

Supplemental

Noncoding RNAs endogenously rule the cancerous regulatory realm while proteins govern the normal

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1. Legends for supplemental figures and tables

Figure S1. Distribution of samples containing zeros. A-B, Histogram of samples containing zeros in cancer (A) and normal(B). most cancer samples contained 30,000 zeros, and few samples contained more than 40,000 zeros. Yet, most of SRA samples contained more than 60,000 zeros.

Figure S2. LOESS regression of PTEN and PTENP1 under normal and cancer. The correlation between PTEN and PTENP1 was weak at normal but very strong in cancer, which validated our inference from FINET.

Figure S3. LOESS regression of RPS23 and UBE2E3 under normal and cancer. The correlation between RPS23 and UBE2E3 was very strong at normal but no-correct in cancer, which validated our inference from FINET.

Figure S4. Zoom in chromosome 14 noncoding RNA interactions circled by Figure 5B. Three panels showed that all interactions are inside chromosome 14 (upper), noncoding RNAs work as regulators (middle), and noncoding RNAs regulate their targets in detail(bottom).

Table S1. Target profile of normal network

Table S2. Target profile of cancer network

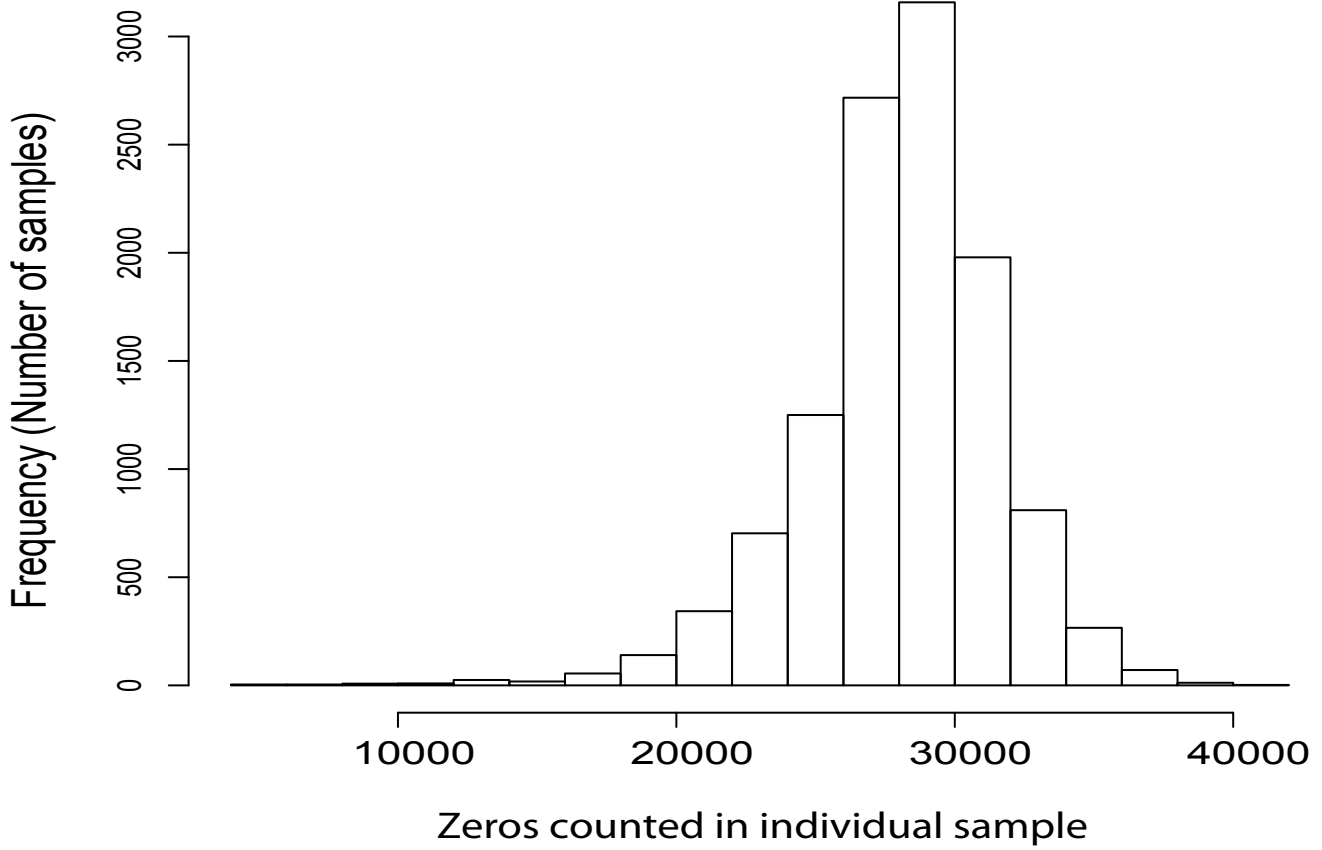
Table S3. Modules and composition of normal network

Table S4. Modules and composition of cancer network.

Fig. S1

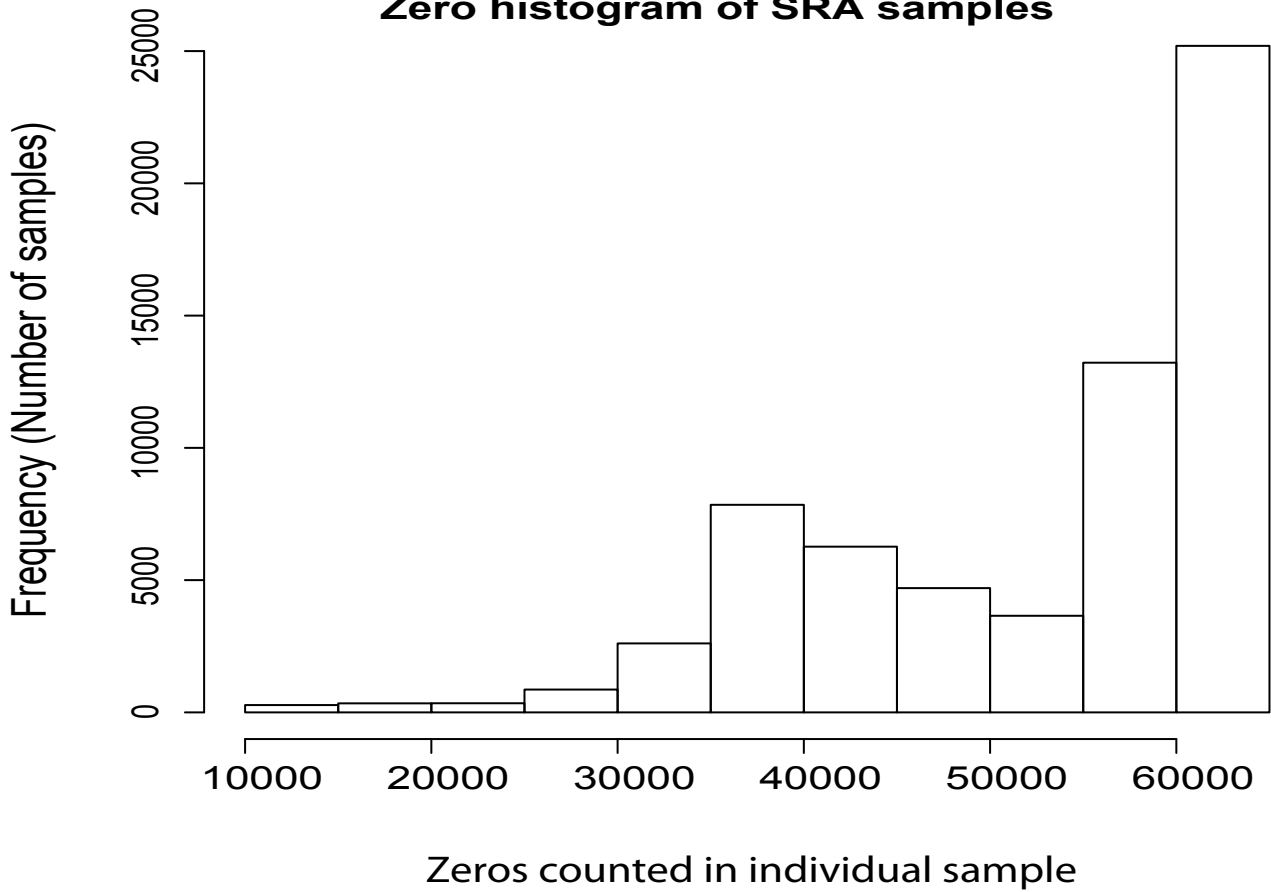
A

Zero histogram of TCGA samples

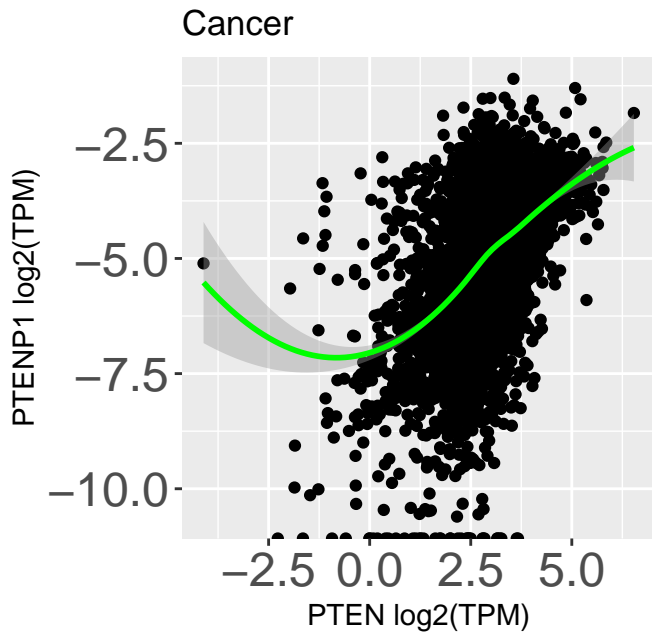
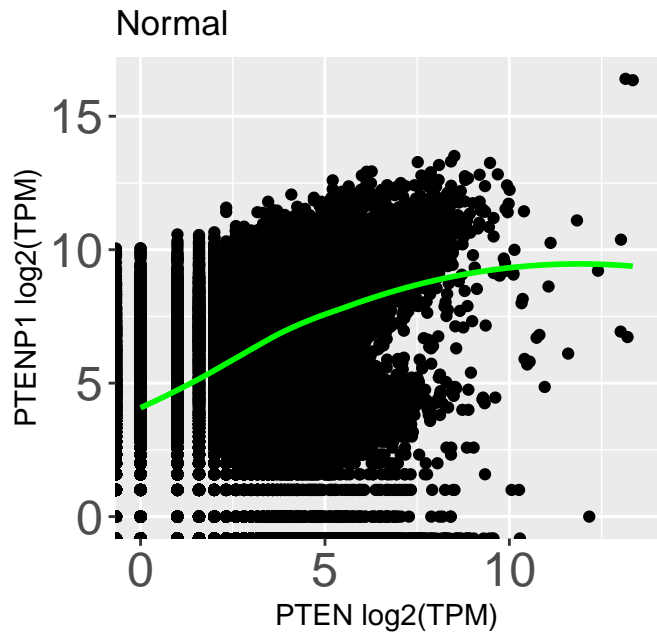


B

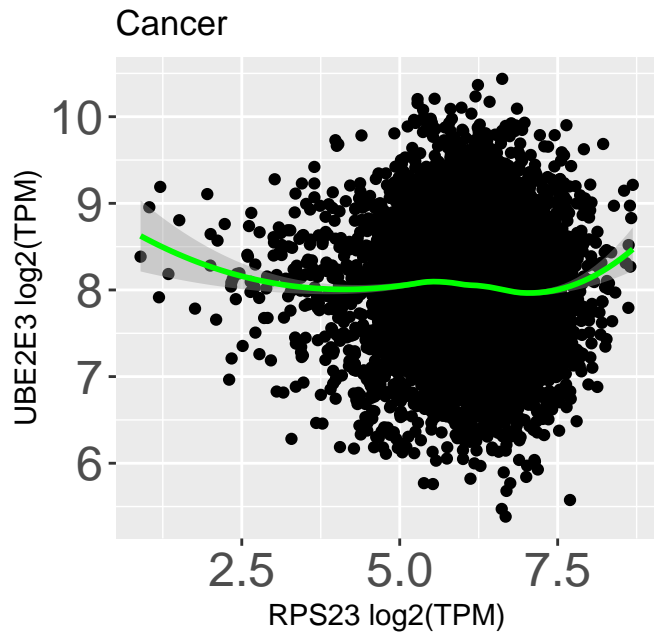
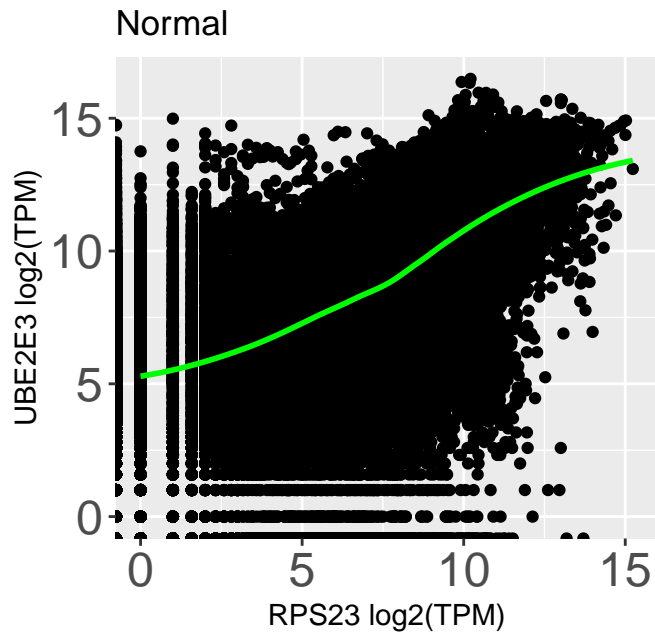
Zero histogram of SRA samples



PTEN and PTENP1



RPS23 and UBE2E3



Figure_S4

