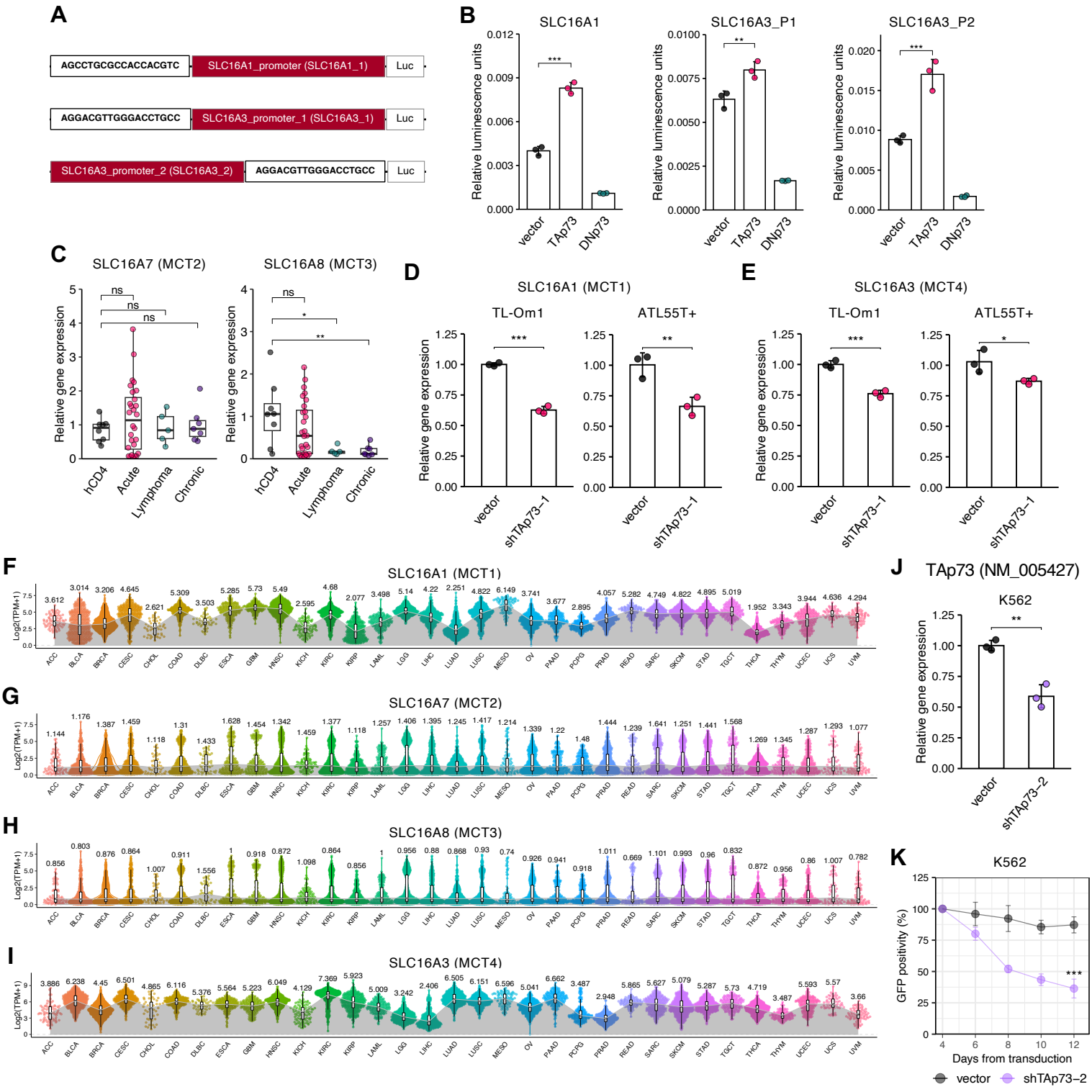

Supplementary Figure S5. Transcriptional regulation of SLC16A1 and SLC16A3 by TAp73, and mRNA expression levels of MCT-coding genes among various cancers.

(A) A schematic of vectors for the luciferase assay related to Fig. 6C and Supplementary Fig. S5B. Enriched TP73 binding motifs from the ChIP-seq peaks for TAp73 (red bars in Fig. 6A) are inserted into the vectors with SLC16A1 and SLC16A3 promoters. Since two kinds of promoters have been reported for the latter, we generated two corresponding vectors as shown.

(B) Relative luciferase activities for the SLC16A1 promoter (left) and the SLC16A3 promoter (center and right) with TP73 isoform expression in Jurkat cells. (n=3).

(C) mRNA expression of SLC16A7 (left) and SLC16A8 (right) by RT-qPCR in healthy human donor CD4$^+$ T cells (hCD4) (n=8) and ATL cells (acute type, n=28; lymphoma type, n=5; chronic type, n=7).

(D and E) mRNA transcripts of SLC16A1 (D) and SLC16A3 (E) in ATL cells after TAp73 knockdown for 48 hours (n=3).

Supplementary Figure S5. Transcriptional regulation of SLC16A1 and SLC16A3 by TAp73, and mRNA expression levels of MCT-coding genes among various cancers. (Continued)

(F-I) Transcripts per million (TPM) of MCT family-coding genes, SLC16A1 (F), SLC16A7 (G), SLC16A8 (H) and SLC16A3 (I), in TCGA data. Tumor type abbreviations are found in the Methods.

(J) TAp73 mRNA expression measured by RT-qPCR in K562 cells 48 hours after shTAp73-2 knockdown (n=3).

(K) GFP competition assay of K562 cells with shTAp73-2 knockdown (n=3).

Results are plotted as mean ± SD, using the Student's t test (D, E, J and K), one-way ANOVA with post-hoc Dunnet (B) or Steel test (C). *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$; ns, not significant.