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

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Erratum

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Erratum

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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.

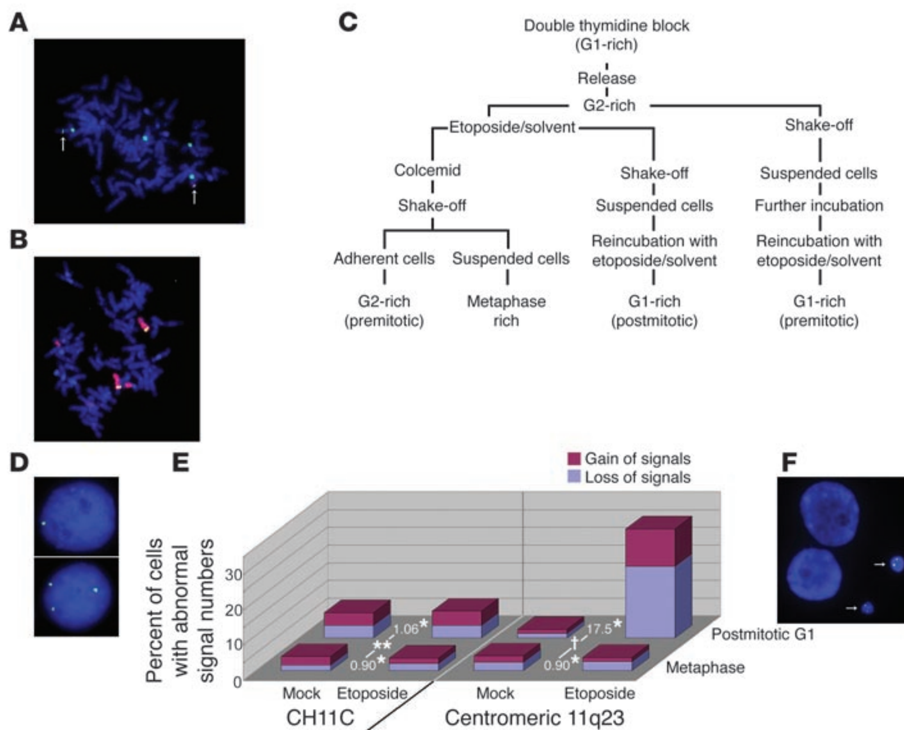

Figure 3

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 $^{\dagger}P < 0.0001$, 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 × 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, ×600.

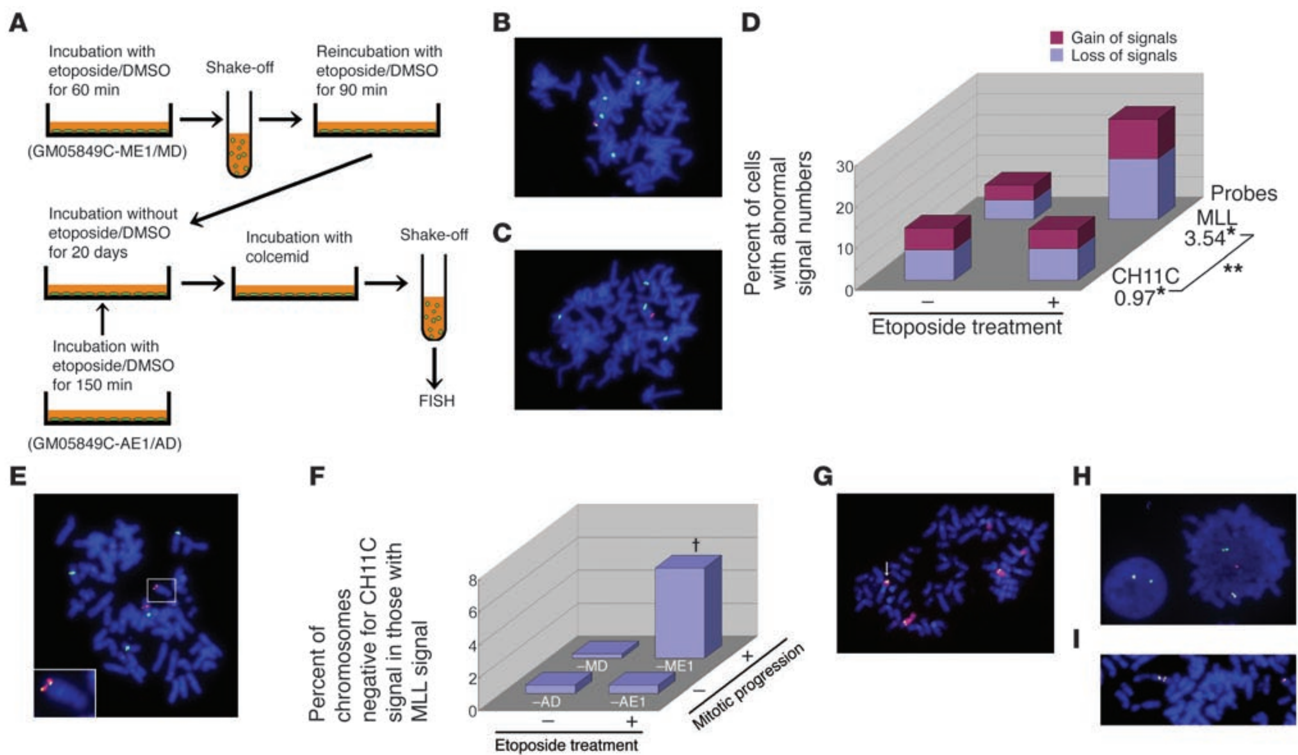

Figure 6

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 × 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. †*P* = 0.028 for interaction term of etoposide treatment × 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, ×600.