ent close proximity to receptors for colony-stimulating factor 1 (Csfmr) and platelet-derived growth factor (Pdgfr) and to a caudal type homeobox gene (Cdx-1) (3).

ACKNOWLEDGMENTS

We thank M. Krall and M. C. Adamson for expert technical assistance. M. spretus (Spain) mice came from a laboratory colony derived from mice originally trapped in Spain and maintained under NCI contract NO1-CB2-5584, S.C., K.K., and R.M.F. are supported by NIH Grant RO1 CA37351.

REFERENCES

12. Pinnell, S. R., and Martin, G. R. (1968). The cross-linking of and platelet-derived growth factor (Csfmr) and platelet-derived growth factor (Pdgfr) and to a caudal type homeobox gene (Cdx-1) (3).

Received April 17, 1992; revised July 15, 1992

Manganese superoxide dismutase (MnSOD/SOD2) (E.C. 1.15.1.1) is one of a group of enzymes involved in the conversion of oxygen free radicals to hydrogen peroxide (6). MnSOD has been linked both to the removal of toxic oxygen radicals formed during hyperoxia and ischemic reperfusion (1) and to cellular differentiation (1, 5, 10). It is also interesting that cellular transformation and the malignant cell phenotype are both associated with a reduction in the enzyme activity of MnSOD (10).

MnSOD was initially localized to chromosome 6 using enzymatic analysis of mouse/human hybrids (4) and sublocalized to 6q21 following similar analyses to a hybrid with an apparent deletion of chromosome 6q21 (12). Further analysis has demonstrated multiple Taq1 RFLPs using the MnSOD amino acid coding region cDNA as a probe (14). The long arm of human chromosome 6 is a frequent site of chromosome abnormalities in leukemia, neuroblastoma, and melanoma (5, 9, 13). Therefore, we sought to more closely define the regional localization of the MnSOD locus in humans by in situ hybridization and hybrid mapping.

1 To whom reprint requests should be addressed.

Manganese superoxide dismutase (MnSOD/SOD2) to 6q25 by Fluorescence in Situ Hybridization and Somatic Cell Hybrid Mapping

Susan L. Church, *1 James W. Grant, * Eckart U. Meese, † and Jeffrey M. Trent‡

*Edward Mallinckrodt Department of Pediatrics, Washington University School of Medicine, St. Louis Children’s Hospital, 840 Speeher Tower, 400 South Kingshighway Boulevard, St. Louis, Missouri 63110; †Department of Human Genetics, BAU 68, Universitaetsklinik, 6650 Homburg/Saar, Germany; and ‡Departments of Radiation Oncology and Human Genetics, The University of Michigan, MSBII C560, 1152 West Medical Center Drive, Ann Arbor, Michigan 48109-0668

BRIEF REPORTS 823
FIG. 1. (A) Partial restriction map of the 3'-portion of the human MnSOD gene used for in situ hybridization (see text). Phage clones depicted contain exons (represented by black boxes) encoding the COOH--terminal 118 amino acids and all but 114 nt of the 3'-untranslated region of the human MnSOD gene. Corresponding amino acid numbers are indicated. TAA signifies the MnSOD stop codon. Boxes enclosing Poly(A) denote alternative polyadenylation sites. B, BamHI; H, HindIII; and SMA, SmaI, are restriction enzyme cleavage sites. (B) Left: Human chromosome 6 content of a hybrid mapping panel [after (7)]. Below each panel member is the autoradiographic documentation (following Southern analysis) indicating that only HAL26-12 fails to provide a hybridization signal [confirming that SOD2 lies within 6q25]. Right: Examples of FISH using the SOD2 probe shown in A localizing this gene to 6q25 (see idiogram).
The MnSOD probes utilized for the fluorescent in situ hybridization (FISH) study were generated using three HindIII genomic fragments isolated from a human chromosome 6 human/hamster cell line genomic library. The partial restriction map and the exon/intron organization of these clones are depicted in Fig. 1. The 4.3-kb phage clone 9 contains MnSOD exon 4 (amino acids 114–176) and exon 5 (amino acids 177–222 and 557 nt of the 3′ untranslated sequence). The 1.4-kb phage clone 9740 contains additional 3′-untranslated region sequence. The 6-kb phage clone 8 contains the terminal 984 nt MnSOD 3′-untranslated region, including the terminal polyadenylation signal (2). The phage clones were subcloned into pSp-72. One microgram of DNA from the three clones was labeled with biotin and hybridized to human metaphase chromosomes as previously described (8). A total of 25 metaphase cells were examined; 23 of these had "double" fluorescent signals, one on each chromatid over the distal long arm of chromosome 6. Only chromosomes in which both chromatids had a signal were included for analyses, making the background hybridization extremely low. The same cells hybridized for FISH signals and catalytic metal ions in human disease: An overview. In "Methods in Enzymology" (L. Packer and A. N. Glazer, Eds.), Vol. 186, pp. 1–85, Academic Press, San Diego.


**Assignment of the Human CD30 (Ki-1) Gene to 1p36**

**Christa Fonatsch,* Ute Latza,† Horst Dürkop,‡ Harald Rieder,* and Harald Steint†**

*Arbeitsgruppe Tumorcryogenetik, Institut für Humangenetik, Medizinische Universität zu Lübeck, Ratzeburger Allee 160, D-2400 Lübeck, Germany; and †Institut für Pathologie, Klinikum Stellgitz, Freie Universität Berlin, Hindenburgdamm 30, D-1000 Berlin, Germany

Received February 10, 1992

In Hodgkin’s disease (HD) the lymphoid activation antigen CD30 (Ki-1) is expressed on the tumor cells (9). Furthermore,