## Human Papillomavirus Type 53

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The cloning and partial characterization of the genome of human papillomavirus type 53 is presented. The virus is a distinct type and is most closely related to human papillomavirus type 30.

The DNA of human papillomavirus type 53 (HPV-53) was cloned from a cervical swab of a pregnant woman without

cytological or clinical abnormality (1). HPV-53 was shown to react positively with the DNAs of many of the known HPV

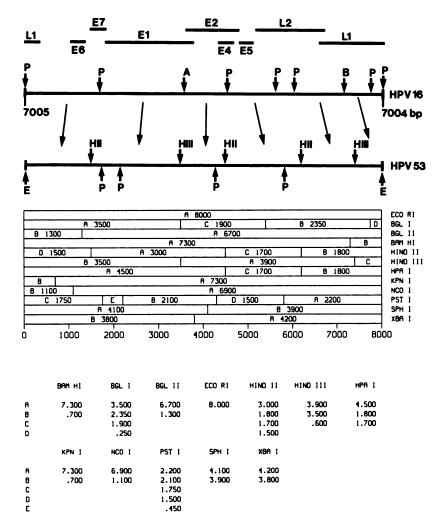


FIG. 1. Genomic alignment between HPV-53 (linearized at the unique EcoRI site [E]) and HPV-16 (linearized at nucleotide positions 7004-7005) DNAs. Subgenomic fragments of HPV-16 (AvaII, A; PstI, P) were labeled with <sup>32</sup>P and hybridized to digested HPV-53 DNA (BamHI, B; HindII, II; HindIII, III) under nonstringent conditions ( $T_m - 40^{\circ}C$ ). The restriction map of HPV-53 is shown. Cleavage sites for various restriction enzymes were determined and plotted on a linear map, using the unique EcoRI site as position 0. The sizes of individual fragments are given below the map. Non-cut enzymes are ClaI, NaeI, PvuI, PvuII, SaII, AvaI, XhoI, and SacI.

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types when tested by Southern blot hybridization under relaxed conditions. Under stringent hybridization condi-

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tions, it reacted weakly with HPV types 8, 18, 32, 43, 44, 45, 48, and 51. Strong homology was detected to HPV-30, and the homology was determined by reassociation kinetics to be 20%. Colinearity of the approximately 8-kilobase genome to that of HPV-30 DNA was demonstrated by heteroduplex analysis. The genomic regions of HPV-53 were located by hybridization with subgenomic fragments of HPV-16 (Fig. 1).

It is not known whether HPV-53 is associated with any kind of genital or extragenital lesion. The prevalence of

HPV-53 infection in the genital tract, however, appears to be very low, because a screening with  $^{32}$ P-labeled HPV-30 DNA, which is closely related to HPV-53, resulted in only 6 positive samples out of 189 cervical swabs obtained from asymptomatic women.

## LITERATURE CITED

1. Schneider, A., M. Hotz, and L. Gissmann. 1987. Increased prevalence of human papillomaviruses in the lower genital tract. Int. J. Cancer 40:198–201.