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Jeffrey M. Trent, Ph.D., is Scientific Director of the National Human Genome Research Institute (NHGRI), and also serves as Chief of NHGRI's Cancer Genetics Branch. He is also an Adjunct Professor of Oncology at the Johns Hopkins University. He specializes in studying the molecular changes related to the predisposition and progression of cancer. After earning his undergraduate degree from Indiana University, Dr. Trent received M.S. and Ph.D. degrees in genetics from the University of Arizona. Prior to his arrival at the NIH, Dr. Trent was the Emanuel N. Maisel Professor of Oncology and Professor of Radiation Oncology and Human Genetics at the University of Michigan, Ann Arbor. He also served as Director of the Division of Cancer Biology, and Director of Basic Sciences in the University of Michigan's Comprehensive Cancer Center. In 1993, Dr. Trent came to the National Human Genome Research Institute to establish and direct its Division of Intramural Research. Under his guidance, the division has become a major research center in human genetics.

Mining melanoma with microarrays

The development and progression of cancer is accompanied by complex changes in patterns of gene expression. In the USA, malignant neoplasms of the skin are the most common cancers of humankind. Incidence rates of melanoma have risen especially steeply since the mid-1970s with an almost complete absence of significant advances in non-surgical treatment of advanced malignant melanoma over this time period. Efforts to reduce mortality must rely on earlier diagnosis, but also on understanding the biology and genetics of this difficult disease. We have used high-density microarrays to search for differences in gene expression profiles associated with melanoma. Applications of multiple data analysis and integration methods including multidimensional scaling to represent the relationships among cell lines and tumor biopsies will be presented in order to document a consistent pattern of gene expression characterizing a subset of these cancers. This information will be related to clinical and known biologic information. These data are providing the leads for further investigation of the genetic basis of the tumorigenic phenotype of melanocytic lesions.

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