et al, 1994a), and that (ii) TIL within 24 different skin-related benign and malignant melanocytic lesions preferentially use three identical AV families also present in the TCR repertoire of normal unaffected skin (Strohal *et al*, 1994b). These observations, which are in keeping with Musette's CLA expression data in halo nevi and TCR clonotype analyses by Drs. Puisieux and Musette, suggest that lymphocytes infiltrating cutaneous melanoma belong to a T cell subset functionally associated with the skin. Hence, to evaluate the concept of distinct organ-related immunologic compartments directly influencing the quality and quantity of T cell mediated antimelanoma host responses, the study herein discussed was successfully intending to define TCR-AV/BV usage, CDR3 region diversity, and melanoma-associated antigen expression patterns in skin-related and noncutaneous coexisting/subsequent metastatic melanoma sites. Notwithstanding the technology being used to analyse the TCR repertoire in TIL of different immunologic compartments, we wish to emphasize again that functional *in vitro* assays are needed to learn more about the actual target profiles of these locally expanded TCR clonotypes.

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Human Herpesvirus 7 and Pityriasis Rosea

To the Editor:

Pityriasis rosea (PR) is a common, acute, self-limited papulosquamous disorder that usually lasts for 4–7 wk. Several clinical features have suggested a viral cause, including occasional association of prodromal symptoms, clustering in families or communities, and an increased incidence in patients with decreased immunity, such as in pregnant or bone-marrow transplantation patients. It has been reported that human herpesvirus 7 (HHV7) was present in cell-free plasma, peripheral blood mononuclear cells, and lesional skin from all 12 PR patients examined (Drago *et al*, 1997a, b); however, their conclusion has yet to be verified by other studies.[1] We investigated the presence of HHV7 sequences and antibodies against HHV7 in 81 plasma samples from 36 patients with PR.

The plasma samples were suspended in K-buffer and DNA was extracted with phenol and chloroform (Drago *et al*, 1997a). All examinations were performed under code. We used previously described primers (Drago *et al*, 1997a, b) to amplify a 264-bp HHV7-DNA that was located between base pairs 83998 and 84261 of the published sequences (Nicholas, 1996). Of 36 patients, 16 were positive at one time point and one (No. 11) was positive at two time points (**Fig 1**). The direct sequencing of the polymerase chain reaction products revealed that all but one plasma sample had exactly the same sequence as in the database, and that the remaining sample (No. 12) had a nucleic acid alteration: G at base pair 84142 was changed to C. The specificity of polymerase chain reaction was demonstrated by the lack of HHV7 sequences in the 31 plasma samples from age- and gender-matched healthy controls.

The same plasma samples were further investigated for the prevalence of antibodies to HHV7. An indirect immunofluorescent assay was carried out according to previously described procedures (Wyatt *et al*, 1991) with some modifications. SupT1 cells infected with HHV7 (Sato strain, provided by SRL, Tokyo, Japan) were fixed and then incubated with 2-fold serially diluted serum. The titer was defined as the reciprocal of the final dilution that gave a positive response. All cases, including 31 healthy controls, were negative (<10) for IgM. A significant increase (>4) of IgG was detected in one PR patient (No. 10); however, neither a significant increase nor a decrease in titers of IgG in the other PR patients was observed.

We cannot fully confirm a direct causal relationship between PR and HHV7 because the viral sequences were transiently found in about half of the patients and the result of IFA did not suggest the reactivation of HHV7 in PR. An insufficient number of viruses may be reactivated during the period of PR to provoke a significant increase of antibodies against HHV7, or the causative factor of PR may be heterogeneous and unattributable to a single infectious agent. The virus was found up to 7 wk after the first consultation, the same amount of time that the symptoms of PR usually persist, although this might have reflected decreased immunity of the host.

Manuscript received April 14, 1999; accepted for publication April 30, 1999.

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[1]Kempf W, Adams V, Kleinhans M, Burg G, Panizzon RG, Campadelli-Fiume G, Nestle F: Human herpesvirus and pityriasis rosea. *J Invest Dermatol* 110:666, 1998 (abstr)

To confirm a relationship between HHV7 and the pathogenesis of PR, viral mRNA and antigens in the lesional skin must be identified.

The authors thank Dr. Seishi Ogawa for help with the DNA sequencing.

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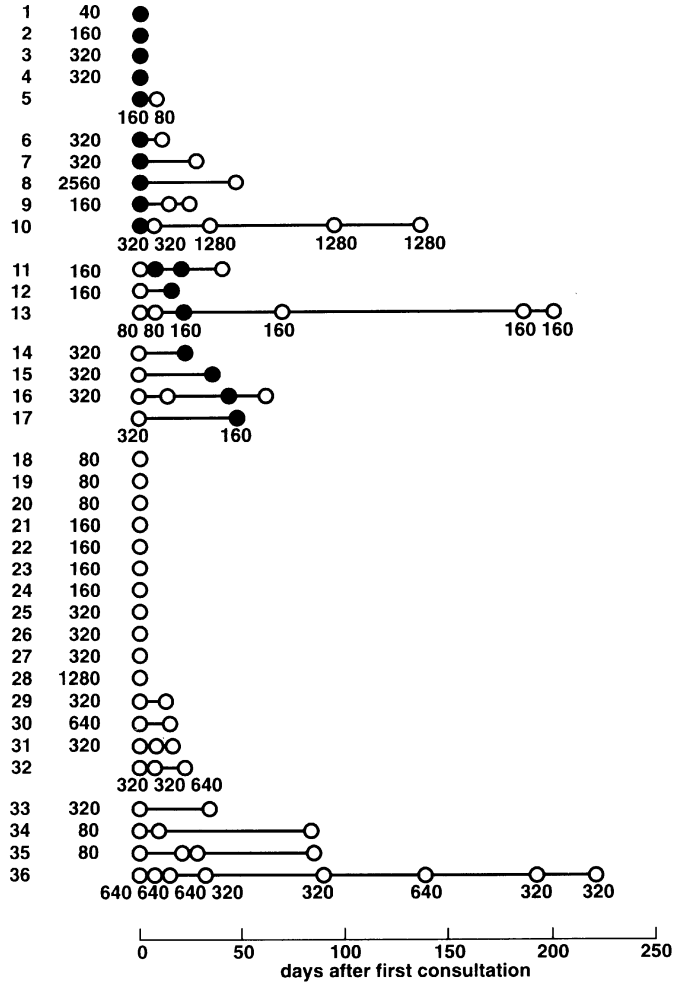

Figure 1. Longitudinal data obtained by nested polymerase chain reaction and the immunofluorescent assay on 36 study patients with PR. ●, Positive results of the nested polymerase chain reaction amplification of HHV7-DNA; ○, negative results. Numbers imply anti-HHV7 IgG titers of the immunofluorescent assay; titers unchanged during the follow-up are shown in the left column.