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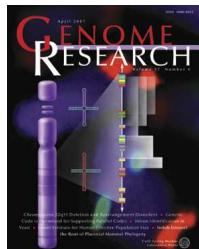
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^{OA}Open Access paper.



Cover A schematic depiction of low copy repeats (LCRs) that mediate genomic instability on chromosome 22 (purple, on the *left*). It has been demonstrated by several groups, including papers in this issue, that palindromic AT rich repeats in 22q11, which could form hairpins and cruciforms (*middle*), mediate translocations between chromosome 22 and other partner chromosomes. Here, the 22q11 region is enlarged and represented by multicolored boxes showing the complex modular LCRs that characterize this chromosome. The BCRL module is depicted by a blue and white striped motif. Zigzag lines represent the location of multiple breakpoints including those for deletions seen in DiGeorge/Velocardiofacial syndrome (DGS/VCFS) patients, duplications seen in Cat Eye syndrome, and the inversion, the translocations, and the deletions involving 22q reported in this issue. Transparent and white overlapping planes show the location of the prevalent LCR-mediated 22q deletions seen in DGS/VCFS and the newer distal deletions reported in this issue, respectively. In the background, a FISH photograph shows the two homologs of chromosome 22 in a patient with a 22q11 deletion. The normal 22 has both the green and red signals, whereas the green signal is lost due to the deletion on the other homolog. (Cover illustration by Connie Funkhouser Balek, Precision Graphics, www.precisiongraphics.com, with concept and design contributions from Tamim H. Shaikh, Ph.D. [For details, see Shaikh et al., pp. 482–491; Gotter et al., pp. 470–481; Kurahashi et al., pp. 461–469; and Babcock et al., pp. 451–460.]