INTRODUCTION

Group A10 includes several of the old group B viruses with the addition of the Pygmy Chimpanzee papillomavirus (PCPV-1). It consists of human papillomavirus types 6, 11, 13, 44, and 55, viruses primarily associated with orogenital lesions with low oncogenic potential, and PCPV-1. Lorinz et al. classified HPV-6, HPV-11 and HPV-44 as “low risk” viruses [1]. DNA from these three viruses and others in the low-risk class was detected in 20.2% of the low-grade cervical lesions, in 4.2% of the high-grade lesions, and in none of the 153 invasive cancers screened [1].

Many researchers view HPV-6 and HPV-11 together as a functional group [1]. These two viruses are primarily responsible for the benign HPV infection of the anogenital tract. Condylomata acuminata have been shown to harbour HPV-6 or HPV-11 DNA in more than 93% of the cases [1]. Conversely, relatively few HPV-6 and HPV-11 positive genital malignancies have been identified despite extensive international screening. In the IBSCC worldwide investigation of over 1000 cervical tumors, a single HPV-6 and a single HPV-11 were identified [2]. Two cases containing HPV-55 were also identified in this study. One type of malignancy, although rare, is strongly correlated with HPV-6 and HPV-11 infection: Buschke-Lowenstein tumors, the highly differentiated squamous cell tumors of the genital region, are associated almost exclusively with HPV-6 and HPV-11 [3,4]. HPV-44 and HPV-55 have been detected in condyloma acuminata of the genital region (vulvar and penile, respectively) [5,6].

The strong association of HPV-6 and HPV-11 with certain types of genital carcinomas (vulvar and vaginal) appears to be inconsistent with their classification as low-risk. Several explanations have been proposed to explain this anomaly. First, Lorincz et al. suggest the limited number of papillomavirus probes available to researchers may have contributed to the false-positive identification of HPV-6 and HPV-11 [1]. Second, researchers have shown a correlation between oncogenic potential and the presence of a duplicate upstream regulatory region in the genome. A species of HPV-11 with this duplication has been shown to transform baby rat kidney cells and such duplications have been found in carcinomas harbouring HPV-11 and HPV-6 DNA [7–10]. However, alterations in the URR should be considered with caution since rearrangements have been observed following amplification of cloned genomes [11]. Recent investigations suggest that rearrangement of the HPV-6b URR occurred during propagation in E. coli [12]. Rubben et al. suggest that cellular and environmental factors following infection may induce this duplication event and/or other rearrangements leading to acquired oncogenic properties [10]. Cofactors which may contribute to malignancy include alcohol and tobacco use and sexual intercourse during menstrual periods [13, 14].

In addition to their involvement with anogenital tract lesions, HPV-6, HPV-11 and HPV-13 are strongly associated with ororespiratory tract infection. In one study, 72% of all laryngeal papillomas and 25% of all oral papillomas were positive for HPV-6 and HPV-11 DNA [15]. HPV6 and HPV-11 have been detected in benign papillomas infecting almost every epithelial lining of the upper digestive and respiratory tracts. These tissues include the larynx, sinonasal area, lung,
tonsil, tongue, and linings of the oral cavity [7, 15–17]. Unexpectedly, a high percentage (60%) of laryngeal carcinomas have been shown to be positive for HPV-11 DNA [18]. HPV-13 was reported by de Villiers to be present in 13% of all oral papillomas [15]. Specifically, HPV-13 has been highly correlated with oral focal epithelial hyperplasia (FEH), a benign lesion situated primarily on the mucosae of the lower lips and cheeks [19]. This disease is frequently found among Indians in Central and South America and in Eskimos in Greenland and Alaska [19]; the prevalence among Caucasians in the same area is much lower [19]. HPV-13 has also been detected in a case of low-grade cervical dysplasia and in Bowenoid papulosis in an HIV-positive male [19].

Several subtypes of HPV-6 have been identified. Subtype 6a has been isolated from tonsillar carcinoma, lung carcinoma and Buschke-Lowenstein tumors [9,16,20]. The complete genomic sequence of HPV-6a has recently been reported (GenBank accession L41216) [21]. As this variant is so similar to the sequence we published last year (HPV-6b) we have chosen not to print it in hard copy form but to make it available on our World Wide Web site (see Part V). HPV-6b, the prototypical HPV-6 subtype, was initially cloned and sequenced from a benign genital wart [22]. It has been subsequently detected in various genital and upper digestive and respiratory tract lesions. The HPV-6c genome was molecularly cloned from both a respiratory-tract papilloma and a condyloma acuminatum of the cervix [23]. This subtype has also been detected in benign laryngeal papillomas and benign nasopapillomas [24]. The HPV-6d genome, cloned from Buschke-Lowenstein tumors, contains a tandem duplication of 459 base pairs in the noncoding region of the genome [16]. HPV-6e was identified in a genital wart and laryngeal papillomas [23–25]. HPV-6f has been cloned from a benign laryngeal papilloma and a non-inverted nasal papilloma [24, 26]. HPV-6vc was cloned from a rapidly growing vulvar verrucous carcinoma [27]. And, finally, a worldwide study of sequence variation in the HPV-6 and HPV-11 URR demonstrating the phylogenetic spectrum of these viruses has been reported [11]; it is perhaps worth noting that the levels of sequence diversity observed in this study are more consistent with considering HPV-6a and HPV-6b to be variants rather than distinct subtypes.

With the release this year of sequences for HPV-44 and HPV-55, complete genomic sequences are now available for all members of Group A10. We consider HPV-11 and HPV-6b to be “close types”—sequences which qualify to be distinct types under the criterion of ten percent dissimilarity at the nucleotide level, but between which most of the changes are “silent”, causing no difference at the amino acid level Human Papillomaviruses 1994 (Part III). The Pygmy Chimpanzee papillomavirus (PCPV-1), which in last year’s compendium was included in the old group “I,” is close enough to HPV-13 for these to be considered “close types.” A sequence related to PCPV-1 has been identified in a buccal scrape from a chimpanzee (Pan troglodytes; like the reference sequence, the variant is more similar to HPV-13 than many HPV types are to each other [28].

What’s new?

The complete genomes of HPV-44 and HPV-55 are the only new sequences in Group A10 released during 1995. The sequences of other members of this group were published in Human Papillomaviruses 1994 pp. I-B-5, I-B-10, and I-B-14.

References


HPV44

**LOCUS**  HPV44  7833 bp  DNA  VRL  18-JUL-1995

**DEFINITION**  Human papillomavirus type 44, complete genome.

**ACCESSION**  U31788

**KEYWORDS**  .

**SOURCE**  Human papillomavirus type 44.

**REFERENCE**  1 (bases 1 to 7833)

**AUTHORS**  Delius,H.

**JOURNAL**  Unpublished, Sequenced by Hajo Delius, Deutsches Krebsforschungszentrum, Angewandte Tumorvirologie, I.N.F. 506, W-6900 Heidelberg, Germany

**REFERENCE**  2 (bases 1 to 7833)

**AUTHORS**  Farmer,A.D.

**TITLE**  Direct Submission

**JOURNAL**  Submitted (18-JUL-1995) Andrew D. Farmer, HIV Sequence Database, Los Alamos National Laboratory, T-10, Mail Stop K710, Los Alamos, NM 87501, USA

**COMMENT**  HPV-44 is a mucosatropic HPV which to date has not been detected in cervical cancer. Prevalence studies indicate that HPV-44 and HPV-43 have been found in 4% of cervical intraepithelial neoplasms, but in none of the 56 cervical cancers tested (Lorincz et al, J. Virol 63, 2829-2834). During the analysis of approximately 1000 anogenital tissue samples, two new HPV types, HPV-43 and HPV-44, were identified. The complete genome of HPV-44 was recovered from a vulvar condyloma and cloned into bacteriophage lambda. The biopsy was taken from a woman from the Detroit Michigan area. The DNA recovered was a single 7.8 kb BamHI fragment. A possible feature of HPV types associated with malignant lesions is the potential to produce a different E6 protein by alternative splicing. This potential has been found in types HPV-16, HPV-18, and HPV-31. HPV-44 has a potential E6 splice donor at nt 229, but does not contain a potential splice acceptor. Phylogenetic analysis indicates that HPV-44 is most closely related to HPV-55, HPV-6, HPV-11 and HPV-13.

**BASE COUNT**  2383 a  1545 c  1678 g  2227 t

**ORIGIN**  105 bp upstream from beginning of E6 cds

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  E6 orf start ->
  signal ->
  E6 cds ->
  E7 orf start ->
  E2 binding
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  E7 orf start -> -> E2 binding
541 aaactatact acctTAAagg aatggttntt atacgctgaa cttcctgacc cttcctgacc
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-- E4 orf start ->

-- E5 orf start ->

-- E5 cds ->

-- E2 end

-- E4 end

-- E5 end

-- E4 end
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1. **ttataataaat gtaagcttta gaaaagagga gggACCGAAT TCGGTtccaA CCGAAAACGG**
   - E2 binding -> E2 binding ->

2. **61 TTATATAAaa accagcccaa aaatattaacg aagcggggaat cAGGaaagt gcaaatgcct**
   - E6 orf start ->
   - E6 cds ->
   - signal ->

3. **611 ccacggtgcg aacaagttat gcaagagattc aaaaatctt atgacaccatt**
   - E7 orf start ->
   - E7 binding ->

4. **541 ctaacctccacc tTGAGaaaggg ttttttatta gctgtgttattt ggcagaaaaaag TGatggaaagt gcaaatggct**
   - E7 orf start ->
   - E7 binding ->

5. **E2 binding ->**

6. **E1 orf start ->**

7. **E2 binding -> E1 orf start ->**

---

**I-A10-158**

**OCT 95**
I-A10-159

HPV55

E2 orf start ->

NH/50 terminus unknown

E4 orf start ->

NH2 terminus unknown

E5 orf start ->
<table>
<thead>
<tr>
<th>Start</th>
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<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>4501</td>
<td>5041</td>
<td>L1 orf start</td>
</tr>
<tr>
<td>5041</td>
<td>5701</td>
<td>L1 cds</td>
</tr>
<tr>
<td>5701</td>
<td>6541</td>
<td>L1 cds</td>
</tr>
<tr>
<td>6541</td>
<td>7201</td>
<td>L1 cds</td>
</tr>
<tr>
<td>7201</td>
<td>7201</td>
<td>L1 cds</td>
</tr>
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</table>

**L1 orf start**

- **Start:** 4501
- **End:** 5041
- **Description:** L1 orf start

**L1 cds**

- **Start:** 5041
- **End:** 5701
- **Description:** L1 cds

**L1 cds**

- **Start:** 5701
- **End:** 6541
- **Description:** L1 cds

**L1 cds**

- **Start:** 6541
- **End:** 7201
- **Description:** L1 cds

**L1 cds**

- **Start:** 7201
- **End:** 7201
- **Description:** L1 cds

---

**E2 binding**

- **Start:** 7391
- **End:** 7401
- **Description:** E2 binding

---

**Signal**

- **Start:** 7291
- **End:** 7301
- **Description:** Signal
7681 agccaacttt taaaagcatt ttttgctact aacactacat ttttgacag ttactgtag 
7741 ttttataaaa tgagtaacct aaggtcacac acctgcaACC GGTATCGGTt gaaacacacc 
    E2 binding ->
7801 ctgtacattt ccttattata gt

//
Group A11 Sequences

**INTRODUCTION**

Group A11 is made up of three viruses (HPVs 34, 64, and 73) formerly placed in old Group B. It is a group primarily associated with orogenital lesions of low oncogenic potential. The reference clone of HPV-73 has not been released as of this writing, however, the sequence HPVMM9, treated in last year's compendium, has been found to be a variant of HPV-73.

These viruses have been predominantly linked to anogenital lesions. HPV-34 was initially isolated and cloned from a squamous cell carcinoma of Bowen’s type and subsequently detected in a genital intraepithelial neoplasia and periungual Bowen’s disease [1]. A study which probed lesions with Bowen’s disease and squamous cell carcinomas for HPV-34 DNA, reported only one case of positive hybridization, indicating that HPV-34 infection of this nature is relatively rare [1]. HPV-64, a recently identified virus, was cloned and isolated from a vulvar intraepithelial neoplasia [2]. MM9 was derived from a genital swab specimen. Initial prevalence data for MM9 is similar to that obtained for characterized “intermediate-risk” viruses [3]. It was observed in 6 cancers (0.6%) in the IBSCC study [4], where it is referred to as PAP 238a.

**What’s new?**

No new sequences in Group A11 were released during 1995. The sequences of members of this group were published in *Human Papillomaviruses 1994* pp. I-B-19, 26, 27, and I-I-37.

**References**


Isolated “A” Sequences

<table>
<thead>
<tr>
<th>CgPV</th>
<th>CP8061</th>
</tr>
</thead>
<tbody>
<tr>
<td>HPV-54</td>
<td>LVX82</td>
</tr>
<tr>
<td>MM7</td>
<td></td>
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</tbody>
</table>

This set of viruses contains supergroup A taxa that do not cluster with any groups within that supergroup.

HPV-54 and CP8061 primarily infect genital mucosa. HPV-54 was isolated from a penile Buschke-Lowenstein tumor in conjunction with HPV-6 DNA. Initial prevalence data indicates that it is a rare genital HPV type [1]. CP8061 was isolated from a cervical lavage sample obtained through clinical studies conducted in the state of New Mexico among a tri-ethnic population [2].

LVX82, which differs by only a few nucleotides from HPVMM7, was isolated from an Amazonian Indian population [3]. HPVMM7 was identified through studies conducted in the state of California. Initial prevalence data for MM7 are similar to those obtained for characterized “intermediate risk” viruses [4]. All samples were obtained from cervical lavages or genital swabs. LVX82/MM7 was detected once (0.1%) in the IBSCC study [5].

What’s new?

My0911 fragments of the four human viruses CP8061, HPV-54, LVX82, and MM7 were published in *Human Papillomaviruses 1994* on pages I-F-45, I-F-40, I-E-12, and I-E-13, respectively. The complete genome of HPV-54 has become available in the past year and is published here in its entirety. The novel Colobus monkey papillomavirus (CgPV), is represented by two partial sequences, CgPV1L1 and CgPV1E1, on the following pages.

References

[1] Favre, M., Kremsdorf, D., Jablonska, S., Obalek, S., Pehau-Arnaudet, G., Croissant, O., and Orth, G. Two new human papillomavirus types (HPV54 and 55) characterized from genital tumours illustrate the plurality of genital HPVs. *Int J Cancer* 45: 40–46 (1990)


<table>
<thead>
<tr>
<th>LOCUS</th>
<th>HPV54</th>
<th>VRL</th>
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<td>SOURCE</td>
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<tr>
<td>REFERENCE</td>
<td>1 (bases 1 to 7759)</td>
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</tr>
<tr>
<td>AUTHORS</td>
<td>Delius,H.</td>
<td></td>
<td></td>
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<tr>
<td>TITLE</td>
<td>Direct Submission</td>
<td></td>
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<tr>
<td>JOURNAL</td>
<td>Unpublished, Sequenced by Hajo Delius, Deutsches Krebsforschungzentrum, Angewandte Tumorvirologie, I.N.F. 506, W-6900 Heidelberg, Germany</td>
<td></td>
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<tr>
<td>COMMENT</td>
<td>HPV-54 was first isolated from a patient with condyloma acuminata.</td>
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<tr>
<td>BASE COUNT</td>
<td>2326 a 1530 c 1718 g 2185 t</td>
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<tr>
<td>ORIGIN</td>
<td>102 bp upstream from beginning of E6 cds</td>
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</table>

<table>
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<tr>
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<th>COUNT</th>
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<tr>
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<td>2326</td>
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<td>c</td>
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<td>g</td>
<td>1718</td>
</tr>
<tr>
<td>t</td>
<td>2185</td>
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**E2 binding** — E2 binding —

1 TaactaTAAA catgatttat aaataaagg aggaggccaA AAGGGtcaA CCGAAACCcG

**E7 orf start** —

541 tggaaatgtg gctacaattg aggatatgct ccttgtgatt taaaccagaA agtttgacct

**E1 orf start** —

841 aaggtcagaa ggagggacg actggctgtgta atgttgtggttt tttgtggaA gcattaattgtag

**E1 cds** —

1481 atagagcgtg caataataat gcaacgctgc acagacgcg caccccggaa tggctggcca
5401 gtaacccac agttccccTA Actgcctctaa cgccatatac acctatacc acatccttaa 
L1 orf start -> 
5461 ggcctctactg agttcacaacc cccatatgct cccacgctcc tataatctca caaacaccca 
5521 tttcctctaa tttgcttgat ttttacctgct atctatgtga tacatgta cgcacaaccga 
5581 gtaaatcttt cccatatcttt cttgcaatag gcgtATGtgcgg gcgcTACgca aaaaagata 
L1 cds -> <- L2 end 
5641 tacctgcctc ctaaaccagtt ttaagttggt gcctgaacag ggctATGtggc ggccTAGcga aaacaaagta 
5701 gagctatattc atacgtgcag cagctctgta cattgttatt ctgttctggc ctgttctggc 
5761 gtaaagaaaa ccataaatata gcaagatttt cctaaagatag cagatgtcct atatagcggta 
5821 tttccttctgac gcctcttcct ttaaaatctta cgtcaattgg atgtaaatgtg acctcagcat 
5881 cctcaagctt gctcaattgc ctatcctctct ttaaaatctta cgtcaattgg atgtaaatgtg 
5941 cctcaagctt gctcaattgc ctatcctctct ttaaaatctta cgtcaattgg atgtaaatgtg 
6001 ccttaaatagt ttggtgaacag gcgttaacat agggaaacta tgtcgtctggg tataatagata 
6061 ccaacaaaaag ctgcaacaggt cctgcaacaggt cctgcaacaggt cctgcaacaggt 
6121 ctatctctct ctggaacttt tttctggtct tttctggtct tttctggtct tttctggtct 
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6301 tacatattct aaaaaatcctta cgtcaattgg atgtaaatgtg acctcagcat 
6361 ccaagttttg cctccttcct cgggtgcagaat gcttaaatata cgtcaattgg atgtaaatgtg acctcagcat 
6421 gcctcaactc ttaaaatgta cttgggtcagc cctgcaacaggt cctgcaacaggt cctgcaacaggt 
6481 gtaaatcttt tttctggtct tttctggtct tttctggtct tttctggtct tttctggtct 
6541 ggcctctactg agttcacaacc cccatatgct cccacgctcc tataatctca caaacaccca 
6601 gtaaatcttt cccatatcttt cttgcaatag gcgtATGtgcgg gcgcTACgca aaaaagata 
6661 ccaacaaaaag ccataaatata gcaagatttt cctaaagatag cagatgtcct atatagcggta 
6721 cagatgtcct atataatctta cgtcaattgg atgtaaatgtg acctcagcat 
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6841 ccaacatctaa aatggtagct cccctctttcg ttaagttggt gcctgaacag ggctATGtggc ggccTAGcga aaacaaagta 
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6961 ataatatttt tttctggtct tttctggtct tttctggtct tttctggtct tttctggtct 
7021 gcacgtcgcg ggcctcttcct cgggtgcagaat gcttaaatata cgtcaattgg atgtaaatgtg acctcagcat 
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7261 ataatatttt tttctggtct tttctggtct tttctggtct tttctggtct tttctggtct 
7321 cttagctttc ccccttttgc cgggtggcct ttaagttggt gcctgaacag ggctATGtggc ggccTAGcga aaacaaagta 
7381 acacaaACCG TTCCTGGTgc tttttcttgc ccccttttgc cgggtggcct ttaagttggt gcctgaacag ggctATGtggc ggccTAGcga aaacaaagta 
E2 binding -> 
7441 acatataatgg tttccttctgac gcctcttcct ttaaaatctta cgtcaattgg atgtaaatgtgacctcagcat 
7501 cccctctttcg ttaagttggt gcctgaacag ggctATGtggc ggccTAGcga aaacaaagta 
7561 tttcctctttcg ttaagttggt gcctgaacag ggctATGtggc ggccTAGcga aaacaaagta 
7621 tttccttttcct ttaagttggt gcctgaacag ggctATGtggc ggccTAGcga aaacaaagta 
7681 gtaaatcttt tttctggtct tttctggtct tttctggtct tttctggtct tttctggtct 
E2 binding -> 
7741 cttttttttat cattattat //
LOCUS   CgPV1E1   202 bp ds-DNA   VRL   23-APR-1991
DEFINITION Colobus monkey papilloma virus (CgPV-1) gene homologous to HPV-16 E1 orf, partial cds.
ACCESSION M64365
SEGMENT 1 of 2
SOURCE Colobus monkey papilloma virus (CgPV-1) DNA.
REFERENCE 1 (bases 1 to 202)
AUTHORS Reszka,A.A., Sundberg,J.P. and Reichmann,M.E.
TITLE In vitro transformation and molecular characterization of colobus monkey venereal papillomavirus DNA
JOURNAL Virology 181, 787-792 (1991)
COMMENT The CgPV1 genome was isolated from a penile biopsy of a Colobus monkey; total length was approximately 7.8 kb. The isolated DNA was inserted into the pUC18 plasmid. Genomes obtained by E. coli replication of the plasmid were analysed by cross-hybridization with other PV genomes, restriction digestions, partial sequencing, and transformation assays. The greatest degree of cross-hybridization was obtained to HPVs rather than to other animal PVs, with significant hybridization under stringent conditions to HPV2a, HPV3, HPV16 and HPV18. The arrangement of the genome is similar to that of other PVs, as determined by hybridization to HPV16. Comparison of DNA sequence fragments from the E1 and L1 ORFs again showed CgPV1 to be more similar to genital HPV types than to animal PV types. The greatest similarity appears to be to Groups A2 and A4. Transformation assays indicate that CgPV1 is capable of stably transforming NIH 3T3 cells, but not C127 nor Vero cells; with respect to the 3T3 cells, transformation potential is comparable for CgPV1 and BPV1. Restriction digestion fragment sizes suggest that the genome of the CgPV1 isolate is integrated into the host’s chromosomal DNA and that there has been a deletion similar to deletions found in HPV16 and HPV18 from cervical cancers. The transforming capabilities, genetic similarity of CgPV to genital HPVs, and apparent integrated and deleted state of the genome suggest that CgPV may provide a useful model for investigations of HPVs. An additional, distinct PV (CgPV-2) has been isolated from a cutaneous site on another Colobus monkey (Kloster et al. Virology 166(1):30-40).

NCBI gi: 332158
BASE COUNT 67 a 39 c 53 g 43 t
ORIGIN
1  gatggttcag  tggcctacga  ccacgatatc  acagagagaa  tttggccta  tgaatatgcc
E1 cds ->
61  agattagcgg  atgtggatag  caatgcagca  gcatttttaa  acagcaactg  ccagGCCAAg
    NF1 bind ->
121  tatgtaaacg  atgcagctac  aatgtgcaga  cattataaagc  ggcagaggc  agcccgagatg
181  acaatgtcac  aatggtattg  ccagagcctt  caggttgctgg  gttgggctta  tgaatatgccc
     E1 cds ->

//
Colobus monkey papilloma virus (CgPV-1) gene homologous to HPV-16 L1 orf, partial cds.

Colobus monkey papilloma virus (CgPV-1) DNA.

Colobus monkey venereal papillomavirus DNA

The CgPV1 genome was isolated from a penile biopsy of a Colobus monkey, total length was approximately 7.8 kb. The isolated DNA was inserted into the pUC18 plasmid. Genomes obtained by E. coli replication of the plasmid were analysed by cross-hybridization with other PV genomes, restriction digestions, partial sequencing, and transformation assays. The greatest degree of cross-hybridization was obtained to HPVs rather than to other animal PVs, with significant hybridization under stringent conditions to HPV2a, HPV3, HPV16 and HPV18. The arrangement of the genome is similar to that of other PVs, as determined by hybridization to HPV16. Comparison of DNA sequence fragments from the E1 and L1 ORFs again showed CgPV1 to be more similar to genital HPV types than to animal PV types. The greatest similarity appears to be to Groups A2 and A4. Transformation assays indicate that CgPV1 is capable of stably transforming NIH 3T3 cells, but not C127 nor Vero cells; with respect to the 3T3 cells, transformation potential is comparable for CgPV1 and BPV1. Restriction digestion fragment sizes suggest that the genome of the CgPV1 isolate is integrated into the host’s chromosomal DNA and that there has been a deletion similar to deletions found in HPV16 and HPV18 from cervical cancers. The transforming capabilities, genetic similarity of CgPV to genital HPVs, and apparent integrated and deleted state of the genome suggest that CgPV may provide a useful model for investigations of HPVs. An additional, distinct PV (CgPV-2) has been isolated from a cutaneous site on another Colobus monkey (Kloster et al. Virology 166(1):30-40).