CORRECTION

Correction to: Contribution of transposable elements and distal enhancers to evolution of human-specific features of interphase chromatin architecture in embryonic stem cells

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The original version of this article unfortunately contained a mistake in publishing the panel C for Figs. 3, 5 and 6. The corrected Figs. 3, 5 and 6 are shown in the next page. The original article has been corrected.

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The online version of the original article can be found at
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Fig. 3 Placements of hESC enhancers and HARs within TADs are directly correlated with the size of TADs in the hESC genome. **a** Significant direct correlations between the size of TADs and SE's span defined as a number of bp between the two most distant SEs located within a given TAD in the hESC genome (top two panels). Top left panel shows a correlation profile between the size of 504 TADs and genomic span of 642 SEs located within TADs. Top right panel shows a correlation profile between the size of 103 TADs harboring at least two SEs and genomic span of 241 SEs residing within TADs. Percentiles within shaded areas indicate the percent of TADs containing HSGRL within a designated set, which increases concomitantly with the increasing quantity of SEs located within TADs and larger TAD size. Note that similar trends were observed in the mESC genome; however, the correlation coefficient values were not statistically significant (bottom two panels). **b** Patterns of significant direct correlations between the size of 147 revTADs and numbers of hESC-enriched enhancers (top left panel) and numbers of HARs (top right panel) located within the revTADs.

The bottom left panel illustrates the profile of significant correlation between the numbers of hESC-enriched enhancers and HARs residing within the revTADs (direct correlation; bottom left panel). In contrast, the bottom right panel shows the previously reported inverse correlation between the numbers of HSTFBS and HARs residing within the revTADs (Glinsky 2016a, b, c). **c** Genome-wide, there is a highly significant direct correlation between the numbers of hESC-enriched enhancers located within TADs and the average size of corresponding TADs harboring hESC-enriched enhancers. The numbers of hESC-enriched enhancers located within each individual TAD were quantified and TADs harboring 0 to 9 enhancers were segregated into subgroups harboring the same numbers of enhancers. The numbers of TADs in each subgroup are indicated. Eighty-one TADs harboring ten or more hESC-enriched enhancers (range 10–30) were segregated into one subgroup with the average enhancers' content of 12.5 per TAD. The average TAD sizes were computed for each subgroup of TADs, and the results were plotted to assess the correlation pattern.

Fig. 3 (continued.)

Fig. 3 (continued.)

Fig. 5 Clusters of Alu elements in the vicinity of putative DNA bending sites near the borders of SEDs and TADs. **a** Clusters of Alu elements near the borders of the ID3 SED on chr1:23,878,033-23,894,300 (16,268 bp). Dotted lines depict the genomic positions of the overlapping CTCF/cohesin-binding sites, interactions of which form the anchor base of the ID3 SED. **b** Clusters of Alu elements near the NANOG SED left border on chr12:7,864,594-7,869,500 (4907 bp). **c** Clusters of Alu elements near the NANOG SED right border on chr12:8,012,400-8,017,400 (5001 bp). Arrows in the fig-

ures **b** and **c** point the overlapping CTCF/cohesin-binding sites, interactions between which form the anchor base of the NANOG SED. Note that clusters of closely-spaced sequences of at least three Alu elements belonging to most ancient AluJ (~ 65 million years old), second oldest AluS (~ 30 million years old), and currently active modern AluY sub-families are observed, suggesting that placement and/or retention of Alu elements at these sites were occurring for millions of years and continues at the present time (see text for details)

Fig. 5 (continued.)

Fig. 5 (continued.)

Fig. 6 Conservation patterns of HSGRL in individual human genomes. Conservation of HSGRL in individual genomes of 3 Neanderthals, 12 Modern Humans, and the 41,000-year old Denisovan genome was carried out by direct comparisons of corresponding sequences retrieved from individual genomes and the human genome reference database (<http://genome.ucsc.edu/Neanderthal/>). Full-length sequence alignments with no gaps of the individual human genome sequences to the corresponding sequences in the human genome reference databases were accepted as the evidence of sequence conservation in the individual human genome. **a** Top two panels show conservation patterns of HSGRL located within the revTADs for 57 HARs and associated 75 CTCF-binding sites (top left panel); 22 HARs harboring 23 LMNB1-binding sites and 23 LMNB1-binding sites residing within 22 HARs (top right panel). Bottom two panels show conservation patterns of 90 HARs harboring 93 LMNB1-binding sites (bottom left panel) and 55 HARs harboring 55 CTCF-binding sites and 55 CTCF-binding sites located within 55 HARs (bottom right panel). **b** Top two panels show conservation patterns of 123 HARs associated with 127 high-confidence overlapping CTCF/RAD21-binding sites (top left panel) and 127 high-confidence overlapping CTCF/RAD21-binding sites associated with 123 HARs (top right panel). Bottom two panels show conservation patterns of 69 CTCF/RAD21-binding sites conserved in all Neanderthals' genome (bottom left panel) and 152 human-specific NANOG-binding sites

highly conserved in individual human genomes (bottom right panel). Note that conservation of all HSGRL is consistently at the lowest level in the Neanderthals' genome, suggesting that creation and/or retention rates of HSGRL are enhanced in modern humans. However, HSGRL sequences manifesting the relatively high conservation levels in the Neanderthals' genome appear most conserved in the individual genomes of modern humans as well, including the 41,000-year old Denisovan genome (bottom left panel). Sequences of HSGRL with assigned biochemical functions, e.g., specific TFBS or Lamin B1 (LMNB1)-binding sites, which are residing within HARs exhibit markedly higher conservation levels compared to sequences of HARs harboring the corresponding HSGRL. This conclusion remains valid for HSGRL sequences with assigned specific biochemical or biological functions that were associated with HARs by proximity placement analyses (figures a, b). **c** Direct correlations of conservation profiles of DNA sequences of distinct HSGRL in individual human genomes (top two panels) and markedly different values of genomic fitness scores (GFS) integrating into a single numerical value sequence conservation data of 909 HSGRL in individual human genomes (bottom panel). Note that values of GFS are inversely correlated with the variation coefficients of GFS values in individual human genomes, consistent with the hypothesis that GFS reflects the intrinsic property of an individual genome to create and/or retain the HSGRL

Fig. 6 (continued.)

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