein kinase catalytic subunit (PRKDC) was up-regulate in HL-60 cells but not in MV4-11 cells.