



Erratum

Early G2/M checkpoint failure as a molecular mechanism underlying etoposide-induced chromosomal aberrations

Shinichiro Nakada, Yoko Katsuki, Issei Imoto, Tetsuji Yokoyama, Masayuki Nagasawa, Johji Inazawa, and Shuki Mizutani

Original citation: *J. Clin. Invest.* **116**:80–89 (2006). doi:10.1172/JCI25716.

Citation for this erratum: *J. Clin. Invest.* **116**:2306–2307 (2006). doi:10.1172/JCI25716E1.

During the preparation of the manuscript, errors were introduced in the labels of Figure 3E and Figure 6A. The corrected figures are provided below.

We regret this error.

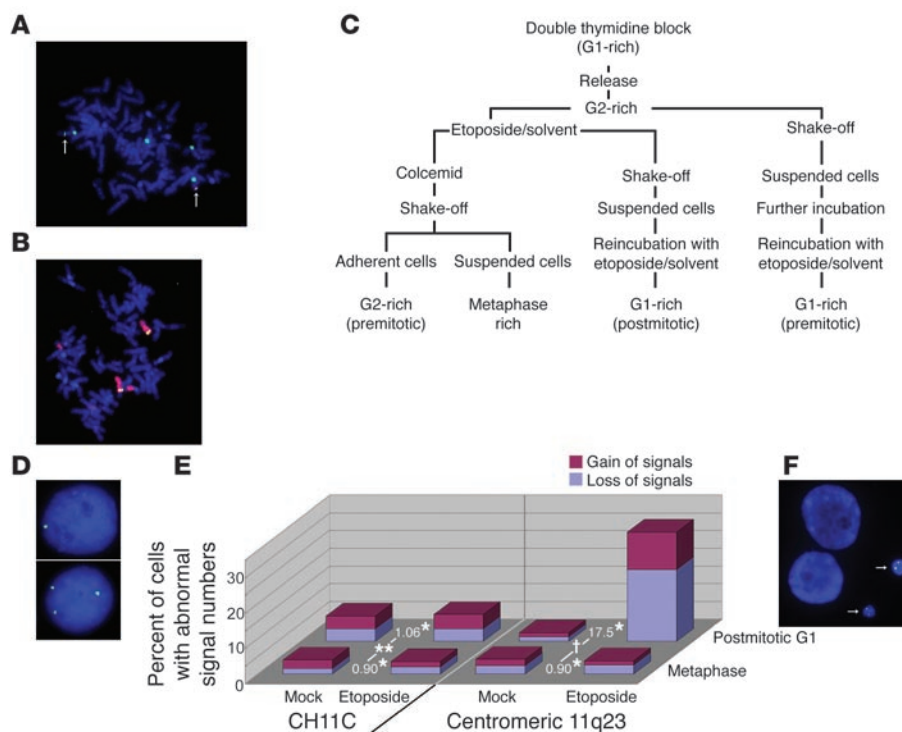


Figure 3

FISH analysis for chromosome 11. (A) Representative image of metaphase ATM-deficient fibroblasts hybridized with the CH11C probe (green) and MLL probes (green and red overlap). Chromosomes were stained by DAPI. The arrows indicate overlapping MLL signals. (B) Representative image of metaphase ATM-deficient fibroblasts hybridized with probes for whole chromosome 11 (red) and MLL (green and red overlap). (C) Flow diagram of the cell fractionation procedure. (D) Etoposide-treated postmitotic G1 phase ATM-deficient fibroblasts with 1 (upper panel) and 3 (lower panel) centromeric 11q23 signals. (E) Proportion of metaphase and postmitotic G1 phase cells with gain (red) and loss (blue) of CH11C (left) or centromeric 11q23 (right) probe signals. $n > 250$ for each. *Odds ratio (OR) for etoposide treatment. ** $P = 0.77$ and $†P < 0.0001$, differences in OR between postmitotic G1 and metaphase cells when cells were hybridized with CH11C and centromeric 11q23 probes, respectively (P for interaction term of cell cycle phase \times treatment by a logistic regression model). (F) Micronuclei containing centromeric 11q23 signals (arrows). Cells were hybridized with centromeric 11q23 probes. Original magnification of FISH images, $\times 600$.

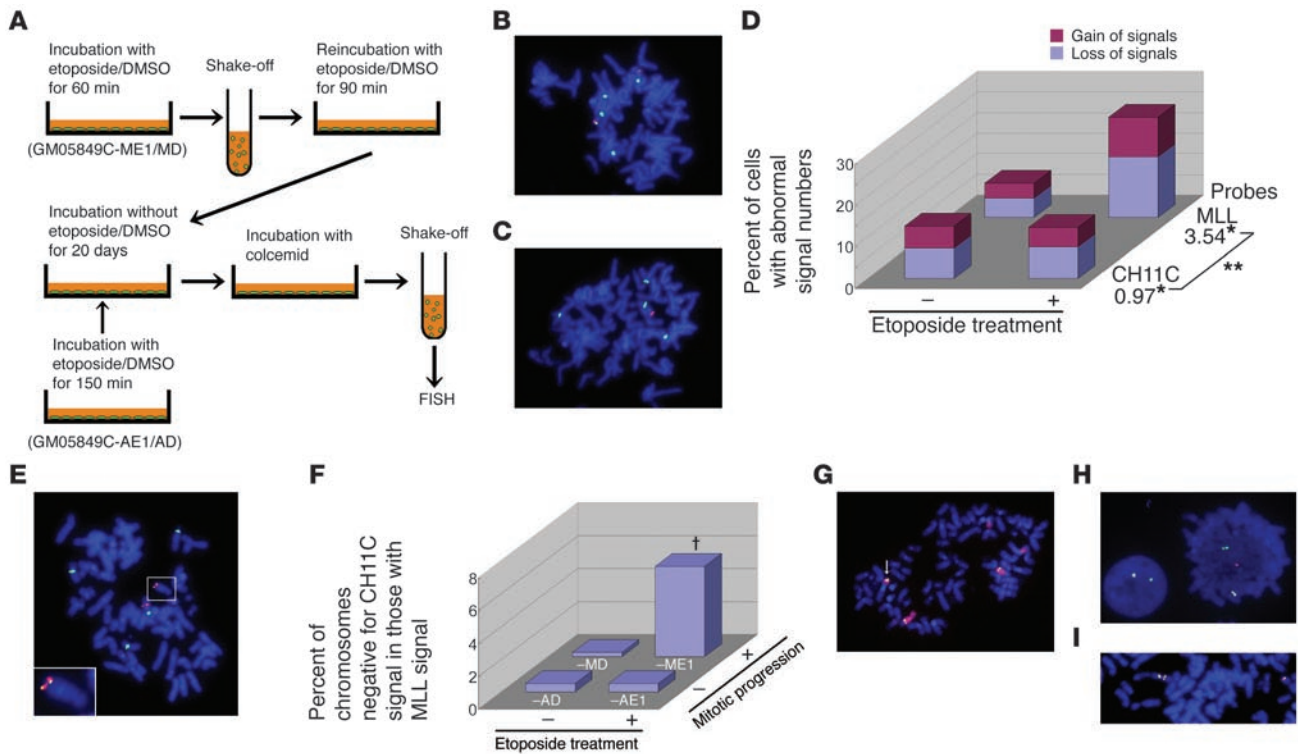


Figure 6

Chromosomal aberrations in a mixture of stable clones of ATM-deficient fibroblasts, which had executed mitosis under etoposide treatment. **(A)** Flow diagram for long-term culture procedure. **(B, C, and E)** Metaphase spreads hybridized with the CH11C probe (green) and MLL probes (green and red overlap). **(B)** One GM05849C-MD cell showing 4 CH11C signals and 2 pairs of MLL signals. **(C)** One GM05849C-ME1 cell showing 4 CH11C signals with only 1 of them bearing MLL signals. **(D)** Proportion of cells with gain and loss of CH11C or MLL signals. $n = 300$ for each. *OR for etoposide treatment. ** $P = 0.0002$, difference in OR for interaction term of probe \times treatment by a logistic regression model. **(E)** One GM05849C-ME1 cell with the *MLL* gene translocated to another chromosome. The inset is a magnified image of the enclosed area. **(F)** Percent of chromosomes negative for CH11C signal among those positive for MLL signals. Data were analyzed by multiple logistic regression. $n = 400$ for each. $^{\dagger}P = 0.028$ for interaction term of etoposide treatment \times mitotic progression. Neither of the pairwise comparisons for etoposide treatment in asynchronous cells nor for mitotic progression in etoposide-untreated cells was statistically significant ($P = 1.0$). **(G)** Representative image of chromosome 11 translocation (arrow) in GM05849C-ME1 cells hybridized with chromosome 11 painting (red) and MLL probes (green and red overlap). **(H and I)** Abnormal *MLL* gene configuration. GM05849C-ME1 cells were hybridized with MLL probes. Chromosomal translocation of *MLL* BCR **(H)** and tandem duplication of the *MLL* gene **(I)**. Original magnification for FISH images, $\times 600$.