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Title:	Large-Scale Functional Organization of Long-Range Chromatin Interaction Networks
Authors:	Sandhu, K.S. (/jspui/browse?type=author&value=Sandhu%2C+K.S.)
Keywords:	RNA polymerase II Chromatin assembly and disassembly
Issue Date:	2012
Publisher:	Elsevier B.V.
Citation:	Cell Reports, 2 (5), pp. 1207-1219.
Abstract:	<p>Chromatin interactions play important roles in transcription regulation. To better understand the underlying evolutionary and functional constraints of these interactions, we implemented a systems approach to examine RNA polymerase-II-associated chromatin interactions in human cells. We found that 40% of the total genomic elements involved in chromatin interactions converged to a giant, scale-free-like, hierarchical network organized into chromatin communities. The communities were enriched in specific functions and were syntenic through evolution. Disease-associated SNPs from genome-wide association studies were enriched among the nodes with fewer interactions, implying their selection against deleterious interactions by limiting the total number of interactions, a model that we further reconciled using somatic and germline cancer mutation data. The hubs lacked disease-associated SNPs, constituted a nonrandomly interconnected core of key cellular functions, and exhibited lethality in mouse mutants, supporting an evolutionary selection that favored the nonrandom spatial clustering of the least-evolving key genomic domains against random genetic or transcriptional errors in the genome. Altogether, our analyses reveal a systems-level evolutionary framework that shapes functionally compartmentalized and error-tolerant transcriptional regulation of human genome in three dimensions. It is becoming increasingly clear that genes are not autonomous transcriptional units; instead, they physically interact with one another to coordinate transcriptional regulation. Using a network approach, Ruan and colleagues unravel an evolutionarily constrained systems organization of transcription-associated chromatin in the human genome. Their observations provide a possible chromatin-level explanation for how disease-associated mutations evolve and how key cellular genes escape genetic and transcriptional errors.</p>
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