SUPPLEMENTAL MATERIAL

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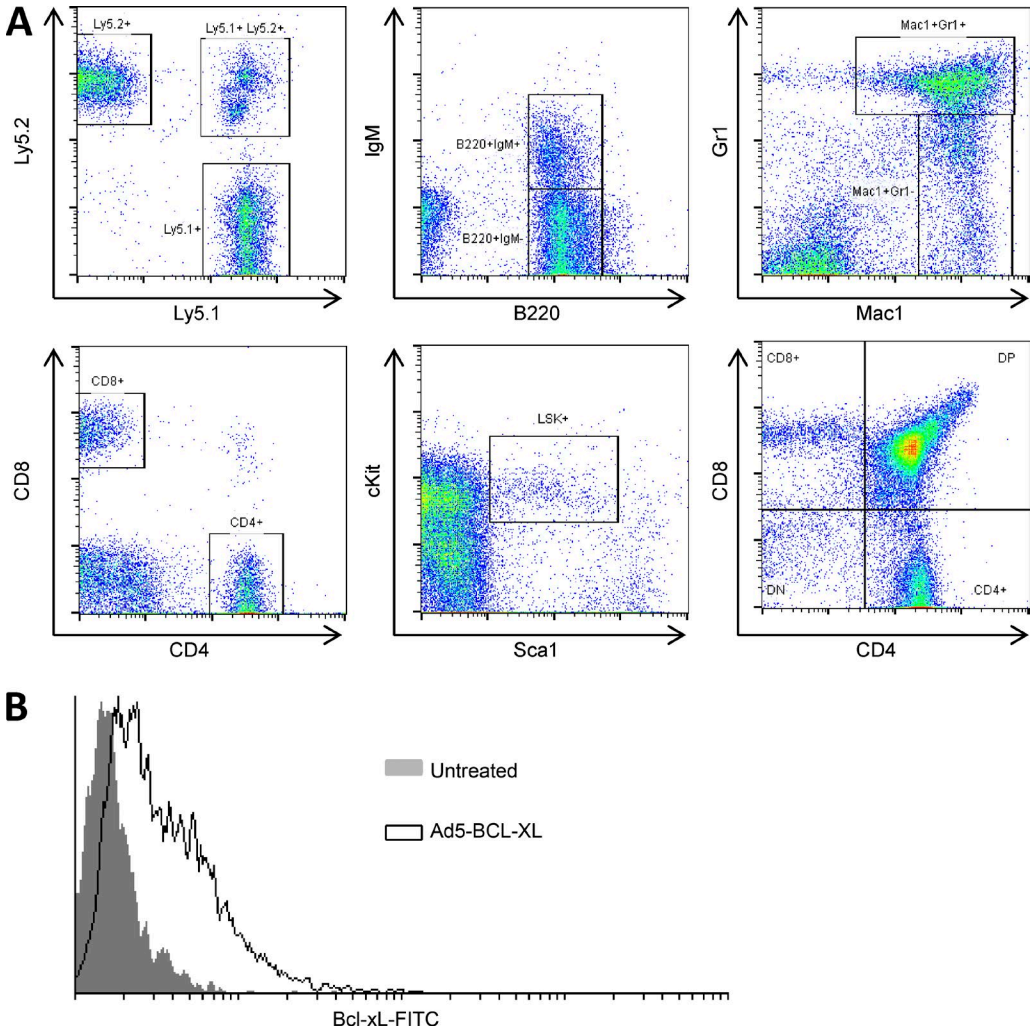

Figure S1. **Gating strategy used for analysis of recipient mice.** (A) Gating strategy. (B) Adenovirally overexpressed BCL-CL is measured by anti–Bcl-xL antibodies in transduced LSK cells.

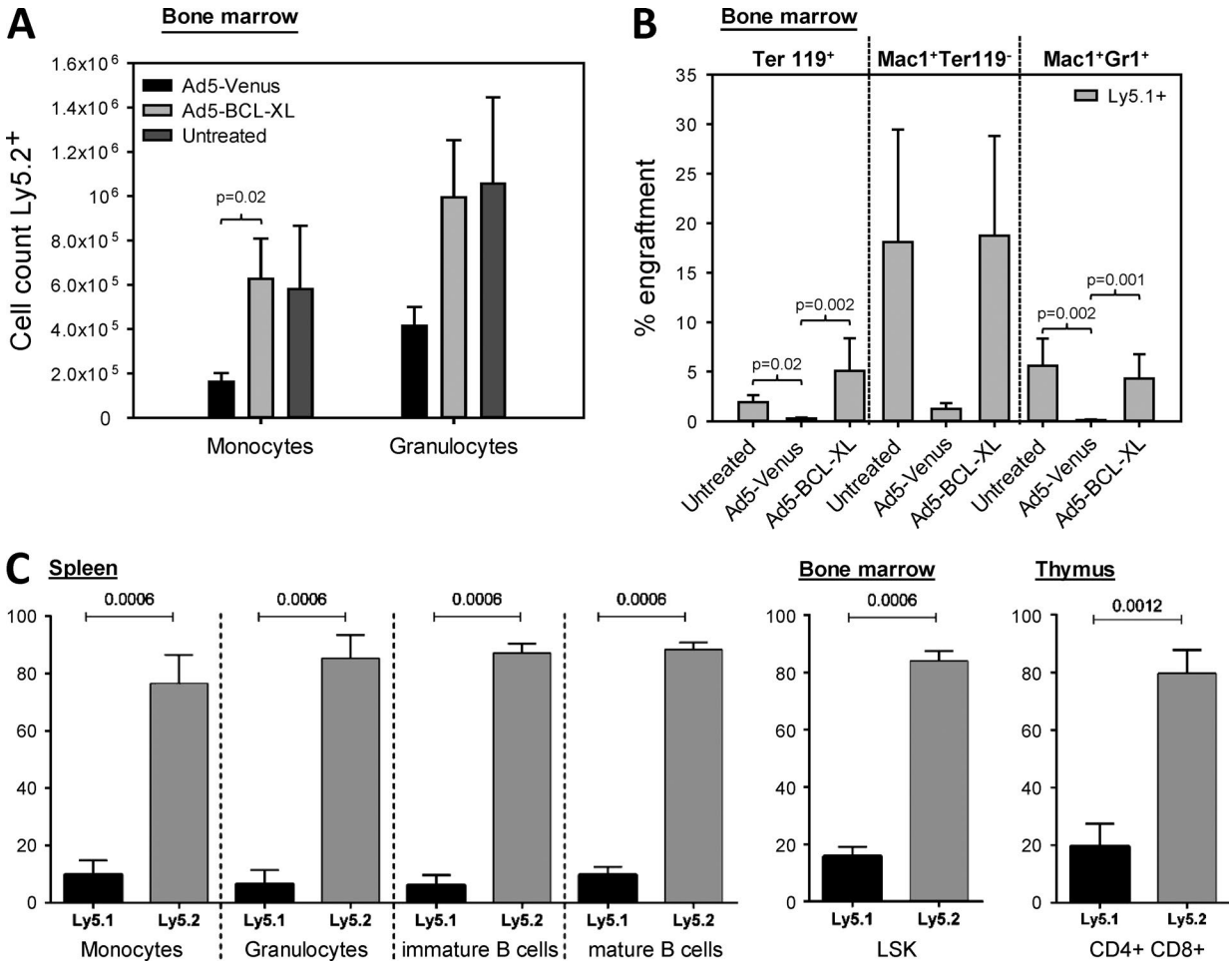

Figure S2. **Competitive reconstitution assays using LSK cells infected with adenoviruses.** (A) Ly5.1$^+$/Ly5.2$^+$ recipient mice were competitively transplanted with Ad5-Venus–treated Ly5.1$^+$ and Ad5-Venus–treated, Ad5–BCL-XL–treated or untreated Ly5.2$^+$ LSK cells in a 1:1 ratio. The experimental scheme is depicted in Fig. 2 A. 10 d after transplantation, recipient mice were sacrificed, and cell numbers of Ly5.2$^+$ monocytes and granulocytes within the BM were determined based on organ cellularity and flow cytometry. Bars represent means of $n = 6–8$ animals of two independent experiments ± SEM (Mann-Whitney test). (B) Ly5.2$^+$ LSK cells were transduced with Ad5-Venus adenoviruses, whereas Ly5.1$^+$ LSK cells were transduced as indicated (untreated, Ad5-Venus, Ad5–BCL-XL). Ly5.1$^+$ and Ly5.2$^+$ LSK cells were transplanted in a 1:1 ratio into lethally irradiated Ly5.2$^+$ *rag1*$^{-/-}$ mice. 10 d later, recipient mice were sacrificed, and the percentage of Ly5.1+ cells were determined within the BM by flow cytometry. Bars represent means of $n = 7–8$ animals of two independent experiments ± SEM. P-values were calculated using the Mann-Whitney test. (C) Ly5.1$^+$/Ly5.2$^+$ recipient mice were competitively transplanted with untreated Ly5.1$^+$ and BCL-XL–treated Ly5.2$^+$ LSK cells in a 1:1 ratio. 16 wk after transplantation, recipient mice were sacrificed, and Ly5.1$^+$ and Ly5.2$^+$ chimerism was determined in the indicated organs and cell types. Bars represent means of $n = 7$ animals of two independent experiments ± SEM (Mann-Whitney test).

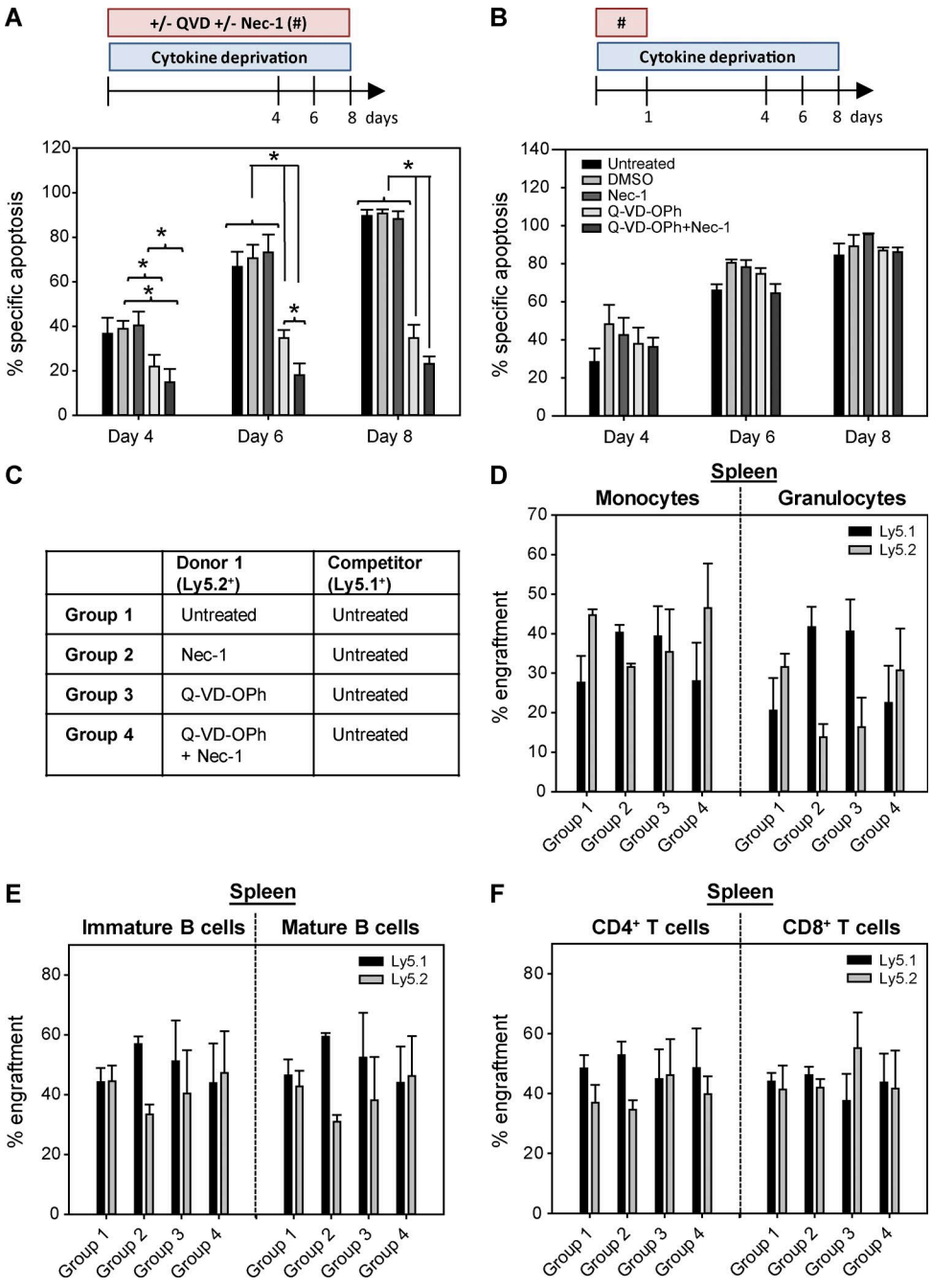

Figure S3. **In vitro and in vivo experiments using LSK cells treated with Q-VD-OPh and/or Nec1.** (A) LSK cells were treated with or without Q-VD-OPh and/or Nec-1 (#) and subjected to cytokine withdrawal. Specific apoptosis was determined by Annexin V/7-AAD staining at the indicated time points ($n = 4$; Mann-Whitney test). The legend is shown in B. (B) In a similar experimental setup, LSK cells were only treated for 24 h with Q-VD-OPh and/or Nec-1 (#) but permanently subjected to cytokine withdrawal ($n = 4$; Mann-Whitney test). (C) Ly5.2$^+$ LSK cells were pretreated with Q-VD-OPh and/or Nec-1 for 24 h and transplanted together with untreated Ly5.1$^+$ LSK cells (1:1 ratio) into irradiated Ly5.1/Ly5.2 heterozygous mice. (D–F) 16 wk later, recipient mice were sacrificed, and donor chimerism was determined within the indicated cell populations by flow cytometry. Bars represent means of $n = 2–3$ per group of one experiment ± SEM. *, $P \leq 0.05$.

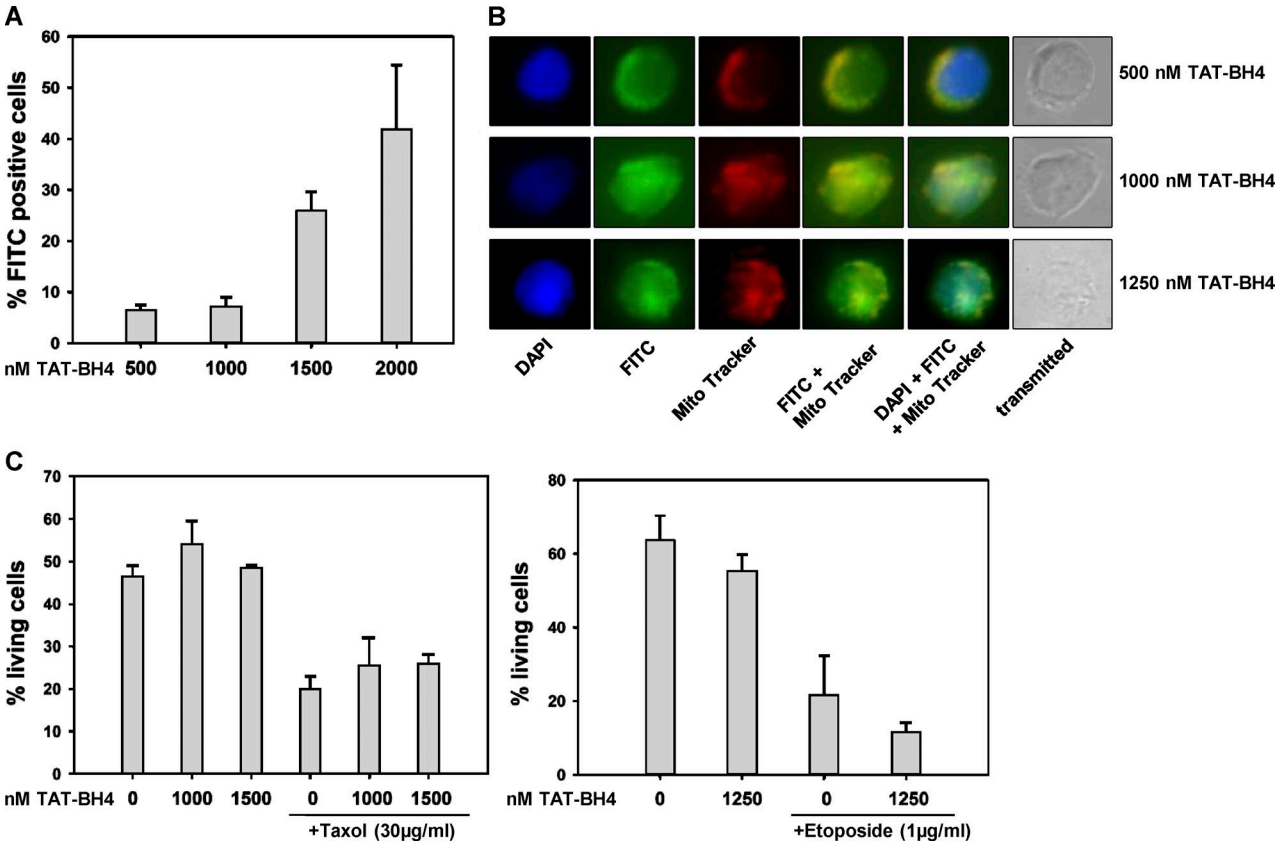

Figure S4. **In vitro experiments using LSK cells treated with TAT–BH4.** (A) WT LSK cells were isolated, pretreated with dextran sulfate and transduced with FITC-TAT-BH4 at the indicated concentrations (12 h). Percentage of FITCTAT-BH4–positive cells was evaluated by flow cytometry. (B) Localization of FITC-TAT-BH4 was investigated by fluorescence microscopy using DAPI and MitoTracker. (C) LSK cell were transduced with TAT-BH4 peptide or left untreated and, in addition, subjected to taxol (left) or etoposide for 24 h (right). Percentage of living cells was determined by flow cytometry.

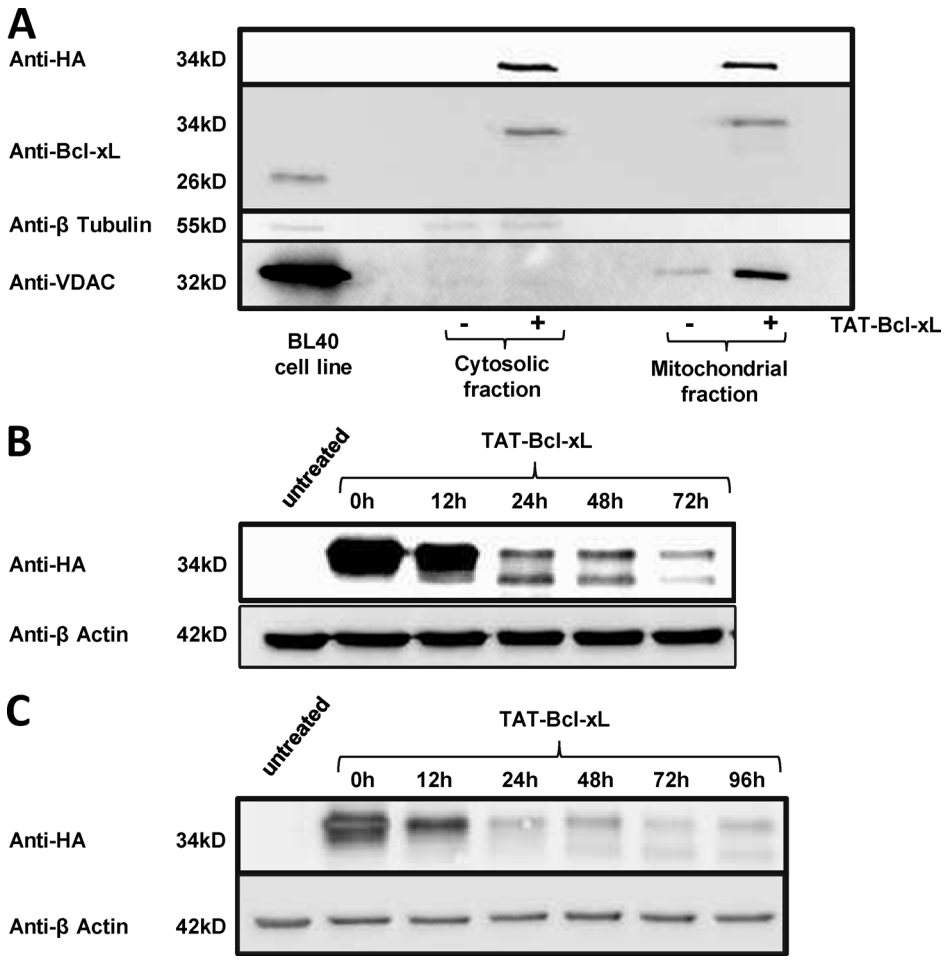

Figure S5. **Localization and degradation of the TAT–BCL–XL protein in HeLa cells.** (A) HeLa cells were transduced with TAT–Bcl-xL protein.24 h later, mitochondrial and cytosolic fractions were separated via SDS-PAGE, and TAT–Bcl-xL protein contents were detected using anti–Bcl-xL and anti-HA antibodies. BL40 cell line cells were used as a positive control. As a loading control, anti–β-Tubulin and anti-VDAC antibodies were used. (B) HeLa cells were transduced with TAT–Bcl-xL protein and treated with Q-VD-OPh (50 µM) and cycloheximide (100 µg/ml). At the indicated time points, protein lysates were generated. An anti-HA antibody was used for detection of TAT–Bcl-xL in order to avoid interference with endogenous Bcl-xL protein. (C) HeLa cells treated as in B were additionally treated with the proteasomal inhibitor MG132 (10 µM).