



## Letter

# Kaposi's Sarcoma Herpesvirus in Oral Kaposi's Sarcoma

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Kaposi's sarcoma (KS) remains the most common HIV-associated tumour. In 50% or more of patients with HIV-related mucocutaneous KS, the lesions are oral or perioral and are often an initial or early manifestation [1]. An infective aetiology to KS has been suggested in view of the high frequency of KS in HIV-infected homosexual males, the association of KS with close faecal contact and possible geographic clustering of affected patients [2–6]. DNA sequences of the provisionally termed Kaposi's sarcoma herpesvirus (KSHV) have been identified in cutaneous KS from patients with AIDS, Mediterranean and African KS, and iatrogenically immunosuppressed allograft recipients [7–13]. The precise frequency of detection of KSHV in KS lesions from the oral cavity is, however, not known [13].

DNA was extracted with proteinase K from paraffin-embedded oral KS tissue from 11 homosexual males with AIDS-related mucocutaneous KS, resuspended in distilled water at a concentration of 0.1 µg/µl, and stored at 4°C. Control DNA specimens were derived from the BCBL 1 cell line. All DNA samples were confirmed to be amplifiable using PCR primers specific for a region of the human β globin gene. PCR primers were synthesised to amplify a 233 bp region of the sequence associated with AIDS-related KS. Amplification products were visualised on 2% agarose gel containing ethidium bromide. Sensitivity of the PCR procedure was attained and specificity assured by subjecting a proportion of the PCR products to a further amplification step using nested primers thereby generating a 210 bp product.

Five of the 11 samples of oral KS were positive for KSHV. It is evident from this study of KSHV in the oral cavity that KSHV DNA may be present in AIDS-related oral KS. As with cutaneous AIDS-associated KS [8] and African KS [12], KSHV DNA may, however, not always be detected in lesional tissue. Despite this and other conflicting data (e.g. KSHV DNA in non-KS malignancies and non-neoplastic cutaneous lesions in immunosuppressed patients [14, 15]), KSHV probably has a primary role in the pathogenesis of KS. KSHV DNA is present in endothelial and spindle cells of all forms of KS [16], detection of KSHV DNA in peripheral blood mononuclear cells of AIDS patients correlates with the presence of KS and, in asymptomatic HIV-infected persons,

KSHV DNA in peripheral leucocytes predicts KS progression [17].

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