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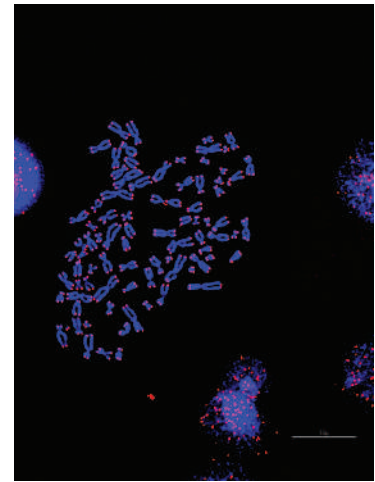
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ABOUT THE COVER

Cultured melanoma cells were treated with colcemid to arrest growth at metaphase, made to swell with a hypotonic solution, fixed with methanol/acetic acid, and dropped on microscope slides to burst them and spread the chromosomes (blue) tipped by telomeres (red) shown in the center area of the cover image (approximately 3000× magnification; courtesy of Brad Stohr and Elizabeth Blackburn). Several nuclei did not burst during slide preparation (also displaying blue chromosomal DNA and red telomeres) and appear adjacent to the spread chromosomes. Chromosomal DNA was stained with 4',6-diamidino-2-phenylindole (DAPI), and telomeres were marked via a fluorescently labeled peptide nucleic-acid probe specific for telomeric DNA repeats. Telomere maintenance (and thus genomic instability) is sustained by the enzyme telomerase in normal cells. Short telomeres are associated with major cancer risk factors including smoking, inflammation, and obesity. Recently, chronic psychological stress, a risk factor for cancer in animal models, also has been shown to take its toll on telomere maintenance in humans. Therefore, the biology of telomeres and telomerase is a potential source of risk and drug-sensitivity markers for integrative, interdisciplinary cancer prevention involving behavioral and basic scientists, epidemiologists, clinical trialists, and biostatisticians. This biology reflects the exciting science of cancer prevention that enables advances in the practice and clinical impact of cancer prevention on several fronts. See articles by Blackburn, Tlsty, and Lippman (beginning on page 394) and Blackburn (on page 393) for more information.



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Cancer Prev Res 2010;3:393-572.

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