

CORRIGENDUM

Aberrant Mer receptor tyrosine kinase expression contributes to leukemogenesis in acute myeloid leukemia

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Since the publication of this article, the authors have identified two inadvertent errors in Figure 3.

First, the phosphoarray in Figure 3c listed as Kasumi-1 was a duplicate of the NOMO-1 cell line phosphoarray. The Kasumi-1 and NOMO-1 data were correctly analyzed and represented graphically in Figure 3d. A revised version of Figure 3c with images of both the Kasumi-1 and NOMO-1 phosphoarrays is shown.

In addition, it came to our attention that some panels in Figure 3e were mislabeled and one set of loading controls were shown in duplicate. In the revised version derived from the Kasumi-1 cell line, correct phospho- and total protein immunoblots and corresponding loading controls are shown.

The revised data support the conclusions originally reported in this manuscript and the text of the manuscript remains unchanged. The authors apologize for any inconvenience this may have caused.



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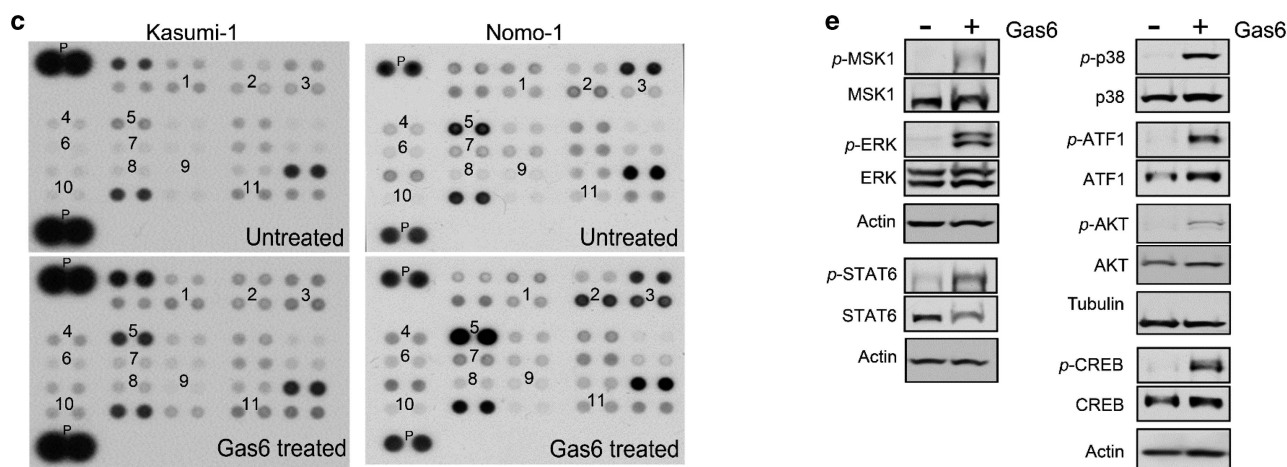


Figure 3. Merstimulation in AML cells activates oncogenic signaling pathways. Kasumi-1 and NOMO-1 cells were incubated in serum-free RPMI medium for two to three hours and then treated with 200nM rhGas6 or buffer. **(c)** Diluted lysates were incubated with human phospho-kinase array membranes and bound phospho-proteins were detected according to kit instructions. Each membrane contains positive control (P) antibodies spotted in duplicate. Proteins that demonstrated a 1.5-fold or greater increase in phosphorylation after stimulation with Gas6 (Gas6 treated) relative to buffer treated samples (Untreated) are marked by numbers between duplicate spots, which correlate with the identification numbers shown in **(d)**. **(e)** Cell lysates were prepared from cultures treated with rhGas6 (+) or buffer (-) and subjected to immunoblot analysis with antibodies specific for phosphorylated and total MSK1, p38, ERK1/2, STAT6, AKT, ATF1, and CREB proteins. Representative Kasumi-1 immunoblots are shown.