SUPPLEMENTARY TABLES

**Table S1.** Average percentage elimination of putative mutation calls by per-sample false-positive filters across all tissues.

**Table S2.** Total number of samples per tissue included for the final set of mutation calls.

**Table S3.** List of all somatic mutations identified in this study.

**Table S4.** P-values (-log₁₀[p-value]) for the coefficients of each feature used in a linear regression on the total number of mutations per tissue.

**Table S5.** Average percentage of each mutation type across samples of the given tissue.

**Table S6.** Significant associations between biological sex and mutation load across tissues and mutation types.

**Table S7.** Tissue correspondence between the GTEx [1] and Roadmap Epigenomics projects [2].

**Table S8.** Significant GO enrichments for genes whose expression is significantly and negatively associated with C>T mutation load across several tissues.
Table S9. Significant GO enrichments for genes whose expression is significantly and positively associated with C>T mutation load across several tissues.

Table S10. List of cancer driver genes used in this study [3].

Table S11. List of mutations in cancer driver genes after further elimination of potential false positives.

Table S12. List of mutations in cancer driver genes annotated in Oncokb [4].

Table S13. Correspondence between GTEx [1] tissues and cancer types from Tomasetti and Vogelstein [5].

References
5. Tomasetti C, Vogelstein B. Variation in cancer risk among tissues can be explained by the