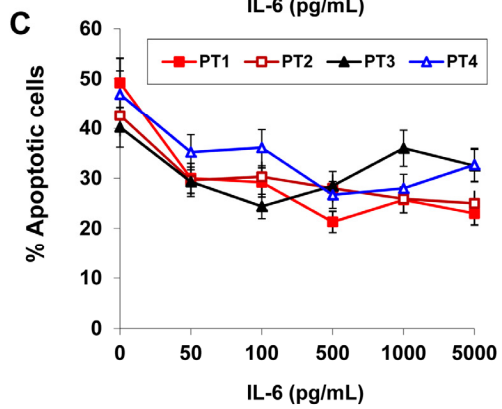
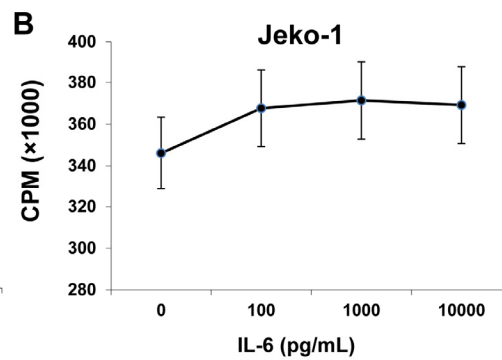
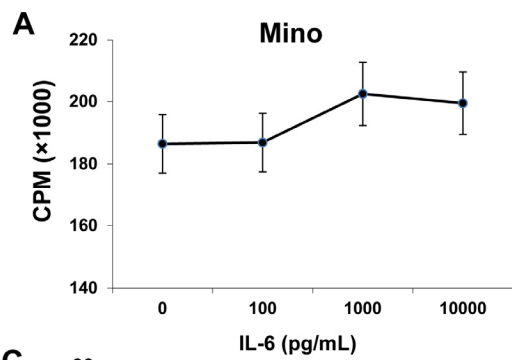


Supplemental Materials

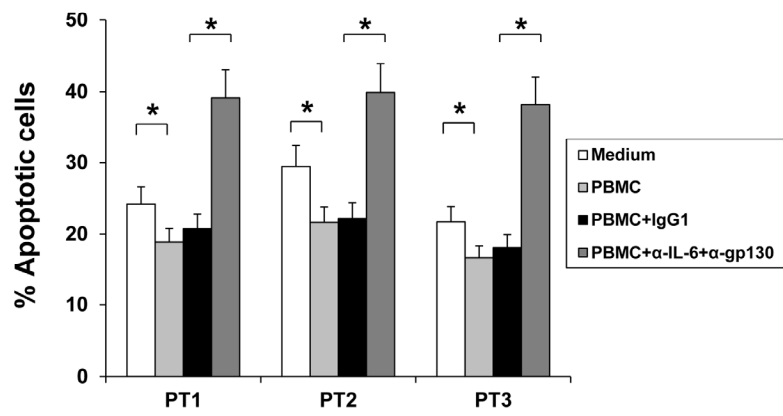
Supplemental Figure 1. Effects of exogenous IL-6 on the growth and spontaneous apoptosis of MCL cells. Proliferation of IL-6⁻ Mino (A) and Jeko-1 (B) cells, detected by ³H-thymidine incorporation assay, in the presence of exogenous IL-6 (0-10000 pg/mL) in a 48-hour culture. (C) Apoptosis, examined by Annexin V-binding assay, of primary MCL cells from 4 patients (PT1-PT4) in ex vivo, 48-hour cultures in the presence or absence of exogenous IL-6 (0-5000 pg/mL).

Supplemental Figure 2. PBMCs partially protect primary MCL cells against ex vivo spontaneous apoptosis. Freshly isolated primary MCL cells from 3 patients (PT1-PT3) were cocultured for 24 hours with or without PBMCs from healthy blood donors in transwell inserts in with or without the addition of IL-6-neutralizing (α -IL-6; 50 μ g/mL) or control IgG1. After the culture, PBMCs in transwell inserts were removed and MCL cells in the culture wells were subjected to Annexin V-binding assay. * $P < 0.01$.

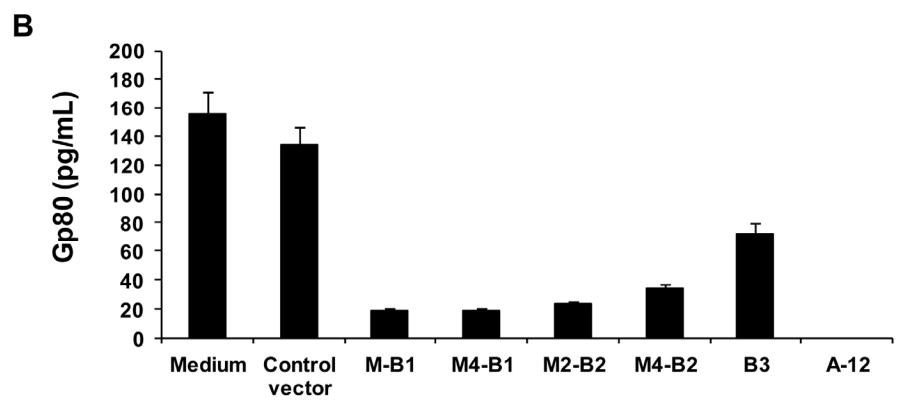
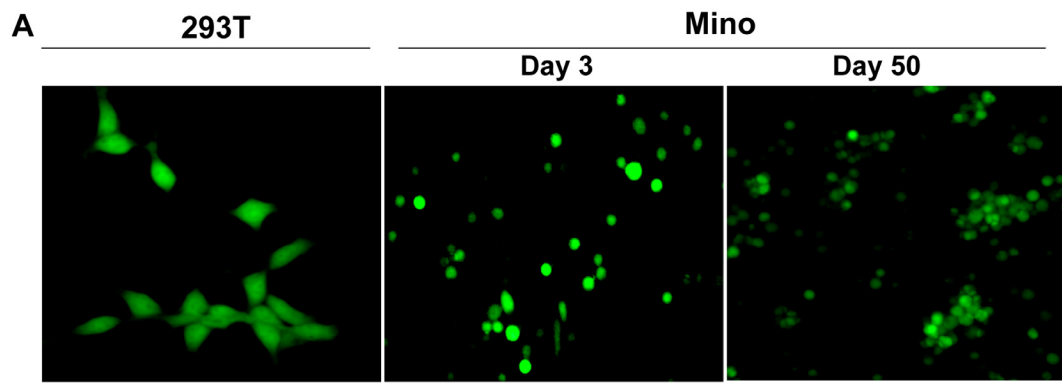
Supplemental Figure 3. Stable knockdown of gp80 in Mino cells. (A) Fluorescence images of 293T and Mino cells at day 3 or day 50 after transfection. Recombinant lentivirus vectors were produced by 293T cells. GFP expression as a control for stable transfection of vector was detected under fluorescence microscope in both 293T cells on day 3 and Mino cells on day 3 and day 50. (B) ELISA showing the knockdown of gp80 in Mino cells. Five shRNA-gp80 lentiviral vectors named M-B1, M4-B1, M2-B2, M4-B2, B3, and A12 were transfected to the cells. Gp80 was completely knocked down in A12-transfected Mino cells, which were used in experiments shown in Figure 4B.



Supplemental Figure 1



Supplemental Figure 2



Supplemental Figure 3