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Supplemental information

Resolution of structural variation in diverse mouse genomes reveals chromatin remodeling due to transposable elements

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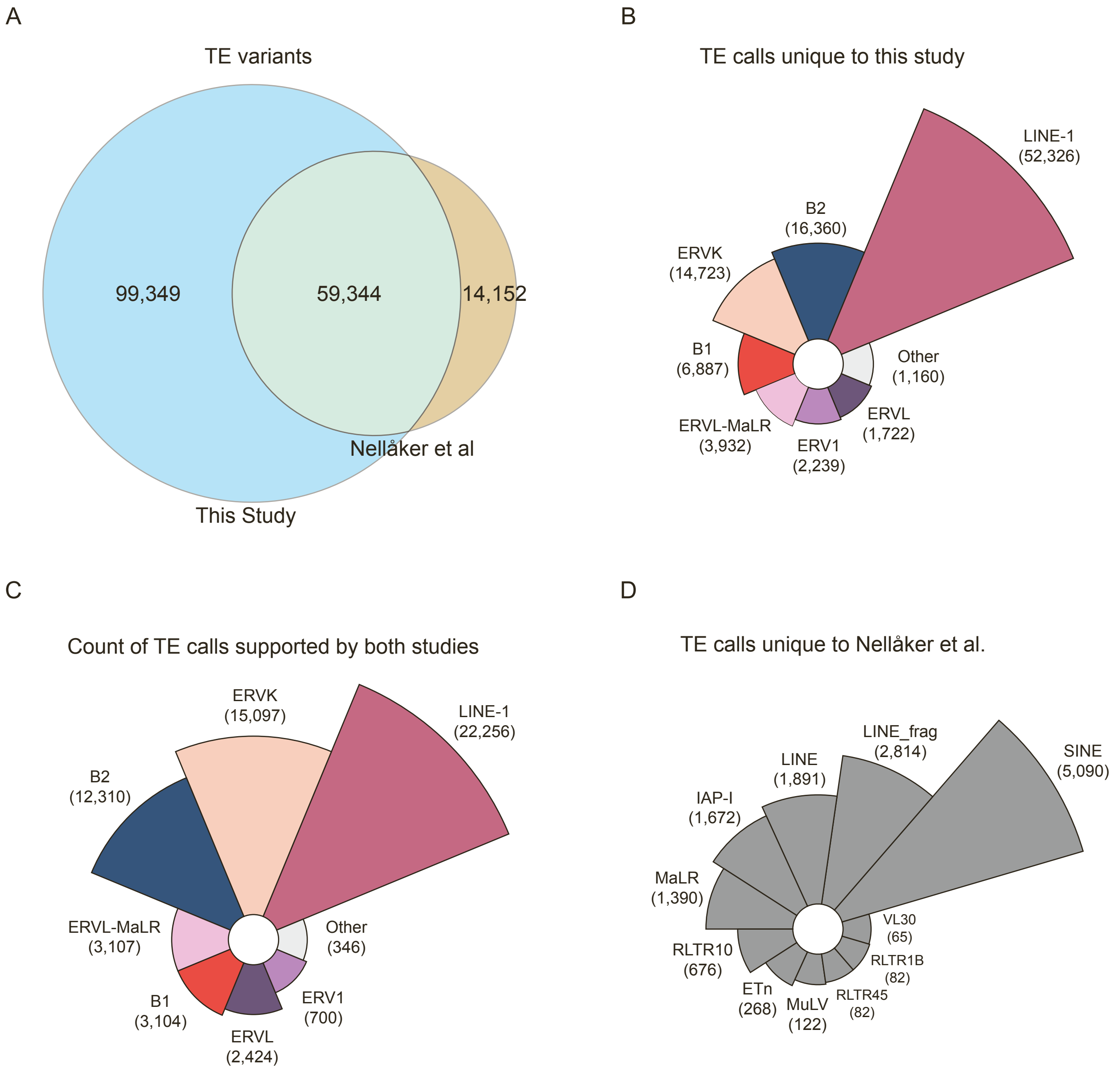

Figure S1. Intersection of TE variant calls with previously published TE variants, Related to Figure 2. (A) Number of TE variant calls unique to this study, concordant with Nellaker et al, and unique to Nellaker et al 2012. (B) Count of TE variant calls by family for calls unique to our study. (C) Count of TE variant calls by family for calls supported by each study. (D) Count of TE variant calls by category for calls unique to Nellaker et al.

Figure S2. Transposable element variant calls, Related to Figure 2. Count of variant calls detected by family and subfamily.

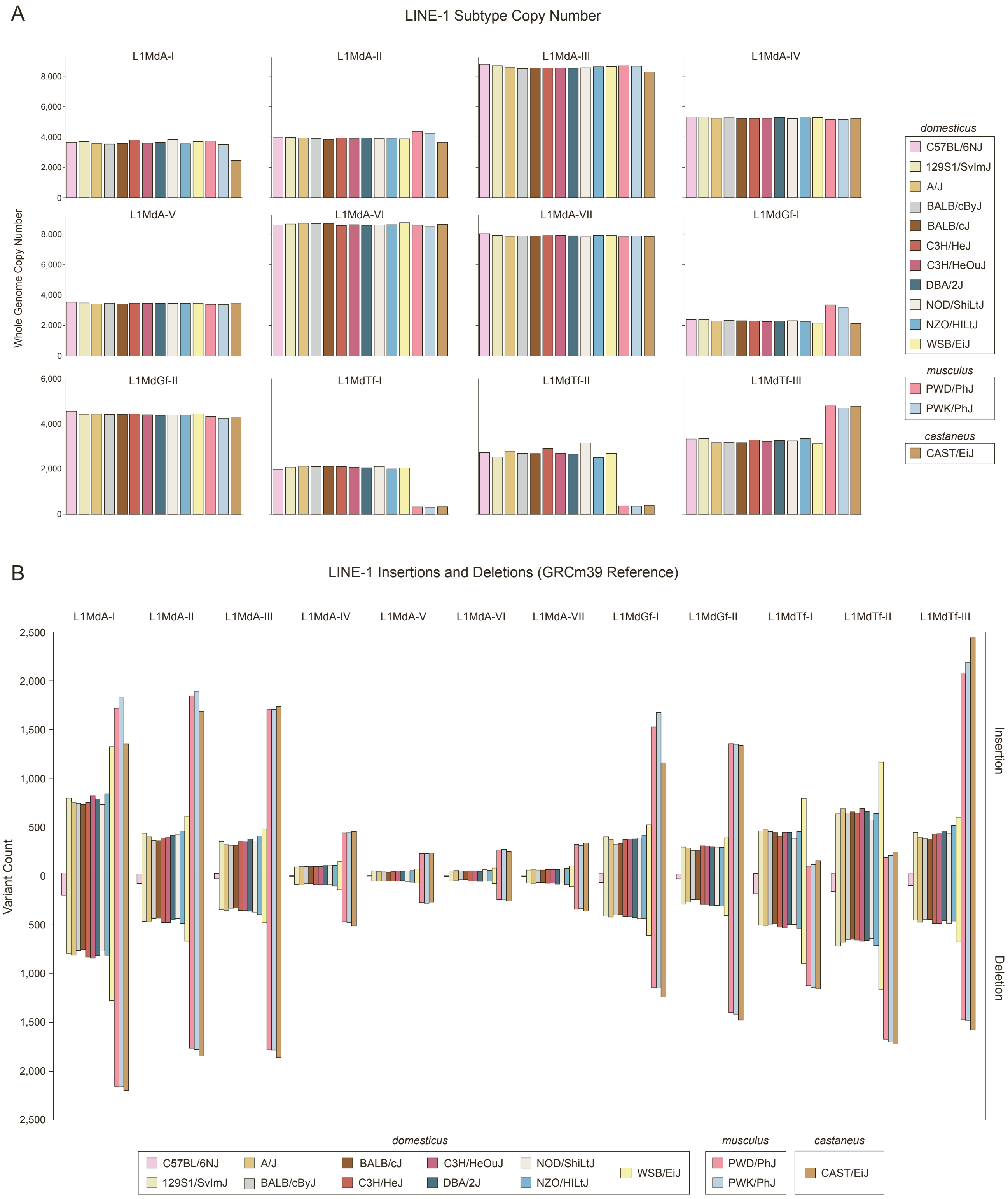

Figure S3. LINE-1 subtype variation within diverse mouse genomes, Related to Figure 2. (A) Whole-genome copy number of LINE-1 subtypes within each mouse assembly. (B) Count of LINE-1 insertions and deletions for each LINE-1 found in the indicated mouse genome divided by LINE-1 subtype.

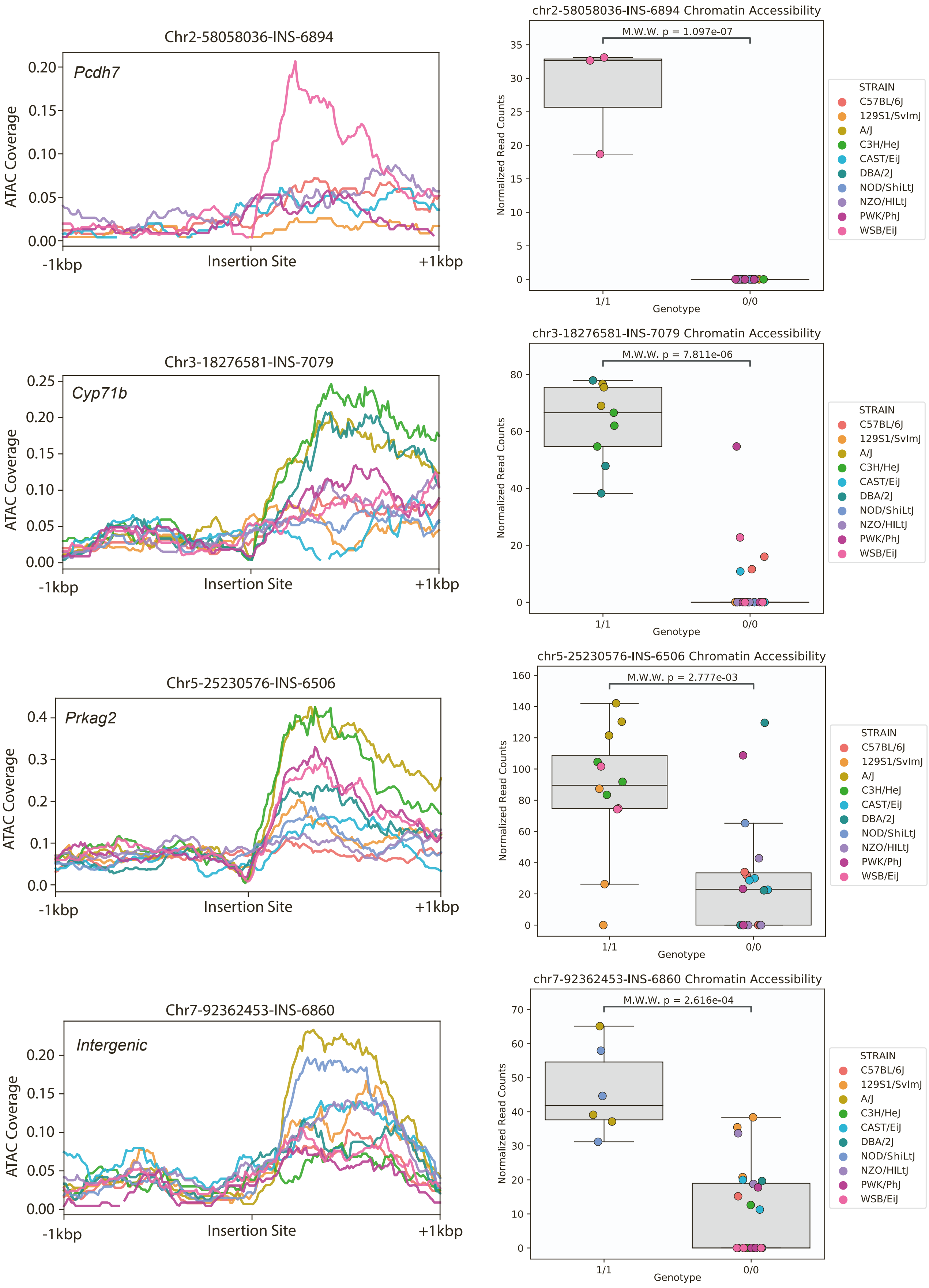

Figure S4. Examples of LINE-1 insertions associated with chromatin accessibility, Related to Figure 4. ATAC-seq coverage profile (left) accompanied by change in ATAC-sequencing read counts for animals that contain the insertion (1/1 GENOTYPE) compared to animals which do not (0/0 GENOTYPE). Examples are labeled with gene name in the top left if the insertion is within a gene. The insertion location is in the middle of the X-axis, with 1Kb up and downstream depicted.

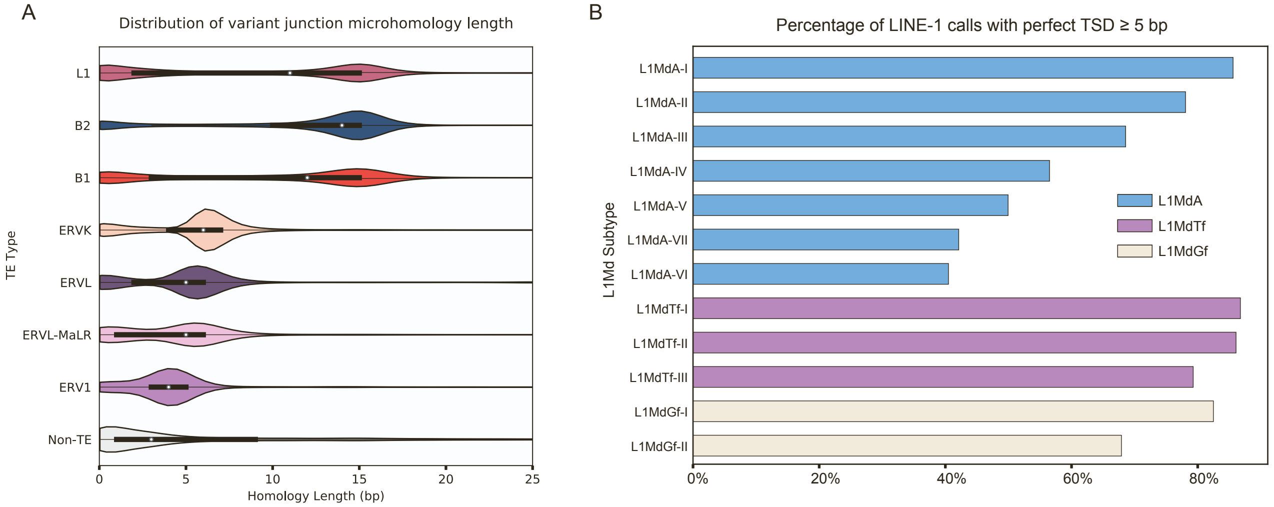

Figure S5. Target site duplications of TE variants, Related to Figure 2. (A) Distribution of target site distribution lengths for each TE type (B) Percentage of LINE-1 variants which contain a target site duplication >= 5 bp grouped by subtype.