Correction to "Poly(ADP-Ribose) Polymerase 1 Modulates the Lethality of CHK1 Inhibitors in Carcinoma Cells"

In the above article [Mitchell C, Park M, Eulitt P, Yang C, Yacoub A, and Dent P (2010) *Mol Pharmacol* **78:**909–917], Figs. 3 to 5 are incorrect because of errors during manuscript processing. The corrected figures and their legends appear below.

The online version of this article has been corrected in departure from the print version.

The printer regrets this error and apologizes for any confusion or inconvenience it may have caused.

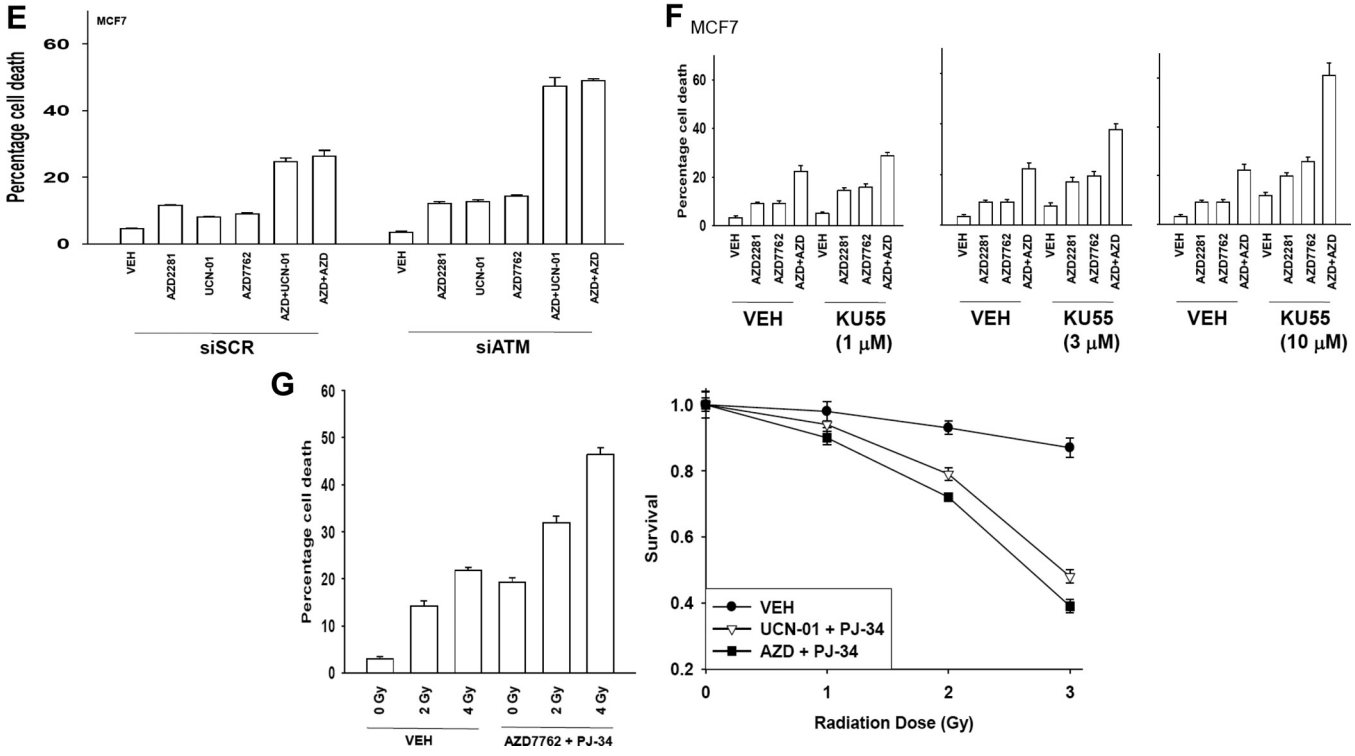

Fig. 3. *Continued.* E, MCF7 cells were transfected with nonspecific siRNA control (siSCR) or an siRNA to knock down ATM (siATM). Twenty-four hours after transfection, cells were treated with vehicle (VEH, DMSO) and/or by AZD7762 (25 nM) or UCN-01 (50 nM). Cells were isolated 48 h after exposure, and viability was determined using trypan blue exclusion. Data for each assay is the mean of all data points from three studies ± S.E.M. F, MCF7 cells were plated in triplicate and treated with vehicle (VEH, DMSO), AZD2281 (0.5 μM), AZD7762 (25 nM), or AZD2281 + AZD7762 in combination. Thirty minutes after exposure, cells are treated with vehicle (DMSO) or with increasing concentrations of the ATM inhibitor 2-(4-morpholinyl)-6-(1-thianthrenyl)-4H-pyran-4-one (KU55933) (1–10 μM). Cells were isolated 48 h after exposure, and viability was determined using trypan blue exclusion. Data for each assay is the mean of all data points from three studies ± S.E.M. G, left, MCF7 cells were plated and treated with vehicle (VEH, DMSO) or the PARP-1 inhibitor PJ34 (3 μM) followed 30 min later by CHK1 inhibitor AZD7762 (25 nM). Cells were irradiated (4 Gy) and used for short-term viability assays 48 h after exposure and for viability determined using trypan blue exclusion. Right, MCF7 cells were plated in sextuplicate as single cells, and 12 h after plating, cells were treated with vehicle (VEH, DMSO) or the PARP-1 inhibitor PJ34 (3 μM) followed 30 min later by CHK1 inhibitors UCN-01 (50 nM) or AZD7762 (25 nM). Cells were irradiated 30 min after drug additions. Forty-eight hours after drug exposure, the media were changed, and cells were cultured in drug-free media for an additional 10 to 14 days ($n = 2 \pm$ S.E.M.).

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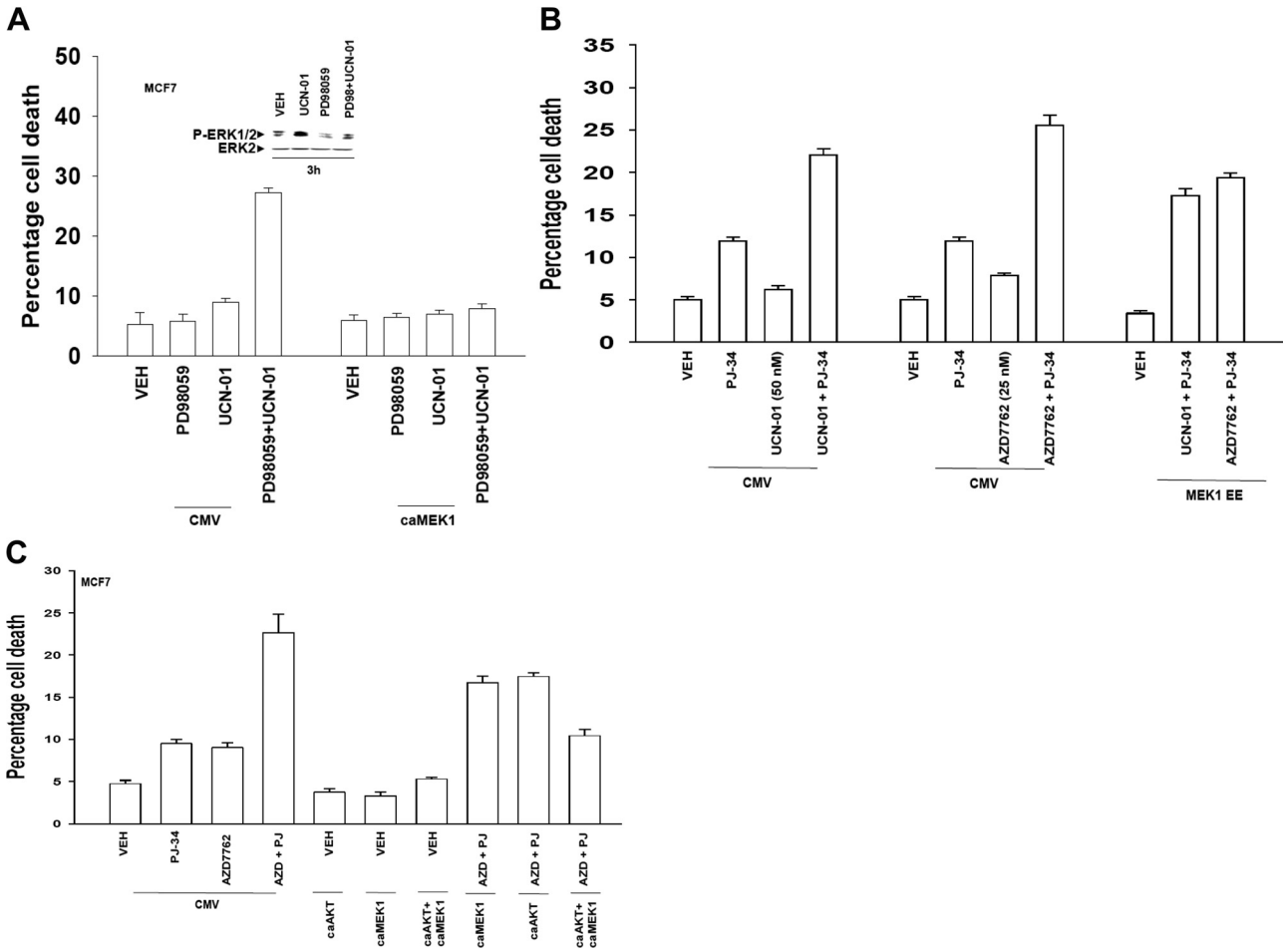

Fig. 4. Inhibition of CHK1 inhibitor-induced ERK1/2 activation is not the sole molecular mechanism of drug interaction. A, MCF7 cells were infected in triplicate at an m.o.i. of 50 with either an empty vector adenovirus (CMV) or with an adenovirus to express constitutively activated MEK1 EE. Twenty-four hours after infection, cells were treated with vehicle (VEH, DMSO), PD98059 (25 μM), or UCN-01 (50 nM) as indicated. Cells were isolated 48 h after exposure, and viability was determined using trypan blue exclusion. Data for each assay are the means of all data points from three studies ± S.E.M. B, MCF7 cells were infected in triplicate at an m.o.i. of 50 with either an empty vector adenovirus (CMV) or with an adenovirus to express constitutively activated MEK1 EE. Twenty-four hours after infection, cells were treated as indicated with vehicle (VEH, DMSO), PJ34 (3 μM), UCN-01 (50 nM), or AZD7762 (25 nM). Cells were isolated 48 h after exposure, and viability was determined using trypan blue exclusion. Data for each assay are the means of all data points from three studies ± S.E.M. C, MCF7 cells were infected in triplicate at an m.o.i. of 50 with either an empty vector adenovirus (CMV) or with adenoviruses to express constitutively activated MEK1 EE and/or constitutively activated AKT. Twenty-four hours after infection, cells were treated as indicated with vehicle (VEH, DMSO), PJ34 (3 μM), and/or AZD7762 (25 nM) as indicated. Cells were isolated 48 h after exposure, and viability was determined using trypan blue exclusion. Data for each assay are the means of all data points from three studies ± S.E.M.

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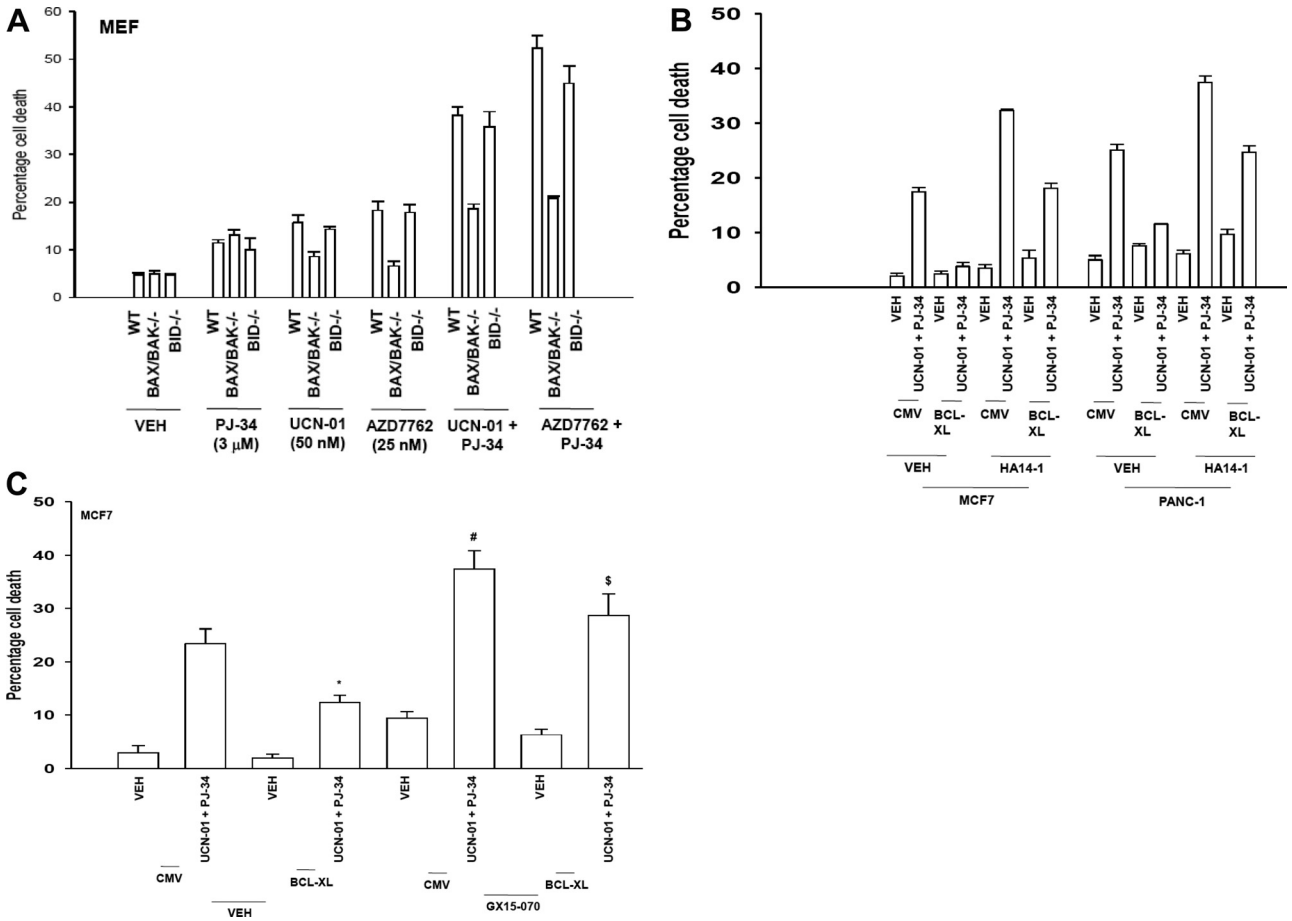

Fig. 5. Loss of BAX/BAK function abolishes the toxic interaction between CHK1 inhibitors and PARP-1 inhibitors; cell killing is potentiated by inhibitors of BCL-2/BCL-xL function. A, transformed mouse embryonic fibroblasts [MEF; wild type, WT; deleted for BAX and BAK, BAX/BAK(−/−); deleted for BID, BID(−/−)] were plated in triplicate and treated with vehicle (VEH, DMSO), PJ34 (3 μM), UCN-01 (50 nM), or AZD7762 (25 nM). Cells were isolated 48 h after exposure, and viability was determined using trypan blue exclusion. Data for each assay are the means of all data points from three studies ± S.E.M. B, PANC-1 and MCF7 cells were infected with either an empty vector adenovirus (CMV) or with an adenovirus to express BCL-XL. Twenty-four hours after infection, cells were pretreated for 30 min with vehicle (VEH, DMSO) or HA14-1 (10 μM) and then treated as indicated with vehicle (VEH, DMSO) or PJ34 (3 μM) and UCN-01 (50 nM). Cells were isolated 48 h after exposure, and viability was determined in triplicate using trypan blue exclusion. Data for each assay are the means of all data points from two studies ± S.E.M. C, MCF7 cells were infected with either an empty vector adenovirus (CMV) or with an adenovirus to express BCL-XL. Twenty-four hours after infection, cells were pretreated for 30 min with vehicle (VEH, DMSO) or obatoclax (GX15-070, 50 nM) and then treated as indicated with vehicle (VEH, DMSO) or PJ34 (3 μM) and UCN-01 (50 nM). Cells were isolated 48 h after exposure, and viability was determined in triplicate using trypan blue exclusion. Data for each assay is the mean of all data points from two studies ± S.E.M. *, $p < 0.05$ less than corresponding value in empty vector virus-infected cells; #, $p < 0.05$ greater than corresponding value in empty vector-infected cells not treated with obatoclax; $, greater than corresponding value in BCL-xL-infected cells treated with obatoclax.

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