spersed among dermal collagen bundles. The factor XIIIa–positive spindle-shaped cells were not S-100 positive, which rules out a Langerhans cell or a Schwann cell origin (*9*). As in normal skin, where factor XIIIa–positive dermal dendrocytes are closely associated with blood vessels, many factor XIIIa–positive spindle-shaped cells in the KS lesions maintained an angiocentric configuration. Occasional factor XIIIa–positive spindle-shaped cells also contained phagocytic hemosiderin deposits, which would be in agreement with the previously recognized ability of dermal dendrocytes to engulf pigments (*5*). In the last two AIDS-associated KS patients, the spindle-shaped cells were identified in cryostat sections as also staining with antibody to an antigen (LFA-1) that has been associated with lymphocyte function (Fig. 3), confirming that they were bone marrow–derived mononuclear cells and not fibroblasts or endothelial cells (*11*).

These results suggest that the factor XIIIa–expressing dermal dendrocyte may be the cell of origin for the spindle-shaped cell population in AIDS-associated KS lesions and in the KS cell line (*4*). Our results confirm an earlier suggestion (*2*) that the spindle-shaped cells in KS lesions are related to the reticuloendothelial cell system by demonstrating a monocyte/macrophage lineage marker (factor XIIIa) in the dermal dendrocytes that make up the spindle-shaped cell population. The cause of the variation in the extent of factor XIIIa expression by the spindle-shaped cells in AIDS-associated KS lesions is not known, but because there was a greater expression when there were more lymphocytes in the infiltrate, we believe that local modulation by interferon-γ may play a role (*12*). In one of our AIDS patients who developed psoriasis, a skin disease with prominent blood vessels and inflammation, we observed large numbers of factor XIIIa–positive dermal dendrocytes in the papillary dermis immediately beneath the hyper epidermis (Fig. 4). These results suggest that upon exposure to human immunodeficiency virus type 1 (HIV-1), factor XIIIa–positive dermal dendrocytes may be activated that can give rise either to expansile lesions such as KS or to altered local immune reactions producing psoriasis in genetically susceptible individuals. In either case, it would appear that the cutaneous manifestations of HIV-1 infection such as KS and psoriasis may represent hyperactivity of the dermal dendrocytic component of the immune system, rather than an immunodeficiency (*13*). If factor XIIIa dermal dendrocytes in vivo possess the same repertoire of cytokine production (including interleukin-1 and basic fibroblast growth factor) as do the cultured KS spindle-shaped cells (*4, 14*), then it is possible to envisage the molecular basis by which HIV-1–activated dermal dendrocytes could stimulate endothelial, keratinocyte, and mononuclear cell proliferation in AIDS-associated KS and psoriatic lesions (*15*).

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Response: Nickoloff's comment is interesting. It could be that the true primary neoplastic cell of Kaposi's sarcoma (KS) is the spindle cell, as he suggests and as we and others believe. Nickoloff also argues that the long-sought-after origin of these cells may be the so-called dermal dendrocytes and suggests that the spindle cells we succeeded in culturing for the first time from KS patients may indeed be these cells. Indeed, many of the properties of our cells are similar to properties of macrophages. However, such cells are difficult to distinguish from endothelial cells. For instance, in terms of uptake of low density lipoprotein, binding of *Ulex europeus* lectin, and presence of cytokeratin our cells are more like endothelial cells than macrophages. Of course, dermal dendrocytes may be in the macrophage lineage but different from classical macrophages in these respects. However, KS is not limited to the skin, but occurs in numerous other tissues. Thus, the suggestion seems far from conclusive, but it does merit testing our cultured KS cells with the antibodies to factor XIIIa and LFA-1. It will be critical to confirm the specificity of the antibody to LFA-1.

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