

KEY QUESTIONS POSED BY ROUND TABLE PARTICIPANTS

What is the causal agent of Kaposi's sarcoma? A retrovirus?

Why are only some homosexuals presenting with Kaposi's sarcoma?

What is the nature of Kaposi's sarcoma? True malignancy or...?

Can lymphaticovenous connections be demonstrated by imaging techniques in the earliest stages of Kaposi's sarcoma?

Does lymphaticovenous coupling proceed up the venous system in some inverse relation to immune function?

Is a disturbance in lymphaticovenous differentiation demonstrable in a potential animal model of Kaposi's sarcoma such as avian hemangiomas?

How can the accumulation of HIV particles in the germinal centers (GC) be explained?

What is the basis for the destruction of the dendritic cell network in the germinal center demonstrated by immunolabeling studies?

What is the reason for the membrane alterations of the cells as shown by EM?

Is Kaposi's sarcoma a malignant tumor?

If Kaposi's sarcoma is a malignant tumor, what is the origin of the spindle cell?

Why is Kaposi's sarcoma declining in the AIDS population in contrast to malignant lymphomas which are increasing in frequency?

What determines whether HIV is expressed productively in a given cell? Can this be controlled by supporting the immune system at near normal levels?

What is the basis of the lymphadenopathy? Is it protective, reactive and/or predictive?

Why does initial infection with HIV lead to antibody production, but apparently does not initiate cell mediated immunity against the virus?

Why does Kaposi's sarcoma arise predominantly in these patients rather than other malignancies?

What are the precipitating or second signals [cofactor(s)] for Kaposi's sarcoma with HIV infection?

What is the role of cytomegalovirus in Kaposi's sarcoma?

Does HIV infection elicit cytokines which activate proliferation of lymphatic endothelium?

How does one explain the multicentric nature of KS?

Which type of cells transport the virus in the lymphatics and the submarginal parenchyma?

What is the importance and mechanisms of lymphatic spread of HIV?

Is there a failure of preferential pathways for clearance of macromolecules in AIDS?

Are the changes in the lymph nodes primary or secondary in this respect?

Is there any redirection of macromolecules from their preferential pathways directly into the vascular system in a disease such as AIDS, and if so, is the direct access of antigens into the vascular system a reason for T-cell suppression?

Do germinal centers contain viral proteins of HIV, and do they harbor morphologically intact retroviruses and/or infected cells?

Is there any sign of destruction of follicular dendritic cells? If yes, is it related to the presence of HIV antigens?

In non-IV drug abusers or blood transfusion recipients, are the lymphatics of the genitalia and intestine the major portal of entry and a primary target of HIV, and is the lymphatic endothelial lining important in HIV propagation and the host immune response?

Is Kaposi's sarcoma an epiphenomenon of AIDS or is it rather part and parcel of the pathophysiologic process accompanying the disease; specifically, is it a clue to primary vascular involvement in AIDS accompanying subclinical as well as clinically manifest lymphostasis?

Even if effective antiviral agents or vaccines are found, will they work *in vivo* and in large populations?

Is "AIDS" a non-specific final common pathway of tissue response seen in a wide variety of lymphologic and related syndromes? Are KS and KS-like lesions likewise a final common pathway of tissue response (viz. "exuberant" lymphangiogenesis)?

Does autoimmunity play a role in the pathogenesis of AIDS?

What genetic (immunogenetic) factors influence the development of AIDS?

Do MHC-directed autoantibodies occur in AIDS?

Do angiogenesis factors cause Kaposi's sarcoma?

Do angiogenesis inhibitors cause repression of Kaposi's sarcoma?

Does AIDS alter the bioregulation of angiogenesis?