

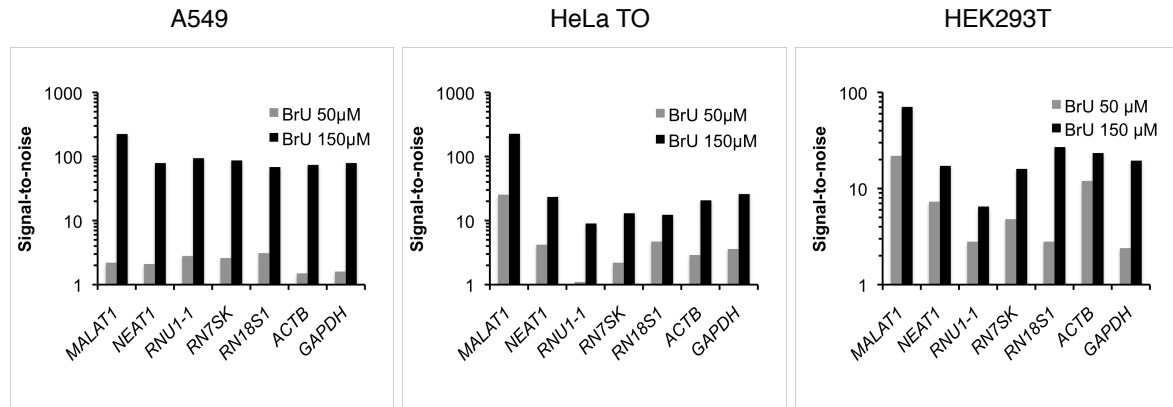
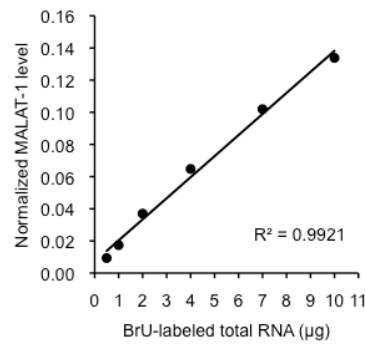
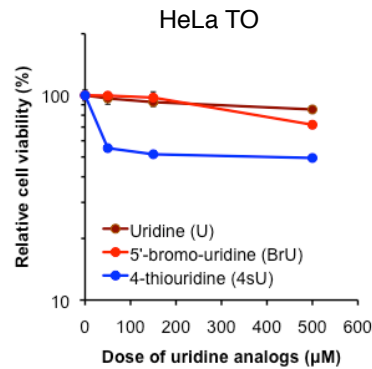
A**B****C**

Figure S1. BrU-labeled RNA was specifically quantified by qPCR in various cell types and cytotoxicity assay. (A) A549, HeLa TO and HEK293T cells were treated with BrU at the concentrations indicated for 24 h. The newly transcribed RNA with BrU was then immunopurified by anti-BrdU antibody, and quantified by qPCR for 7 representative RNAs. Quantitative values obtained from untreated cells were used for normalization. (B) Quantification of *MALAT1* by qPCR in BrU-labeled total RNA obtained from HeLa TO cells, treated with 50 μM BrU for 24 h. (C) HeLa TO cells were treated with uridine analogs at the concentrations indicated for 48 h. Viable cells were counted using Cell Counting Kit-8. The relative abundance (100% in untreated cells) is shown in the graph. Values represent mean \pm SD obtained from four replicate experiments.

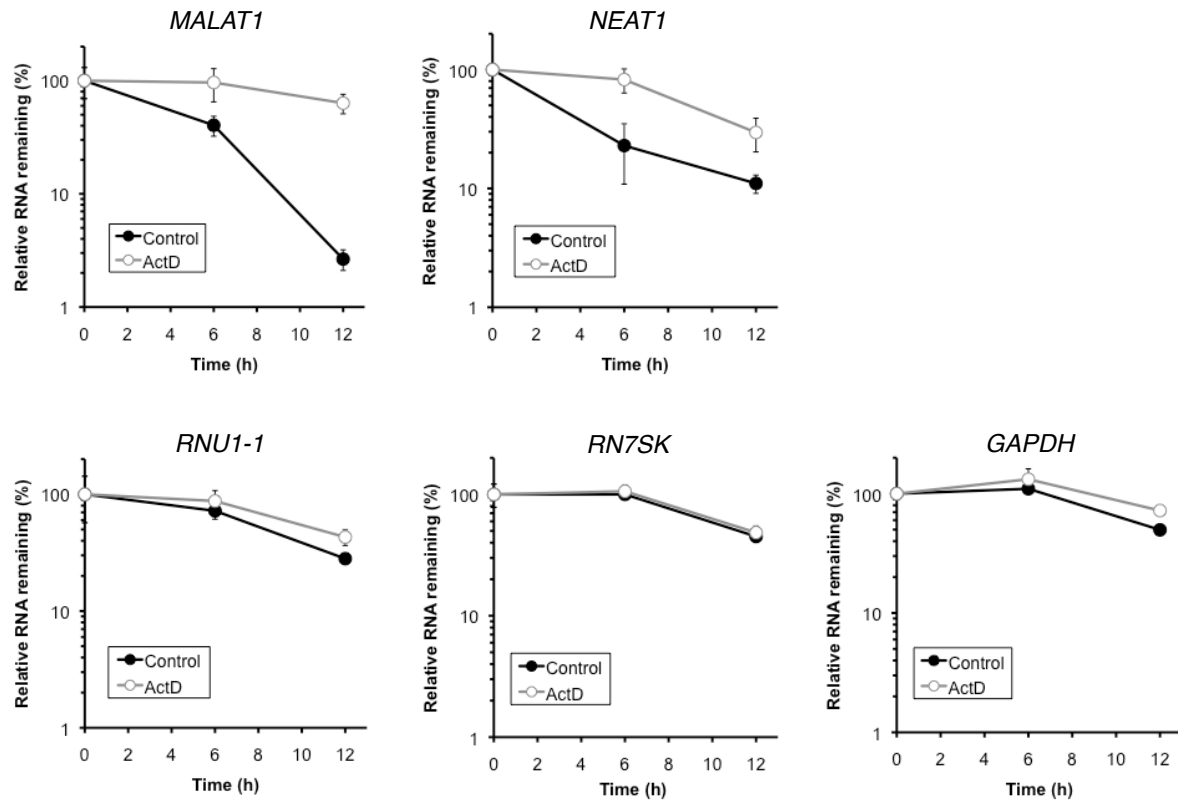


Figure S2. Actinomycin D (ActD) inhibits the degradation of MALAT-1 and NEAT1 in HeLa TO cells. HeLa TO cells were untreated (solid circles and solid lines) or treated with ActD at the concentration of 2 μ g/ml (open circles and grey lines). Decay rates of 5 representative RNAs were then determined by BRIC through qPCR. The relative quantitative values at time 0 h were arbitrarily adjusted to 100%. Values represent mean \pm errors obtained from duplicate experiments.

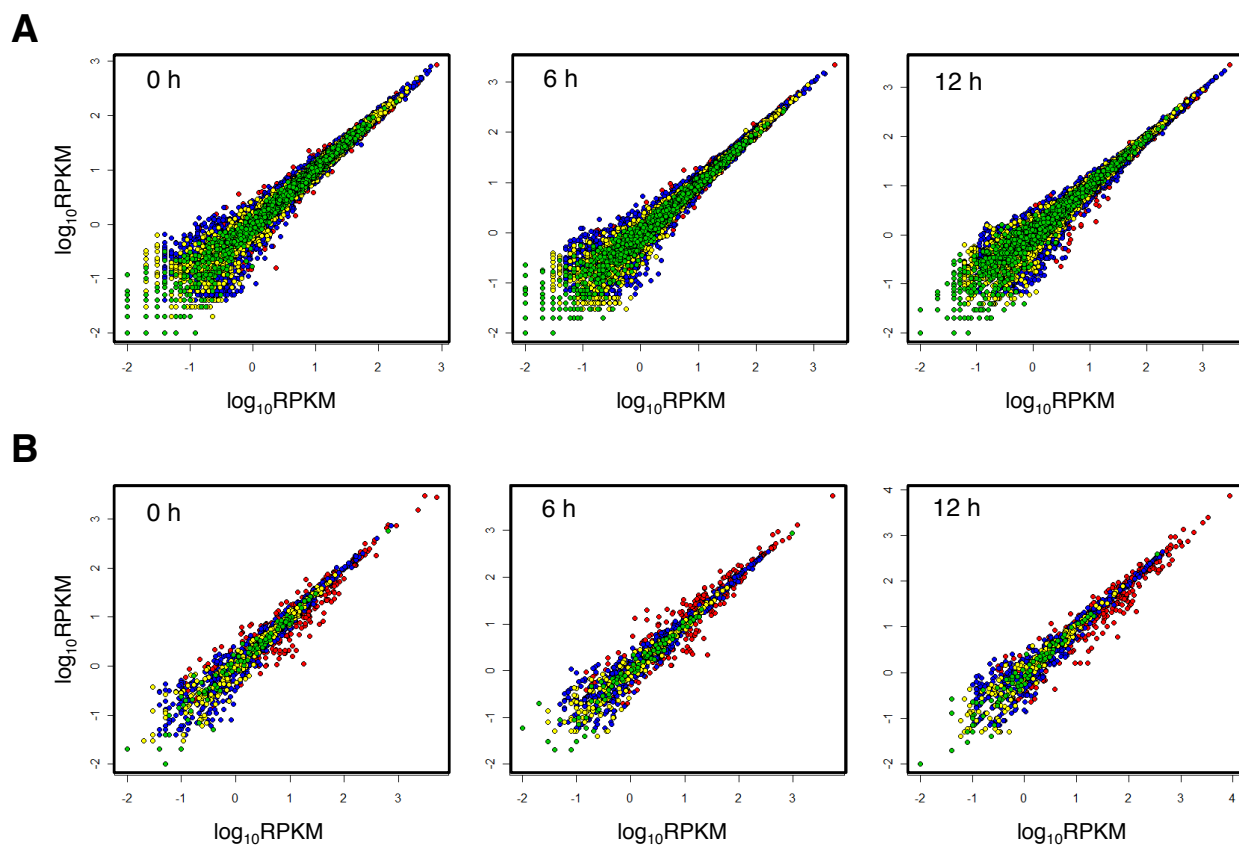


Figure S3. Scatter plot of RNA-seq tags in HeLa TO cells determined by BRIC-seq. RPKMs (Reads Per Kilobase of exon model per Million mapped reads) were measured independently from two replicate experiments, 0 h, 6 h, and 12 h after removal of BrU from the culture medium. The x-axis shows the RPKM of each gene for the first replicate and the y-axis shows the corresponding RPKM for the second replicate. NCBI Reference Sequences (Refseq) were used in the NM category (A) and NR category (B). The color of plots indicates the length of RNA: < 1 kb in red, 1-3 kb in blue, 3-5 kb in yellow and > 5 kb in green.

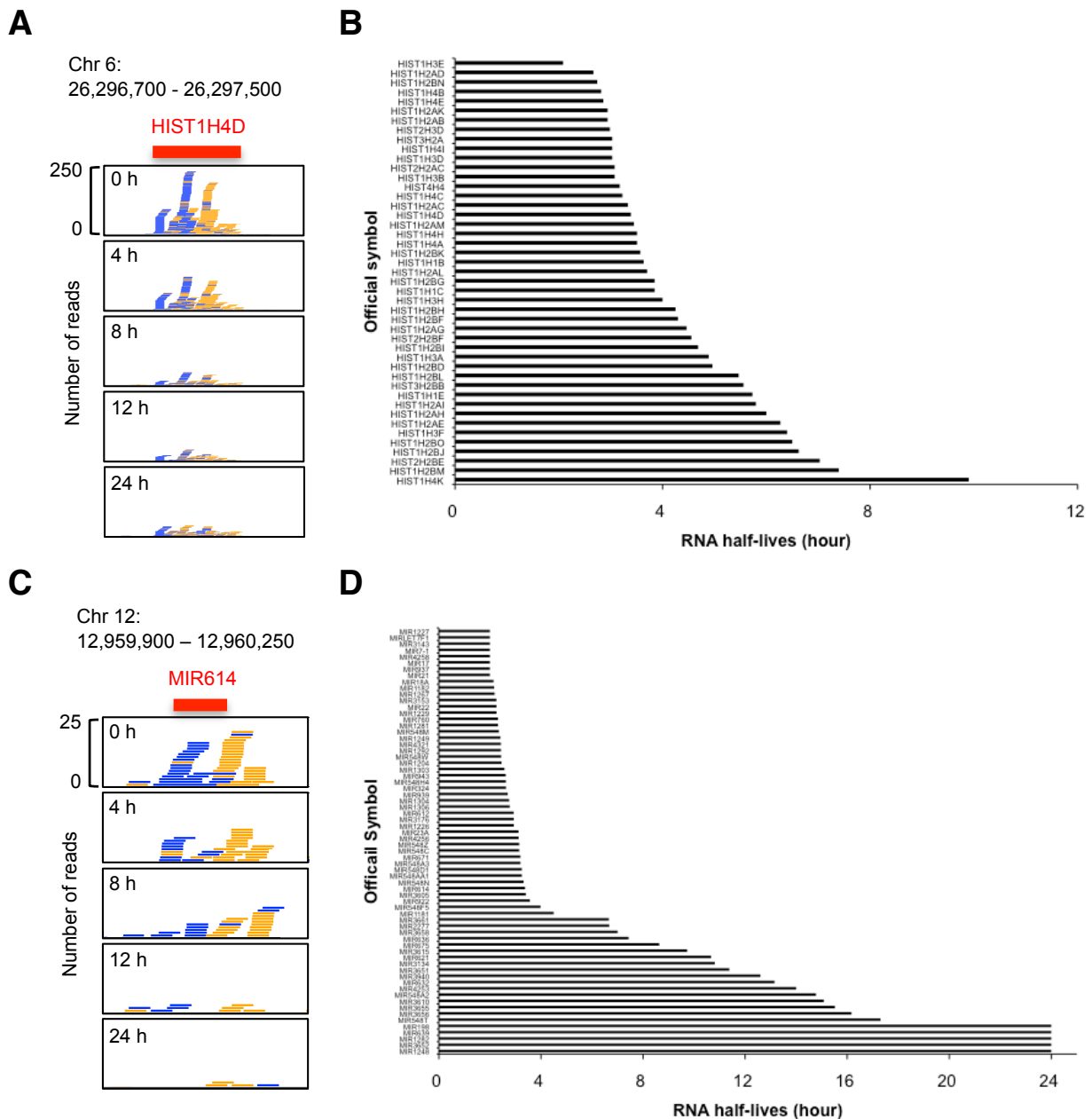


Figure S4. The half-lives of histone mRNAs and pre-miRNAs were determined by BRIC-seq. (A) Typical mapping data of RNA-seq tags obtained from BRIC-seq on a Genome Analyzer (Illumina). The chromosomal locus of HIST1H4D is shown. (B) The half-lives of 45 histone mRNAs. (C) The chromosomal locus of MIR614 is shown. (D) The half-lives of 64 pre-miRNAs.

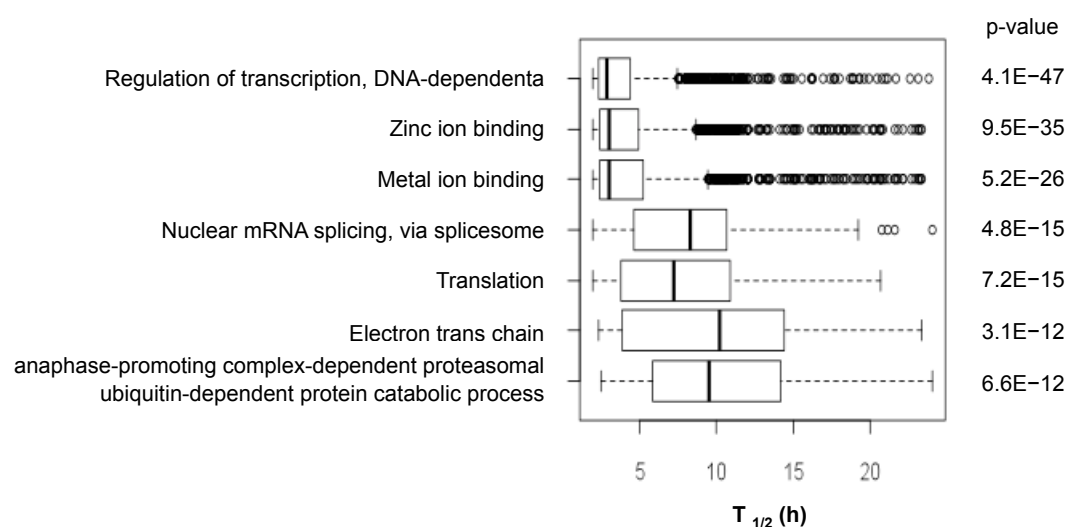


Figure S5. Seven representative GO categories that significantly associated with distribution of mRNA half-lives. Horizontal bars indicate the range of half-lives. Box plots show the fifth, 25th, 50th, 75th, and 95th percentiles. The median for each GO categories is indicated by short vertical lines. The degree of enrichment for a given GO category was analyzed by the Wilcoxon signed-rank test (p-value).

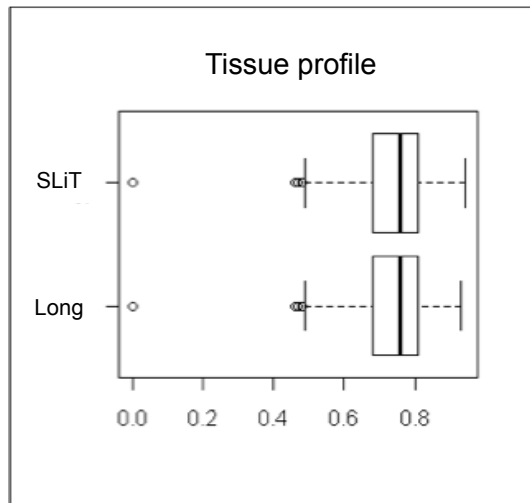


Figure S6. The tissue profiling. The z-score was calculated using Adult TSSseq database (See also Supplemental Table S8). The z-score averages of SLiTs and long-lived ncRNA were 0.75 and 0.74, respectively.

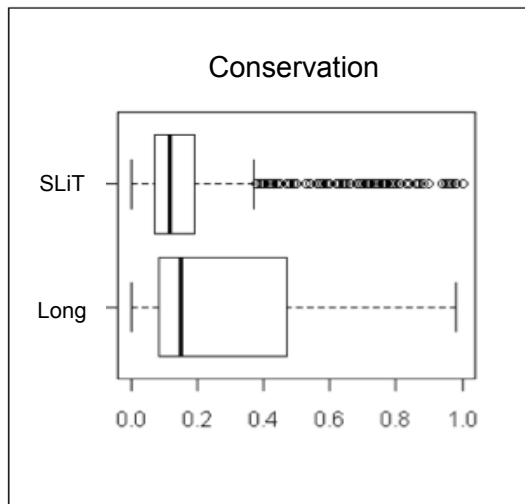


Figure S7. The evolutionary sequence conservation. The phastCons score was calculated using the public database from <http://hgdownload.cse.ucsc.edu/goldenPath/hg18/phastCons44way/vertebrate/> (See also Supplemental Table S9). The average phastCons score of SLiTs and long-lived ncRNA were 0.08 and 0.29, respectively, and the p-value was < 0.001 .

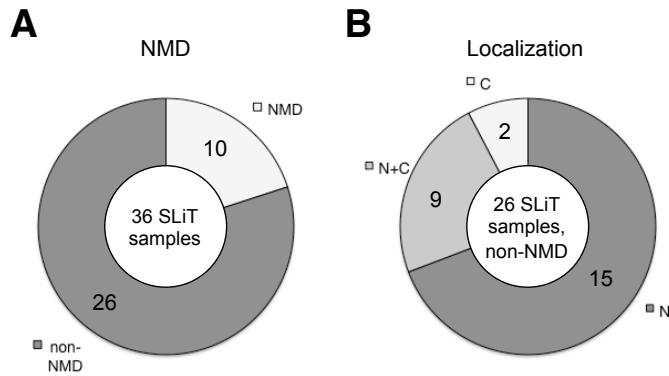


Figure S8. Summary of the selection of ncRNAs from SLiTs. (A) Discrimination of NMD-targeted transcripts among 36 SLiTs. (B) Localization of 26 SLiTs that are non-NMD targets. "N" indicates nucleus, "N+C" indicates nucleus and cytoplasm, and "C" indicates cytoplasm. We assessed that the transcript was localized at "N", "N+C" or "C" when the N/C localization ratio was over 2, from 2 to 0.5 or below 0.5, respectively. As controls, the N/C ratios of *GAPDH* and *MALAT1* were 0.5 and 10.6, respectively.

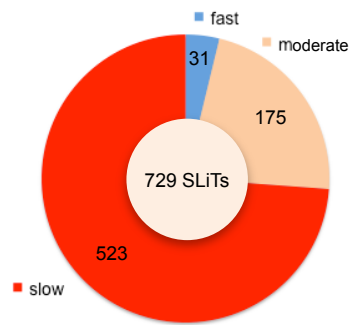


Figure S9. The degradation of 729 SLiTs is changed by actinomycin D (ActD) in HeLa TO cells. HeLa TO cells were untreated or were treated with ActD at 2 $\mu\text{g}/\text{mL}$. The stability of 729 SLiTs ($t_{1/2} < 4$ h; except tRNAs) were then assessed using BRIC-seq. We assessed that the transcript decay was accelerated (indicated as fast), not changed (indicated as moderate) or inhibited (indicated as slow) when the ratio of RPKM (12 h, treated with ActD) to RPKM (12 h, untreated) was below 0.5, from 0.5 to 2 or over 2, respectively. Data were excluded when either RPKM (12 h, ActD) or RPKM (12 h, control) was 0. See also Supplemental Table S14.

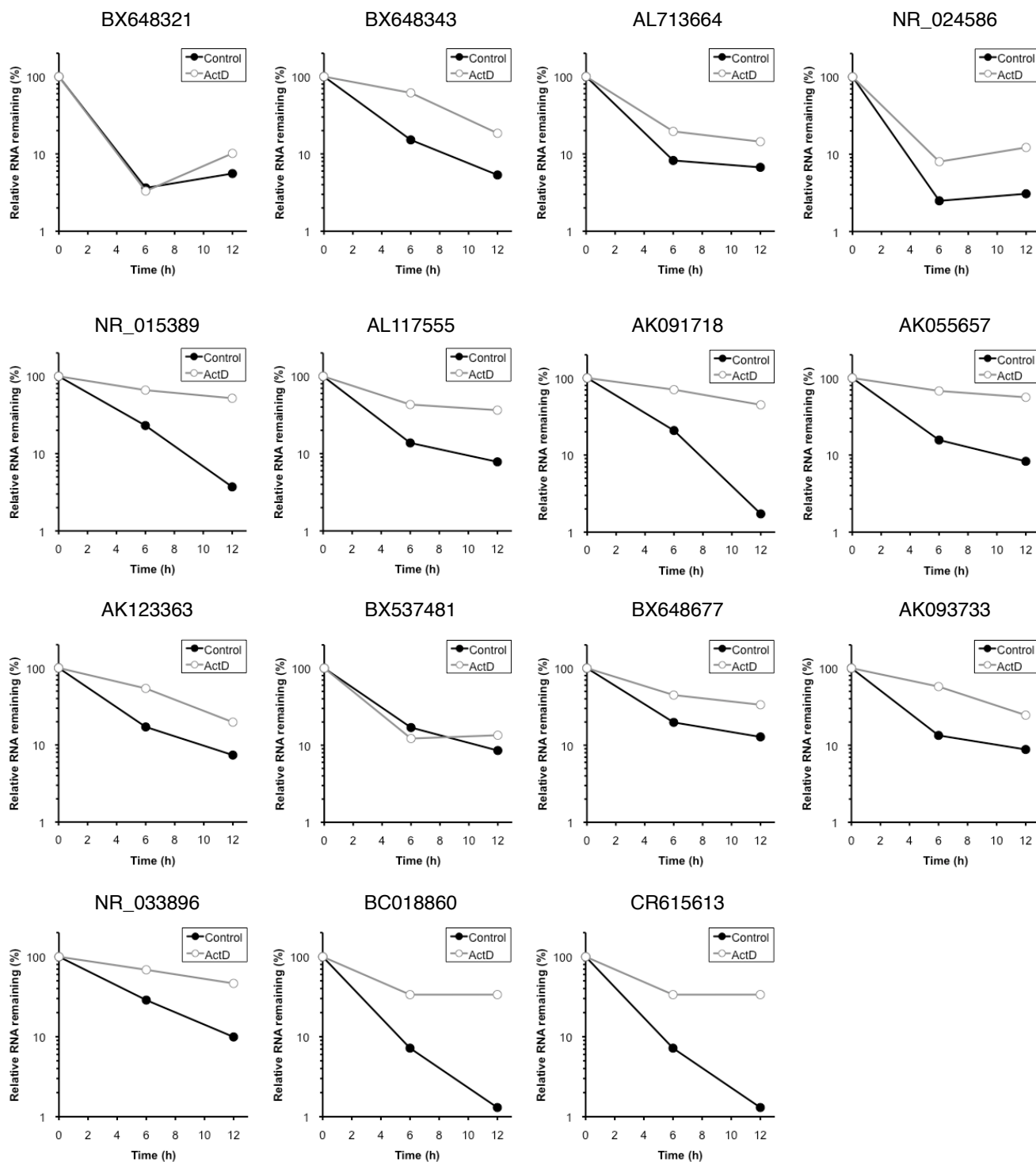


Figure S10. The degradation of 15 selected SLiTs was changed by actinomycin D (ActD) in HeLa TO cells. HeLa TO cells were untreated (solid circles and solid lines) or treated with ActD at 2 μ g/ml (open circles and grey lines). The decay rates of 15 selected SLiTs were then determined by BRIC through deep sequencing. Relative quantitative values at time 0 h were arbitrarily adjusted to 100%.

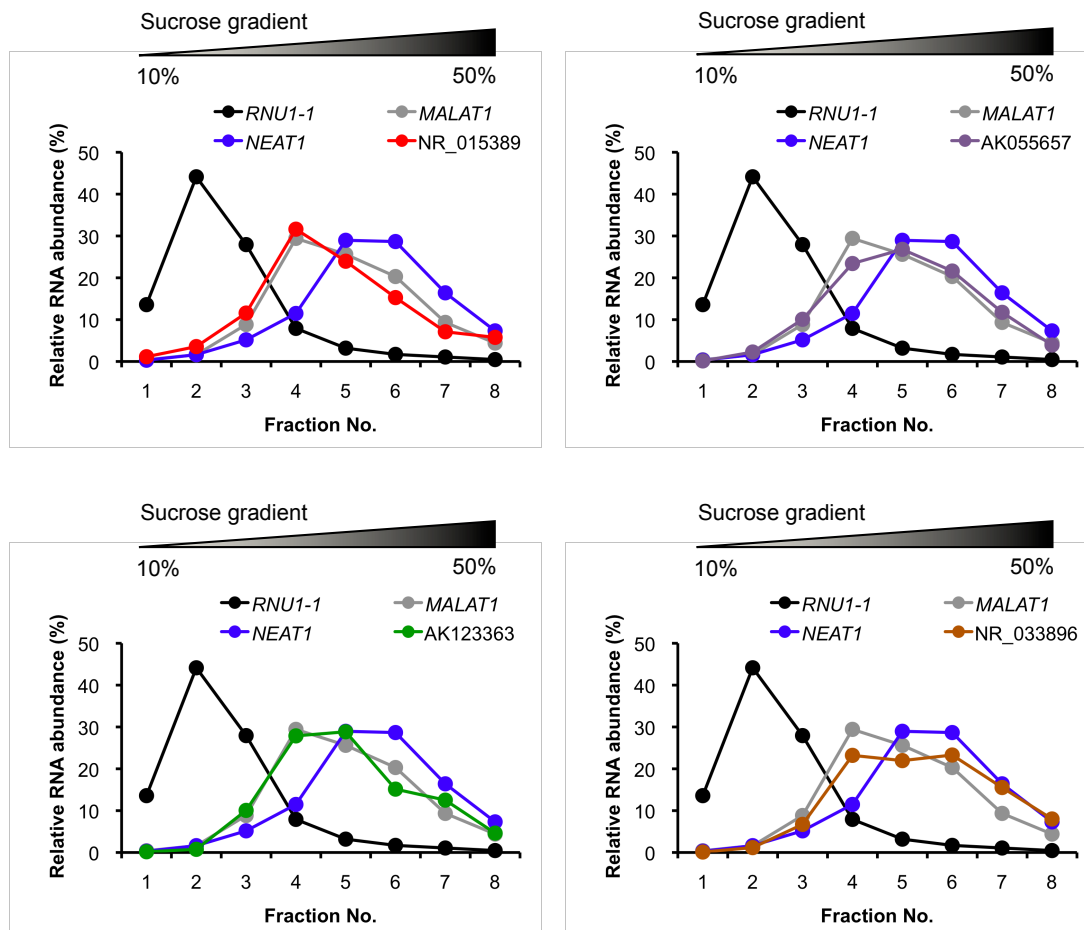


Figure S11. The relative amount of RNA was quantified by RT-qPCR from nuclear fractions obtained by sucrose gradient centrifugation.

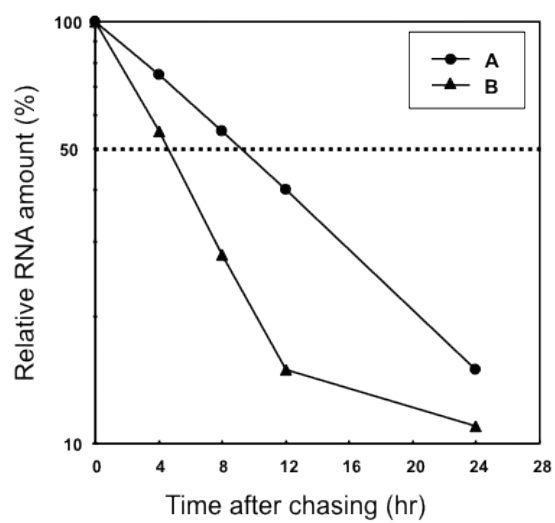


Figure S12. Scheme for determining half-life. The half-life of A RNA was determined by using time point 0, 4, 8, and 12 h. By contrast, the half-life of B RNA was determined by 0, 4, and 8 hr.

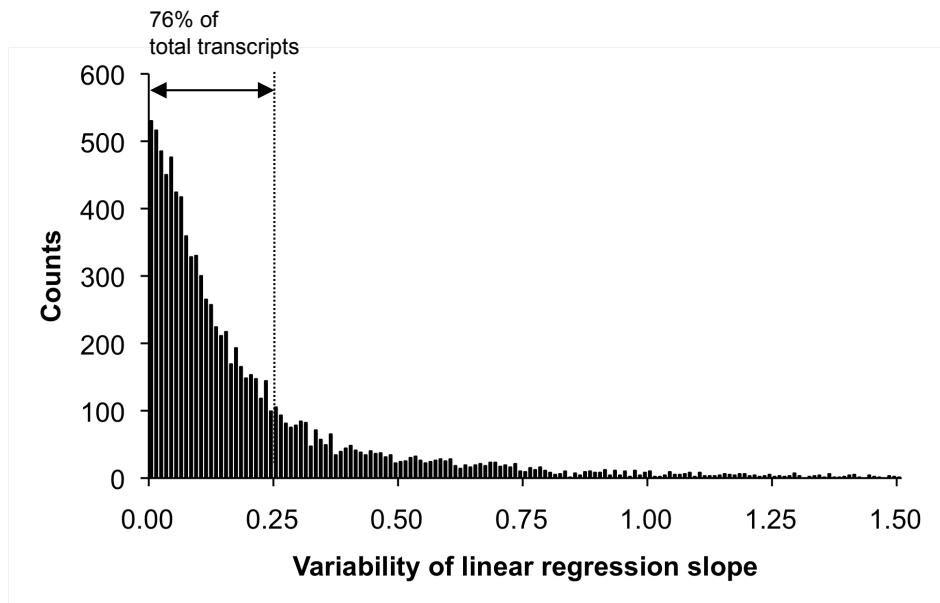


Figure S13. The distribution of the errors associated with RNA half-life measurements. The error values were calculated by determine the $[|ave2-ave1| / ave1]$ for each transcripts and drew the distribution of these values in the graph. Dashed line distinguishes the point of 0.25 error value.