**Supplemental Figure S1-S7**

**Figure S1.** Correlation of protein expression between PMA- and PMA + ionomycin-treated cells and between GMCSF- and GMCSF + IL4-treated cells. (A) Correlation of protein expression between PMA- and PMA + ionomycin-treated cells from experiment 1 dataset. (B) Correlation of protein expression between PMA- and PMA + ionomycin-treated cells from experiment 2 dataset. (C) Correlation of protein expression between GMCSF- and GMCSF + IL4-treated cells from experiment 1 dataset. (D) Correlation of protein expression between GMCSF- and GMCSF + IL4-treated cells from experiment 2 dataset.
Figure S2. Flow cytometric analysis of surface expression CD11b on human monocytes that were treated with two different differentiation protocols. (A) Dot plots representing fluorescence of Abs against CD11b on treated human monocytes. Grey population is the isotype control. Black population is the experimental sample. (B) Quantification of surface CD11b on treated monocytes. Fold change in MFI was determined by subtracting out the background fluorescence and normalizing data to monocyte surface expression. N=4.
Figure S3. CALM protein expression in monocytes, macrophages, and dendritic cells by TMT-MS/MS. (A). MS/MS spectrum of precursor ion at m/z 794.1307 (3+) corresponding to peptide sequence TMT6-ATTLSNAVSSLASTGLSLTHK(TMT6). (B). The reporter ions for quantification indicating CALM is up-regulated (~2 folds) in dendritic cells (DC) as compared in macrophages (Mac) and monocytes (Mono). (C). Western-blot analysis of CALM expression.
**Figure S4.** Validation of the anti-H3K9me3S10pK14ac antibody. (A) Sequences of the H3 competitor peptides used in the competition ELISA. (B) Competition ELISA assay of the anti-H3K9me3S10pK14ac antibody for its specificity toward H3 competitor peptides with concentration up to 50 µg/mL.
Figure S5. Histone H1 expression during myeloid cell differentiation. A heat map of histone H1.2, H1.4, and H1.5 expression profiles progressively changing from monoblast/premonocyte in U937 cells to monocytes, macrophages, and dendritic cells.
Figure S6. Inhibition of HDACs arrests differentiating monocytes in the G1/G0 phase of cell cycle. (A) Flow cytometry analysis of PI staining of monocytes in the presence or absence of apicidin, macrophages, and dendritic cells after 6 days of differentiation. Histograms represent the cell cycle profile. The furthest peak to the left represents G1/G0 phase, the second peak represents G2/M phase, and the space in between the peaks represents S phase. (B) Quantification of cells in the G2/M phase from panel A. Bar graphs (mean ± SEM) are representative of 20 replicates. Statistical analysis was performed using Two-way Anova.
Figure S7. Histone acetylation and differentiation. (A) H4 K16 acetylation is involved in monocyte differentiation. Bar graph (mean ± SEM) of the percent of cells positive for H4 K16 acetylation (N=4). Statistical analysis was performed using Two-way Anova. (B) Western blot of increasing acetylation at H4K16 in monocytes in response to 1µM apicidin treatment. Approximately 20µg of nuclear lysate as determined by A280 was loaded per lane. (C) Acetylation and methylation is associated with CD14, CD11b, and CD11C gene expression by analysis of public Geo Expression Omnibus data set (Accession number GSE36402). Primary normal adult lymphocytes were used for a ChIP- chip assay. After DNA to protein crosslinking, the chromatin was immunoprecipitated with either anti-H3K9me3, H3K9Ac, or H4K16Ac. Next, the crosslinking was reversed and the DNA subjected to tiling DNA array. The control input was sample with no antibody.