MEETING REPORT

THE MOLECULAR BIOLOGY OF CANCER

BOSTON—It was in a spirit of guarded optimism that *Nature*'s editor, John Maddox, welcoming over 400 scientists and physicians to a conference on the Molecular Biology of Cancer, spoke of the rapidly dissolving boundaries between basic research and clinical oncology. While understanding the molecular mechanisms of oncogenesis may not contribute very much to preventing neoplastic disease, as Richard Peto of Oxford University cautioned, that understanding is certain to have a profound impact on cancer diagnosis and therapy.

Robert Gallo of the NIH presented his very exciting work on the relationships between human T cell lymphotropic viruses. A kind of complementarity exists between HTLV-I, a chronic transforming virus, and HTLV-III, a genetically related virus that has quite opposite effects. One has the feeling that continued study of this system will soon lead to a very basic understanding of how these viruses cause their respective disease manifestations.

In his extended discussion, which went well beyond the results presented in the recent paper in *Science*, Dr. Gallo raised the possibility (as did by Samuel Broder presenting the clinician's view) that HTLV viruses may be involved in a much wider range of lymphoid neoplasias than is currently believed. In support of this, he reported finding HTLV-I virus in the T cells of patients with B cell lymphomas. One attractive hypothesis is that virus infected T cells produce a lymphokine that activates B cells to a malignant state.

As expected, a majority of the conference's talks focused on some aspect of the genetics of cancer. Robert Weinberg of M.I.T. reviewed the ideas behind the classification of oncogenes into two complementation groups: the family of *ras* oncogenes, which are transforming and confer anchorage-independence on cells; and the *myc*-like oncogenes, which are immortalizing and allow the *in vitro* establishment of cell lines. He went on to discuss recent work that provides evidence for perhaps a third group of autocrine-type oncogenes and reported the identification of a large cell surface protein (p185) whose structural gene shows homology with *erb-B*, which encodes a truncated epidermal growth factor receptor. The p185 protein is not encoded by the EGF receptor gene however.

Michael Sporn of the National Institute of Health continued the discussion of autocrine cancers, and raised a number of other points that were also pervasive themes of the conference. In his discussion of transforming growth factor β (TGF-β), he pointed out that in some tumor-derived cell lines the protein antagonizes the anchorage independent growth of cells. This bifunctionality of a gene product involved in transformation points to the idea that TGF-β acts as a negative effector of cell proliferation. It raises the idea, once again, that loss-of-function mutations may be as important as either mutations that change function, or mutations that lead to overproduction of a proto-oncogenic product. Raymond White from the University of Utah echoed this in his discussion of retinoblastoma, which appears to be a recessive cancer.—**Harvey Bialy**