

Supplemental Materials

Glucocorticoids Inhibit Oncogenic RUNX1-ETO in Acute Myeloid Leukemia with Chromosome Translocation t(8;21)

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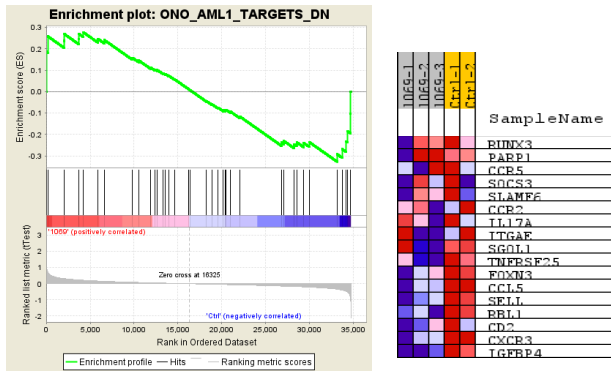
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Table S1. Primers for qPCR.

| Primer name | Sequence |
|--------------|--------------------------------|
| cd14_317F | TTTACAGAAGGGAGGCCCAAGT |
| cd14_317R | CTGTGATATCCCTGAGGACCA |
| TRKA-2936F | GCACCTCCATGAGGGTCATTT |
| TRKA-2936R | TTAGGGCCCAATCCCTACAG |
| p14ARF-86F | AGGAAGAAAGAGGAGGGGCT |
| p14ARF-86R | CCAGCCAGTCAGCCGAAG |
| Non-Trans-F1 | AACCTCACTTTTCATTGTTACTAGCCATA |
| Non-Trans-R1 | CGCTCAAGGATGTCAGTAGCAT |
| egr1-1F | GGTCAGTGGCCTAGTGAGC |
| egr1-1R | GTGCCGCTGAGTAAATGGGA |
| id1-1F | CTGCTCTACGACATGAACGG |
| id1-1R | GAAGGTCCCTGATGTAGTCGAT |
| p21-1F | TGTCCGTCAGAACCCATGC |
| p21-1R | AAAGTCGAAGTTCCATCGCTC |
| sla-1F | CGACTTCCTTGCCGTGCTAA |
| sla-1R | TCTCGACCAGTGCTAAGAGAA |
| cd11a-1F | TGCTTATCATCATCACGGATGG |
| cd11a-1R | CTCTCCTTGGTCTGAAAATGCT |
| cd11b-1F | ACT GGT GAA GCC AAT AAC GCA |
| cd11b-1R | TCC GTG ATG ACA ACT AGG ATC TT |
| cd34-1F | ACC AGA GCT ATT CCC AAA AGA CC |
| cd34-1R | TGC GGC GAT TCA TCA GGA AAT |
| baalc-1R | AGT CGG TGT AGG TGA GCC A |
| csf1r-1F | GGAATCCCAGTGATAGAGCC |
| csf1r-1R | TTGGAAGGTAGCGTTGTTGGT |

Figure S1. GSEA plot shows that although Bet treatment did not significantly cause down-regulation of the RUNX1-downregulated gene set, it reduced expression of many of these genes, as exemplified in the right panel (lower).



NES = -1.14; FDR = 0.243

Figure S2. Dose-response inhibitory activities for combination treatment of Kasumi-1 cells with Dex and Doxorubicin (upper), together with calculated combination indices (CI) of 0.42-0.90 showing synergy (lower).

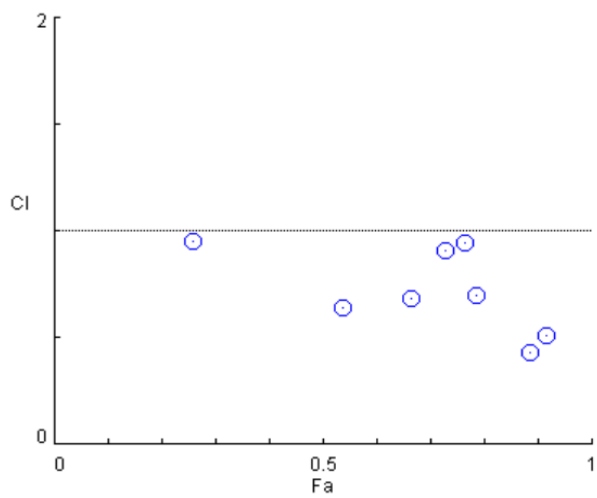
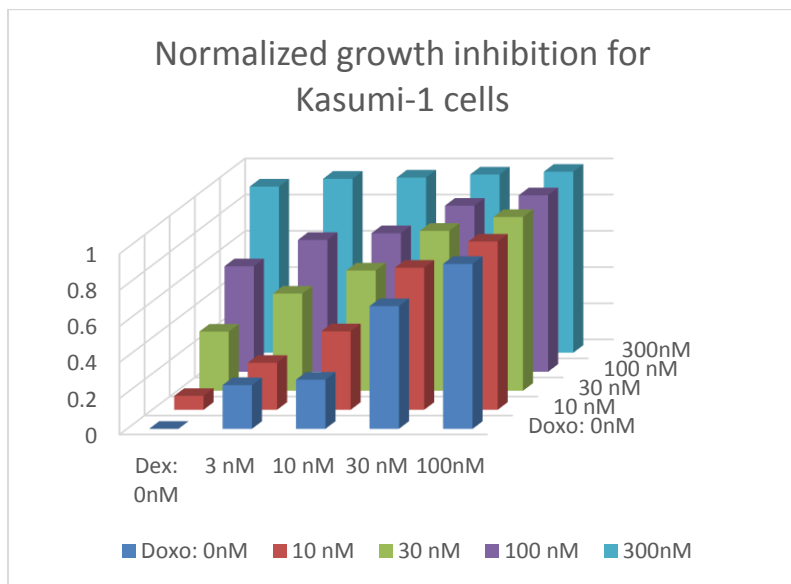


Figure S3. Immunoprecipitation showed GR is associated with RUNX1, but not R-E in Kasumi-1 cells, using a RUNX1 antibody (#4336 AML1 (D33G6) Rabbit mAb, from Cell Signaling) that recognizes both RUNX1 and R-E.

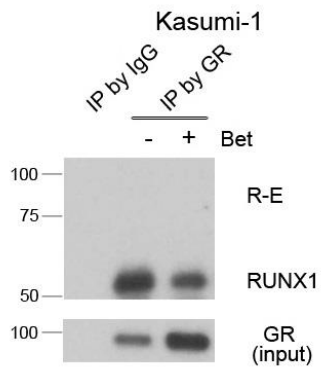


Figure S4. Promoter analysis for CD14, p14ARK and TRKA, showing potential RUNX1 (also known as AML1) and GR binding sites.

