**Supplementary data**

**Supplementary Table S1. List of multiple myeloma patients.**

**Supplementary Table S2. List of extranodal marginal zone B-cell lymphoma patients.**

**Supplementary Table S3. List of MGUS/SMM patients.**

**Supplementary Table S4. List of patients with bone marrow involved B-cell lymphoma.**

**Supplementary Figure S1. Gating strategy for BM CD8+T cells in flow cytometric analysis of BMMCs.**

**Supplementary Figure S2. Gating strategy for malignant plasma cells and normal plasma cells in flow cytometric analysis of BMMCs from MM patients.**

**Supplementary Figure S3. Gating strategy for immune cell subsets in flow cytometric analysis of BMMCs.**

**Supplementary Figure S4. Phenotypic characteristics of IM-9 myeloma cells.** (A-B) Expression of HLA-A2 (A) and PD-L1 (B) in IM-9 cells.

**Supplementary Figure S5. Establishment of NY-ESO-1157-165-specific CD8+ T cells.** (A) Purity of an NY-ESO-1157-165-specific CD8+ T cell line. (B) Expression of PD-1 in NY-ESO-1157-165-specific CD8+ T cells which were cocultured with NY-ESO-1157-165–pulsed IM-9 cells for 72 hours.

**Supplementary Figure S6. Frequency of myeloma antigen-specific CD8+ T cells from BM of EMZL, MGUS, SMM, and MM patients.** (A-B) BMMCs from newly diagnosed EMZL (n=10), MGUS (n=4), SMM (n=2), or MM (n=29 for NY-ESO-1157-165, n=12 for HM1.2422-30) patients with HLA-A2+ were stained with HLA-A\*0201 NY-ESO-1157-165 and HM1.2422-30 dextramers and analyzed by flow cytometry. The relative frequency of NY-ESO-1157-165-specific (A) and HM1.2422-30-specific (B) cells among total BM CD8+ T cells was compared. Error bars represent SD. Statistical analysis was performed using the Mann-Whitney U-test. \*, *P* < 0.05; \*\*\*\*, *P* < 0.0001.

**Supplementary Figure S7. Expression of PD-L1 on malignant cells and immune cells from the BM of DLBCL and HL with BM involvement.** (A) The relative frequency of PD-L1+ cells was analyzed in malignant plasma cells among BMMCs from MM patients (n=38), malignant B cells in DLBCL (n=10, CD45+CD19+CD20+κ or λ LC+), or Reed-Stenberg cells in HL (n=2, CD15+CD30+) with BM involvement. (B) The relative frequency of PD-L1+ cells was analyzed in CD11c+ dendritic cells (CD45+CD14-CD3-CD11c+HLA-DR+, left), B cells (CD45+CD14-CD3-CD19+HLA-DR+, middle), and monocytes (CD45+CD3-CD14+, right) among BMMCs from MM patients (n=38), DLBCL with BM involvement (n=10), and HL with BM involvement (n=2). Error bars represent SD. N.S., not significant. Statistical analysis was performed using the Mann-Whitney U-test. \*\*, *P* < 0.01.

**Supplementary Figure S8. Impaired proliferation of BM CD8+ T cells from MM patients.** (A-C) PBMCs from normal healthy donors (n=10) or MM patients (n=10), or BMMCs from EMZL patients (n=11) were labeled with CTV and stimulated with anti-CD3 (1 ng/mL) for 108 hours. The mitotic index of CD8+ T cells of PBMCs from normal healthy donors (A) or MM patients (B), or BMMCs from EMZL patients (C) was compared with that of BM CD8+ T cells from MM patients (n=22). Error bars represent SD. Statistical analysis was performed using the Mann-Whitney U-test. \*\*\*\*, P < 0.0001.

**Supplementary Figure S9. Effect of combined blockade of PD-1 and other immune checkpoint receptors on the proliferation of BM CD8+ T cells from MM patients.** (A-B) BMMCs from MM patients (n=14) were labeled with CTV and stimulated with anti-CD3 (1 ng/mL) in the presence of anti-PD-1 and/or other immune checkpoint receptor inhibitors such as anti-Tim-3, anti-Lag-3, and anti-TIGIT blocking antibodies, or isotype control antibody (5 μg/mL for each antibody) for 108 hours. Representative histograms are presented (A). Lines represent the data from each patient. N.S., not significant. Statistical analysis was performed using Wilcoxon matched-pairs signed rank test.

**Supplementary Figure S10. Effect of PD-1 blockade on the proliferation of BM CD8+ T cells from EMZL, MGUS, and SMM patients.** (A-D) BMMCs from HLA-A2+ EMZL (n=8), MGUS (n=4), or SMM (n=3) patients were labeled with CTV and stimulated with a mix of NY-ESO-1157-165 (SLLMWITQC) and HM1.2422-30 (LLLGIGILV) peptides in the presence of anti-PD-1 or isotype control antibody for 144 hours. CD8+ T cell proliferation was compared between isotype control antibody- and anti-PD-1 antibody-treated BMMCs from EMZL patients (A and B) or MGUS/SMM patients (C and D). Representative figures are presented (B and D). Lines represent the data from each patient. N.S., not significant. Statistical analysis was performed using Wilcoxon matched-pairs signed rank test.

**Supplementary Figure S11. Effect of TGFβ on BM CD8+ T cells.** (A-B) BM CD8+ T cells from MM patients (n=6) were purified and stimulated with plate-bound anti-CD3 (1 μg/mL), anti-CD28 (1 μg/mL), and PD-L1 (10 μg/mL) in the presence or absence of TGFβ1 (50 ng/mL) for 30 hours to measure the expression of PD-1 (A) and T-bet (B). Lines represent the data from each patient. Statistical analysis was performed using Wilcoxon matched-pairs signed rank test. \*, *P* < 0.05; \*\*, *P* < 0.01.

**Supplementary Figure S12. Proliferation capacity of BM CD8+ T cells from MM patients treated with anti-PD-1 and TGFβ inhibitors.** (A-D) BMMCs from MM patients (n=9) were labeled with CTV and stimulated with anti-CD3 (1 ng/mL) in the presence of anti-PD-1 and TGFβ inhibitors for 108 hours such as anti-TGFβ1 neutralizing antibody or galunisertib. PBMCs from normal healthy donors (A; n=10) or MM patients (B; n=10), or BMMCs from EMZL patients (C; n=11) or MGUS/SMM patients (D; n=5) were labeled with CTV and stimulated with anti-CD3 (1 ng/mL) for 108 hours. Error bars represent SD. Statistical analysis was performed using the Mann-Whitney U-test. \*, *P* < 0.05; \*\*, *P* < 0.01; \*\*\*, *P* < 0.001.

**Supplementary Figure S13. Effect of combined blockade of PD-1 and TGFβ on the proliferation of BM CD8+ T cells from EMZL, MGUS, and SMM patients and PB CD8+ T cells from normal heathy donors and MM patients.** (A-H) BMMCs from EMZL (A-B; n=11) or MGUS/SMM patients (C-D; n=6) and PBMCs from normal healthy donors (E-F; n=10) or MM patients (G-H; n=10) were labeled with CTV and stimulated with anti-CD3 (1 ng/mL) in the presence of anti-PD-1, TGFβ inhibitors such as anti-TGFβ1 neutralizing antibody or galunisertib, or isotype control antibody for 108 hours. Lines represent the data from each patient. N.S., not significant. Statistical analysis was performed using Wilcoxon matched-pairs signed rank test.

**Supplementary Figure S14. Effect of combined blockade of PD-1 and TGFβ on the production of effector cytokine production in BM CD8+ T cells from MM patients.** (A-B) BMMCs from MM patients (n=7) were stimulated with anti-CD3 (1 ng/mL) in the presence of anti-PD-1, TGFβ inhibitors such as anti-TGFβ1 neutralizing antibody or galunisertib, or isotype control antibody for 36 hours. Then Brefeldin A and monensin were added 24 hours after incubation to measure effector cytokine production. The ratio of IFNγ+TNF+ cells with and without anti-PD-1 and TGFβ inhibitors was calculated as follows: (IFNγ+TNF+ cells in anti-PD-1 and TGFβ inhibitors-treated BM CD8+ T cells) / (IFNγ+TNF+ cells in control BM CD8+ T cells). Representative figures are presented (A). Lines represent the data from each patient. N.S., not significant. Statistical analysis was performed using Wilcoxon matched-pairs signed rank test. \*, *P* < 0.05.

**Supplementary Figure S15. Effect of anti-PD-1 antibody and/or TGFβ inhibitors on the proliferation of BM CD8+ T cells from MM patients without antigen stimulation.** (A-B) BMMCs from HLA-A2+ MM patients (n=5) were labeled with CTV and incubated in the presence of anti-PD-1, TGFβ inhibitors such as anti-TGFβ1 neutralizing antibody (A) or galunisertib (B), or isotype control antibody for 144 hours. Lines represent the data from each patient. N.S., not significant. Statistical analysis was performed using Wilcoxon matched-pairs signed rank test.

**Supplementary Figure S16. Effect of anti-PD-1 antibody and/or TGFβ inhibitors on the proliferation of BM CD8+ T cells from EMZL, MGUS, and SMM patients.** (A-D) BMMCs from HLA-A2+ EMZL patients (A-B; n=8) or HLA-A2+ MGUS/SMM patients (C-D; n=7) were labeled with CTV and stimulated with a mixture of NY-ESO-1157-165 (SLLMWITQC) and HM1.2422-30 (LLLGIGILV) peptides in the presence of anti-PD-1, TGFβ inhibitors or isotype control antibody for 144 hours. Lines represent the data from each patient. N.S., not significant. Statistical analysis was performed using Wilcoxon matched-pairs signed rank test.

**Supplementary Figure S17. Proliferation of BM CD8+ T cells from MM patient without KRAS G12D mutation upon stimulation with KRAS peptides.** (A-E) BMMCs from MM patients (n=7) without KRAS G12D mutation were labeled with CTV and stimulated with a mixture of 9 overlapping peptides (9-mer) spanning KRAS codon 12 of wild-type (YKLVVVGAGGVGKSALT; B and C) or G12D mutant-type (YKLVVVGADGVGKSALT; D and E) in the presence of anti-PD-1, TGFβ inhibitors such as anti-TGFβ1 neutralizing antibody or galunisertib, or isotype control for 144 hours. Lines represent the data from each patient. N.S., not significant. Statistical analysis was performed using Wilcoxon matched-pairs signed rank test.

**Supplementary Figure S18. Expansion of myeloma antigen- or viral antigen-specific BM CD8+ T cells.** (A-I) BMMCs from HLA-A2+ MM patients were stimulated with NY-ESO-1157-165 (SLLMWITQC) (A-C; n=13), HM1.2422-30 (LLLGIGILV) (D-F; n=10), or HCMV pp65495-503 (NLVPMVATV) (G-I; n=14) in the presence of anti-PD-1, TGFβ inhibitors such as anti-TGFβ1 neutralizing antibody or galunisertib, or isotype control for 21 days to evaluate the expansion of antigen-specific CD8+ T cells. The frequency of peptide-specific cells among total CD8+ T cells was determined by MHC-I dextramer staining. Representative figures are presented (A, D, and G). Lines represent the data from each patient. Error bars represent SD. N.S., not significant. Statistical analysis was performed using Wilcoxon matched-pairs signed rank test. \*\*, *P* < 0.01; \*\*\*, *P* < 0.001.

**Supplementary Figure S19. Coculture assays with NY-ESO-1157-165-specific PD-1+CD8+ T cells and IM-9 target cells.** (A-F) Cytotoxic activity (A-C) and effector cytokine production (D-F) of NY-ESO-1157-165-specific PD-1+CD8+ T cells against IM-9 cells pulsed with NY-ESO-1157-165 peptide in the presence of anti-PD-1, TGFβ inhibitors such as anti-TGFβ1 neutralizing antibody (B and E) or galunisertib (C and F), or isotype control during 6 hours. Representative figures are presented (A and D). Error bars represent SD. Statistical analysis was performed using Wilcoxon matched-pairs signed rank test. \*\*, *P* < 0.01.

**Supplementary Figure S20. Another set of experiments for a mouse model of MOPC315.BM plasmacytoma.** (A) In addition to the experiment presented in Fig. 6L, independent set of experiments for a mouse model of MOPC315.BM plasmacytoma was performed. MOPC315.BM myeloma cells were subcutaneously injected into Balb/c mice (n=4 for each group), and isotype control antibodies, anti-PD-1 antibodies, anti-TGFβ antibodies, or a combination of anti-PD-1 with anti-TGFβ antibodies were administered every three days from 11 days after tumor injection. Tumor volume was measured 20 days after tumor injection. Error bars represent SD. Statistical analysis was performed using the Mann-Whitney U-test. P-values were indicated. \*, *P* < 0.05.

**Supplementary Figure S21. CD8+ T cell proliferation in the presence of myeloma antigen peptides in BMMC cultures.** (A-B)BMMCs from HLA-A2+ EMZL (n=8), MGUS (n=4), or SMM (n=3) patients were labeled with CTV and stimulated with a mixture of NY-ESO-1157-165 (SLLMWITQC) and HM1.2422-30 (LLLGIGILV) peptides for 144 hours. CD8+ T cell proliferation was calculated by subtracting the frequency of CTV- cells in unstimulated BMMCs from that in peptide-stimulated BMMCs among CD8+ T cells and compared between EMZL and MM patients (A) or between MGUS/SMM and MM patients (B). Error bars represent SD. Statistical analysis was performed using the Mann-Whitney U-test. \*, *P* < 0.05; \*\*\*, *P* < 0.001.