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A novel animal model for the study of AIDS and non-AIDS associated skin disorders

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Background

Since the emergence of AIDS in the early 1980's; HIV infection has had a major impact on the field of dermatology. Almost all patients with AIDS suffer from dermatologic lesions, many primarily related to HIV infection and some others similar in nature to those seen in the non-HIV infected population but more chronic and more resistant to treatment. We developed an HIV-1 Transgenic (Tg) rat that shows mild, moderate and severe forms of skin pathologies, similar as those seen on patients in all stages of AIDS (PNAS, July 31st, 2001). The pathogenesis of HIV-1 associated skin disorders is poorly understood and of great complexity, but as we have better understanding of these diseases we will be able to develop improved therapeutic strategies. The objective of this study is to validate that the Tg rat represents a suitable model for the comprehension of the pathogenesis of skin disorders associated with HIV-1 infection and for many other skin diseases of autoimmune or co-infectious origin.

Methods

The vector was generated as follows: a 3-kbp SphI-MscI fragment encompassing the 3' region of gag and the 5' region of pol was removed from pNL4-3, an infectous proviral plasmid, to make the noninfectious HIV-1 gag-pol clone pEVd1443. An env region that expresses gp-120 of HIV-1 was contained. A 7.4-kbp EaeI-NaeI fragment containing the provirus and host cell flanking regions was microinjected into fertilized one cell Sprague-Dawley x Fisher 344/NHsd F1 eggs. Histological and immunological studies were performed in pathological samples of the Tg rats.

Results

Skin lesions were noted in the sampled colony at a rate of approximately 20%. The skin manifestations were described as mild to severe lesions located on the tail, feet, anus, vagina, scrotal sacs and on the haired portion of the entire body. The lesions were described as scaly raised epidermal plaques, and ulcerated lesions especially on the tail. Histologically the lesions showed epidermal hyperplasia, hyperkeratosis, papylomatous and psoriatic-like lesions. Infiltrates of neutrophils, eosinophils, mononuclear cells and mast cells were observed in the suprabasal layer. Lesions in the most severely affected rats resembled erythema multiforme. Immunostaining for HIV-1 gp120 showed signal in epidermal and dermal cells. Serum cytokines showed an increase in IL-4, and IL-5.

Conclusion

The results support a role for HIV-1 gene products, especially gp-120, and Th2 related cytokines in the pathogenesis of HIV-1 related skin disorders and probably in some autoimmune conditions such as psoriasis and erythema multiforme.