Lymphology 23 (1990) 102-103

ROUND TABLE PARTICIPANTS

Top row, left to right: T. Ryan, M.H. Witte, T. Shirai, J. Hay, A.A. Gottlieb. Bottom row, left to right: R. Good, J.R. Sninsky, K. Tenner-Racz, P. Racz, J.M. Yoffey.

SUMMARY OF PANEL DISCUSSIONS

PANEL DISCUSSION #1 opened with a consideration of animal models of AIDS, their applicability to the human disease, and associated opportunistic neoplasms, specifically lymphoma, found in murine models. Discussion then turned to Kaposi sarcoma and specifically whether this multicentric tumor is indeed "opportunistic" due to underlying immunosuppression or somehow related directly to a retroviral or other infectious agent. The same question was raised regarding lymphomas associated with AIDS, and possible mechanisms of retroviral-induced malignancies were reviewed. The panel then turned to the phenomenon presented by Dr. Hay of "virus shut-down" in an acutely infected lymph node, and specifically whether lymph flow and protein transport were also affected, which Dr. Hay indicated was not the case. Dr. Good then explored the implications of the lymphogenous route of viral infection (described by both Drs. Yoffey and Witte), specifically in relation to the manifestations of HIV-1 infection and the development of Kaposi sarcoma, which he emphasized occurs extremely rarely in children with HIV infection or intravenous drug addicts. Dr. Witte suggested that the lymphatic route of drainage from infections originating in the genitalia, lower extremities, or even oral cavity may be crucial in the development and distribution of KS lesions (AIDS-associated or not), independent of whether a specific etiologic infectious agent is involved. Dr. Ryan wondered about the KS-promoting role of endothelial growth factors circulating in lymph and tissues and suggested that the presence of edema fluid itself may be a stimulus to migration of endothelial cells and their ultimate malignant transformation.

PANEL DISCUSSION #2 focused on the variety of target cells infected or affected by HIV-1 and the kinds of functions altered in these cells. Analogies were drawn to other viral infections, which lead to incorporation of viral sequences into the genome, cell death, alterations in protein synthesis, syncytia formation, and various transient effects. The applicability of the polymerase chain reaction (PCR) to the detection and elucidation of immunodeficiency and other lymphologic syndromes, including those of genetic as well as known viral origin, was discussed along with problems with the method, including false-positives and false-negatives, new techniques to handle autopsy specimens, paraffin sections, or poorly preserved tissues. The ability of PCR to detect retrovirus in different types of cells was compared. The role of cytokines in activating or suppressing HIV infection was then discussed, and specifically, which drugs used in AIDS treatment potentiate (or suppress) endogenous cytokine formation. The panel then considered the mechanism of action of different immunomodulator drugs and how some immune functions might be enhanced and others suppressed, on balance favoring stimulation.