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A scan for linkage disequilibrium across the human genome.

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Linkage disequilibrium (LD), the tendency for alleles of linked loci to co-occur nonrandomly on chromosomal haplotypes, is an increasingly useful phenomenon for (1) revealing historic perturbation of populations including founder effects, admixture, or incomplete selective sweeps; (2) estimating elapsed time since such events based on time-dependent decay of LD; and (3) disease and phenotype mapping, particularly for traits not amenable to traditional pedigree analysis. Because few descriptions of LD for most regions of the human genome exist, we searched the human genome for the amount and extent of LD among 5048 autosomal short tandem repeat polymorphism (STRP) loci ascertained as specific haplotypes in the European CEPH mapping families. Evidence is presented indicating that approximately 4% of STRP loci separated by <4.0 cM are in LD. The fraction of locus pairs within these intervals that display small Fisher's exact test (FET) probabilities is directly proportional to the inverse of recombination distance between them (1/cM). The distribution of LD is nonuniform on a chromosomal scale and in a marker density-independent fashion, with chromosomes 2, 15, and 18 being significantly different from the genome average. Furthermore, a stepwise (locus-by-locus) 5-cM sliding-window analysis across 22 autosomes revealed nine genomic regions (2.2–6.4 cM), where the frequency of small FET probabilities among loci was greater than or equal to that presented by the HLA on chromosome 6, a

region known to have extensive LD. Although the spatial heterogeneity of LD we detect in Europeans is consistent with the operation of natural selection, absence of a formal test for such genomic scale data prevents eliminating neutral processes as the evolutionary origin of the LD.

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