Supplemental material

Supplemental table 1: qPCR primer sequence

CXCL12α forward	CCAAACTGTGCCCTTCAGAT
CXCL12α reverse	CGTCTTTGCCCTTTCATCTC
CXCL12γ forward	CCAAACTGTGCCCTTCAGAT
CXCL12γ reverse	CTTTTCTGGGCAGCCTTTCT
RPLPO forward	GCTTCCTGGAGGGTGTCCGC
RPLPO reverse	TCCGTCTCCACAGACAAGGCCA

Supplemental figure legends

Figure 1. CXCL12 α mRNA expression in HS5-WT and HS5-CXCL12 γ KO cells. A representative for 2 independent experiments. ns, P > 0.05 using unpaired student's t-test.

Figure 2. MTT assay analysis of the growth of HS5-WT, HS5-CXCL12γKO and HS5-EXT1KO cells. Mean ±SD of three independent experiments in triplicate is shown.

Figure 3. (A) Adhesion of the HMCL L363 to HS5-WT and two HS5-CXCL12 γ KO clones. Mean ±SD of three independent experiments in triplicate. *, P ≤ 0.05 using one-way ANOVA analysis. **(B)** Adhesion of the HMCL L363 to HS5-WT and HS5-EXT1KO. Mean ±SD of 3 independent experiments in triplicate. *, P ≤ 0.05 using unpaired student's t-test.

Figure 4. (A) Flow cytometric discrimination of MM cells from BMSCs expressing GFP. **(B)** BMSC viability is not affected by bortezomib. HS5 cells were co-cultured with XG1 cells in the presence of bortezomib at indicated concentrations for 3 days. HS5 viability was analyzed by flow cytometry.

Figure 5. CXCL12 γ mediates carfilzomib resistance in XG1 cells. XG1 was cultured alone, or co-cultured with HS5-WT, HS5-CXCL12 γ KO or HS5-EXT1KO, in the presence carfilzomib for 3 days. Mean ±SD of 3 independent experiments in triplicate. *, P ≤ 0.05; **, P ≤ 0.01 using one-way ANOVA analysis.

Figure 6. Recombinant CXCL12 γ (or CXCL12 α) induced adhesion to VCAM-1 does not protect HMCLs from bortezomib-induced cell death. The HMCLs XG1 or MM1.S were cultured on a surface co-coated with CXCL12 γ or CXCL12 α and VCAM-1 in the presence

of bortezomib for 3 days. Cell viability was analyzed by flow cytometry. A representative plot for 2 independent experiment is shown.

Supplemental data

