Fig S1

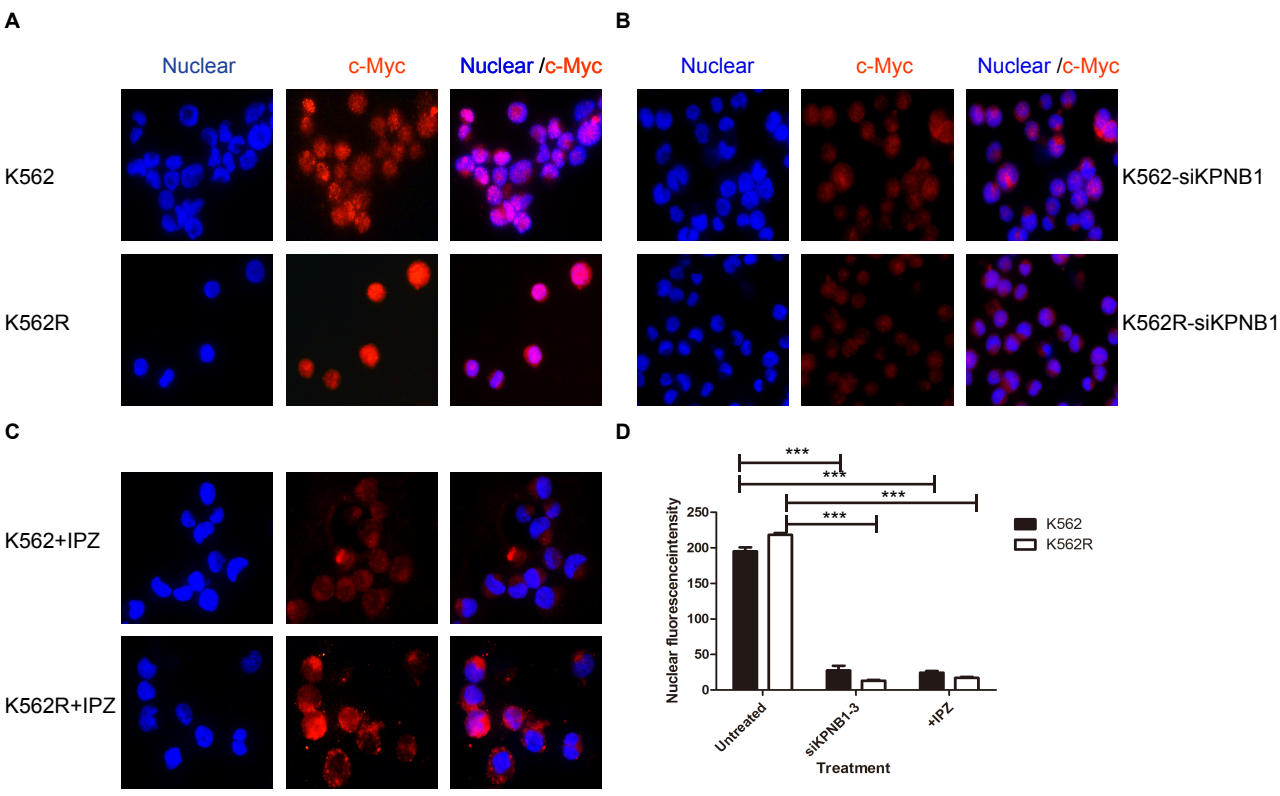

Fig S2

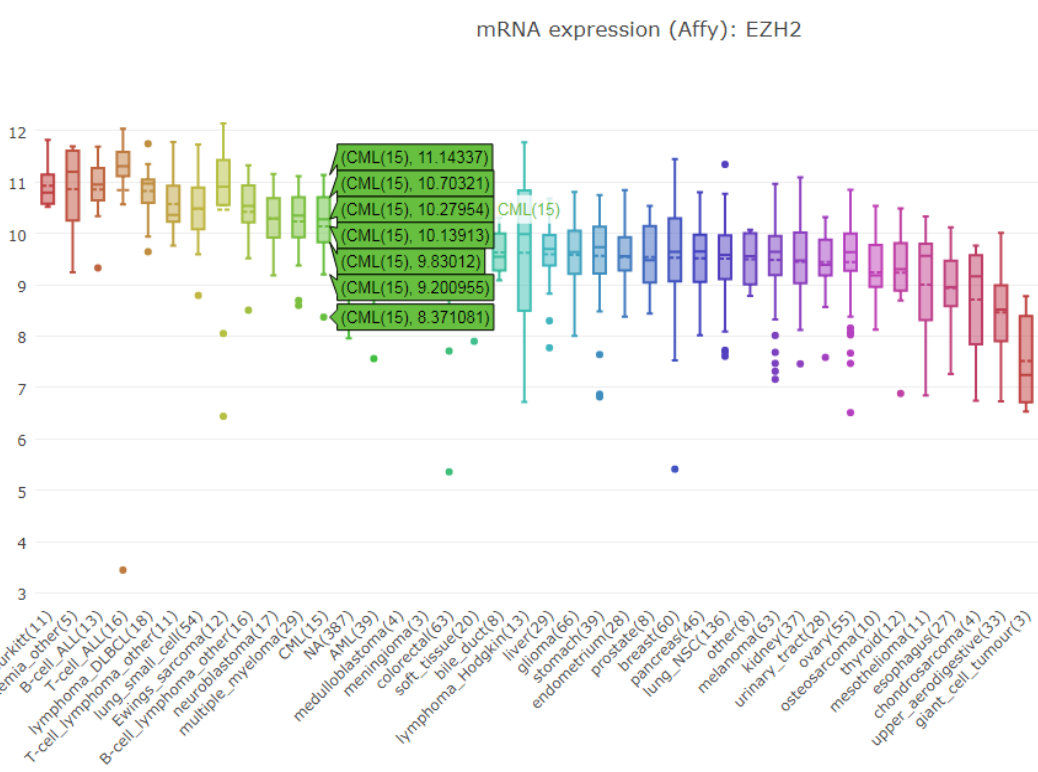

Fig S3

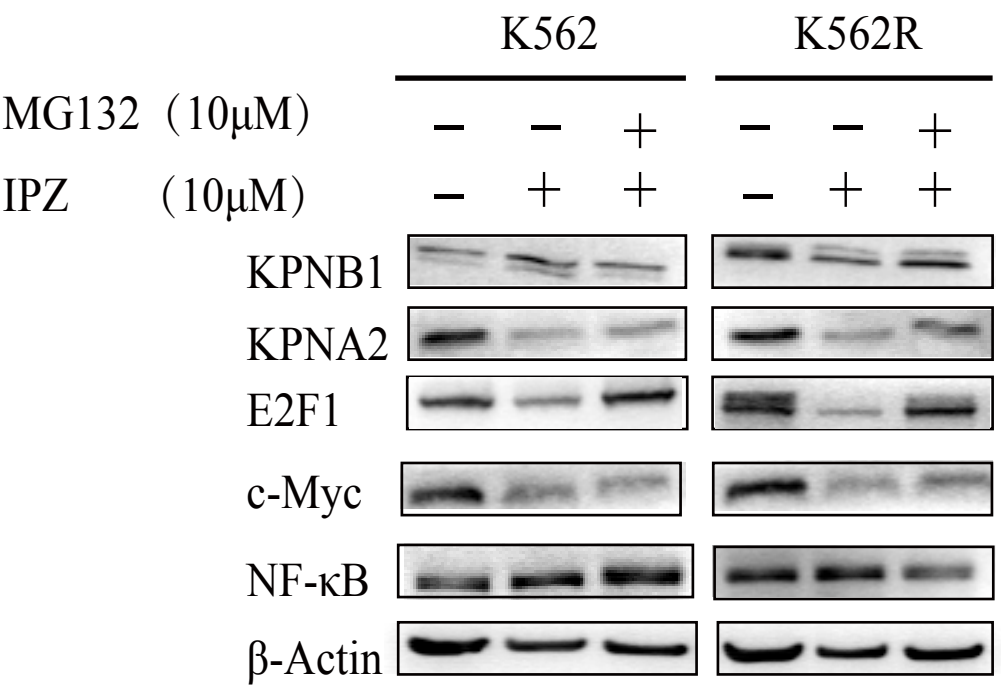

Fig S4

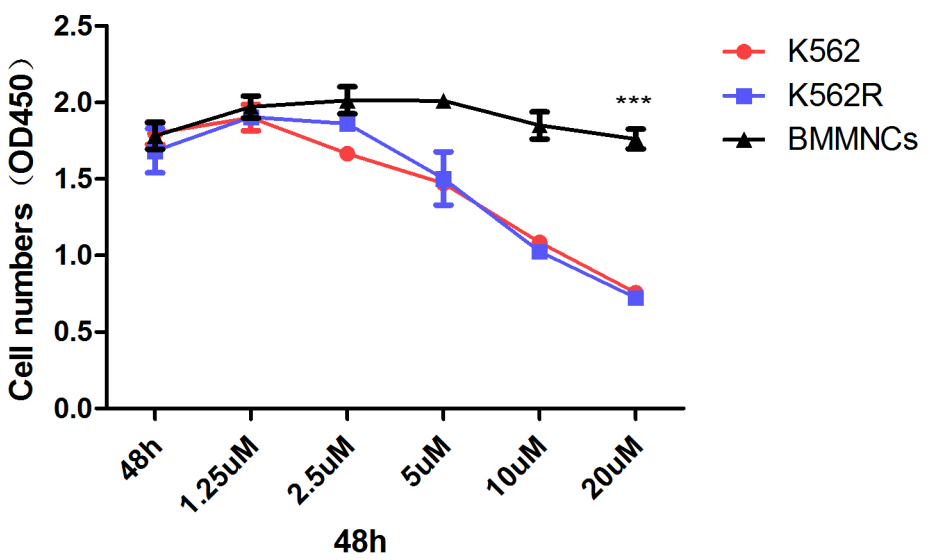

Fig S1. Inhibition of KPNB1 by small interfering RNA and IPZ down-regulated the nuclear location of E2F1. A, the subcellular location of c-Myc in K562 and K562R cells. B, silencing of KPNB1 blocks the entry of c-Myc. C, inhibition of KPNB1 by IPZ (5μM) for 24h change the subcellular distribution of c-Myc. D, statistical histogram of fluorescence intensity after been inhibited KPNB1. ***P<0.001.

Fig S2. The analyse of EZH2 mRNA level by using Broad Institute Cancer Cell Line Encyclopedia (CCLE). EZH2 functions as a transcription repressor and plays an important role in coordinating gene expression and repression during many physiological and developmental processes. Deregulation of EZH2 is involved in human diseases, including diabetes and cancers. In malignant peripheral nerve sheath tumors (MPNST) cells, EZH2 expression is significantly up-regulated, and EZH2 positively regulates KPNB1 through miR-30d. We analyze the expression of EZH2 at Broad Institute Cancer Cell Line Encyclopedia (CCLE), and we find that EZH2 exhibit a high mRNA level in CML cells.

S3. The protein expression of KPNB1, KPNA2, E2F1 and other regulators after treatment of IPZ and (or) MG132. 5μM IPZ plus 10μM MG132 significantly reduced the expression of KPNA2, c-Myc. However, proteasome inhibition by MG132 combined with IPZ prevent the decrease in E2F1.

S4. IPZ affects the proliferation ability of K562 and K562R cells without harming the normal bone marrow mononuclear cells. ***P<0.001.