

Zeitschrift:	Bulletin der Schweizerischen Akademie der Medizinischen Wissenschaften = Bulletin de l'Académie suisse des sciences médicales = Bollettino dell' Accademia svizzera delle scienze mediche
Herausgeber:	Schweizerische Akademie der Medizinischen Wissenschaften
Band:	33 (1977)
Artikel:	A model for the study of human chromosomes associated with malignancy
Autor:	Koprowski, Hilary / Croce, Carlo M.
DOI:	https://doi.org/10.5169/seals-308124

Nutzungsbedingungen

Die ETH-Bibliothek ist die Anbieterin der digitalisierten Zeitschriften auf E-Periodica. Sie besitzt keine Urheberrechte an den Zeitschriften und ist nicht verantwortlich für deren Inhalte. Die Rechte liegen in der Regel bei den Herausgebern beziehungsweise den externen Rechteinhabern. Das Veröffentlichen von Bildern in Print- und Online-Publikationen sowie auf Social Media-Kanälen oder Webseiten ist nur mit vorheriger Genehmigung der Rechteinhaber erlaubt. [Mehr erfahren](#)

Conditions d'utilisation

L'ETH Library est le fournisseur des revues numérisées. Elle ne détient aucun droit d'auteur sur les revues et n'est pas responsable de leur contenu. En règle générale, les droits sont détenus par les éditeurs ou les détenteurs de droits externes. La reproduction d'images dans des publications imprimées ou en ligne ainsi que sur des canaux de médias sociaux ou des sites web n'est autorisée qu'avec l'accord préalable des détenteurs des droits. [En savoir plus](#)

Terms of use

The ETH Library is the provider of the digitised journals. It does not own any copyrights to the journals and is not responsible for their content. The rights usually lie with the publishers or the external rights holders. Publishing images in print and online publications, as well as on social media channels or websites, is only permitted with the prior consent of the rights holders. [Find out more](#)

Download PDF: 20.08.2025

ETH-Bibliothek Zürich, E-Periodica, <https://www.e-periodica.ch>

A MODEL FOR THE STUDY OF HUMAN CHROMOSOMES
ASSOCIATED WITH MALIGNANCY

HILARY KOPROWSKI and CARLO M. CROCE

Abstract

Nonrandom chromosomal abnormalities have been described in several cancerous or pre-cancerous conditions but, with a single exception (the translocation of the long arm of chromosome 22 to chromosome 9 has been related to chronic myelogenous leukemia), the association between a specific chromosomal abnormality and a specific malignancy remains uncertain. With human cells that have been transformed by the oncogenic virus SV40, however, such an association can be made. When maintained in culture, these cells displayed the permanent hereditary changes characteristic of cancer cells. SV40-transformed cells fused with normal mouse fibroblasts resulted in the formation of hybrid cells in which the human chromosomes were partially and preferentially lost, whereas, the complement mouse chromosomes was retained. Both the presence of SV40-T (tumor) antigen and the rescue of SV40 were associated with human chromosome 7 in the hybrid cell clones.

When nondividing mouse cells such as macrophages were hybridized with human cells transformed by SV40, all hybrid cells able to replicate in culture contained human chromosome 7 with the SV40 genome. Some of the hybrid cells contained only human chromosome 7 and produced hemangiosarcomas when they were injected into athymic "nude" mice. Cells from these tumors grown in culture again showed human chromosome 7 with the SV40 genome. Molecular hybridization studies of these cells indicated that SV40 DNA was integrated into the cellular DNA, and that the number of copies of viral genome per cell equalled the number of human chromosomes 7 per cell. Hybridization of SV40-transformed cells with normal human fibroblasts resulted in the production of clones, of cells that retained the transforming phenotype with a near tetraploid number of chromosomes.

These studies show that in human cells one specific chromosome is responsible both for expressing the function of an oncogenic virus genome and for maintaining the malignant phenotype. They also show that tumorigenicity is inherited as a dominant trait in human cells transformed by DNA virus.

Address of author: Hilary Koprowski, M.D., Director of The Wistar Institute, Thirty-Sixth Street at Spruce, Philadelphia, Pa. 19104 (USA)

