An association between pityriasis rosea and human herpesvirus 7 (HHV-7) has been reported but remains controversial. The purpose of the present study was to investigate the association between HHV-6 and HHV-7 with pityriasis rosea. Fifteen patients aged 6–54 years with a diagnosis of pityriasis rosea and 15 age-matched controls were recruited. None of the patients had HHV-6 or HHV-7 DNA detected by polymerase chain reaction in the acute or convalescent plasma specimen. In the acute peripheral blood leukocytes specimen, 3 patients and one control had HHV-6 DNA detected (p = 0.299; NS), while 7 patients and 5 controls had HHV-7 DNA (p = 0.355; NS). Antibody to HHV-6 was detected in the acute specimen of 13 patients and 13 controls, while antibody to HHV-7 was found in all 15 patients and controls. We thus found no evidence of recent HHV-6 or HHV-7 infection in patients with a diagnosis of pityriasis rosea. 

Keywords: skin disease; virus; primary infection; reactivation.

(Material and Methods)
with PR were aged between 6 and 54 years (mean age: 26.8 years). Three were children aged 6, 9, and 11 years. Six (40%) were males and 9 (60%) were females. Apart from an English lady, aged 29, the others were Chinese. Clinical features and findings of the 3 children have been reported previously (8).

A lesion biopsy was performed in one study subject with atypical PR features, revealing focal spongiosis with perivascular lymphocytic infiltrates compatible with PR. Biopsy was not performed for the other 14 patients with typical PR.

The 15 matched control subjects ranged in age from 8 to 52 years (mean age: 27.3 years).

PCR for HHV-6 DNA was performed in acute and convalescent blood samples of all 15 patients and in the initial blood of the controls. None (0%) of the patients had HHV-6 or HHV-7 DNA detected by PCR in the acute or convalescent plasma. Three (20%) patients and one (7%) control had HHV-6 DNA detected in the acute PBL specimen (p = 0.299; NS). Seven (47%) patients and 5 (33%) controls had HHV-7 DNA detected in the acute PBL specimen (p = 0.355; NS). Antibody to HHV-6 was detected in the acute specimen of 13 (87%) patients and 13 (87%) controls, and antibody to HHV-7 was found in all 30 (100%) patients and controls.

One of the controls, an 11-year-old boy presenting with fever and acute otitis media, had evidence of a primary HHV-6 infection – HHV-6 DNA present in the acute plasma specimen in the absence of HHV-6 antibody and seroconversion demonstrated in the convalescent blood. Otitis media is in fact one of the manifestations of primary HHV-6 infection (9).

DISCUSSION

Neither HHV-6 nor HHV-7 appears to play an aetiological role in our series of 15 patients with PR. Although HHV-7 DNA was detected in PBL in 47% of patients with PR, this was not significantly different from that in the matched controls. More importantly, HHV-6 or HHV-7 viral DNA was not detectable in the plasma of any of these patients. Our results concur with those of Kempf et al. (4) and Kosuge et al. (5) suggesting lack of good evidence of active or primary HHV-7 infection in patients with PR. PCR for HHV-7 DNA in plasma was not performed in either study. PR might be associated with primary infection or reactivation of HHV-7 and detection of HHV-7 in the plasma may be useful in detecting either. In our study, we investigated HHV-6 and HHV-7 DNA in the plasma as well as in PBL and we had age- and sex-matched controls available for comparison.

Based on the detection of antibodies to the Epstein-Barr virus (EBV) early antigen, Bonafe et al. (10) reported an association between PR and EBV. They found no association with influenza A, B, parainfluenza 1, 2, 3, adenovirus, respiratory syncitial virus, Mycoplasma pneumoniae, ornithosis-pasteurella, Q-fever, herpes simplex virus, varicella or cytomegalovirus.

It has been demonstrated in one controlled trial that erythromycin might have been of benefit in patients with PR (11, 12). In addition to its effect on bacteria (including atypical agents), this antibiotic has anti-inflammatory and immunomodulatory effects (13) that could contribute towards its action in PR. If the latter is true, dysfunction of the immune system, in particular cellular immunity, may be important in the pathogenesis of PR. EBV infection has immunomodulatory effects and the age distribution of primary EBV infection matches that of PR. We thus consider it worthwhile that future studies investigate the roles of EBV, atypical bacteria and cellular immune dysfunction in PR.

Since PR is a transient self-limiting disease, recruiting patients in a primary care setting is important. None of our patients required hospitalization and these cases would have been missed if our study had been hospital based. Some patients with PR may be referred to a specialist dermatology department. However, because the waiting list usually exceeds 1–2 months in the local setting, the rash would usually have faded by the time these patients are seen by the dermatologist. The present study had the advantage that the primary care physician had training and certification in both paediatrics and dermatology.

In a case control study of 15 patients with PR, we failed to identify an association of HHV-6 or HHV-7 infection or reactivation in active PR. We conclude that HHV-7 is unlikely to be a significant factor in the pathogenesis of PR. We recommend further studies of PR to be along other directions including EBV, atypical bacteria and cellular immune dysfunction.

REFERENCES