WELCOME
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AETC AIDS Education & Training Center Program Northeast/Caribbean

NEW YORK STATE Department of Health AIDS Institute

Disclosures

“This program is supported by the Health Resources and Services Administration (HRSA) of the U.S. Department of Health and Human Services (HHS) as part of an award totaling $3,879,101 with 0 percentage financed with nongovernmental sources. The contents are those of the author(s) and do not necessarily represent the official views of, nor an endorsement, by HRSA, HHS or the U.S. Government.”

AETC AIDS Education & Training Center Program Northeast/Caribbean

Reminders

Complete and turn in at end of presentation:

- **Sign in sheet**
- **Participant Information Form if not already completed on-line**
- **CE application form**

Evaluation form and CE form will be emailed to you following presentation

Oral Health Preceptorships

Audience: Dentist, Dental Hygienists and other primary care clinicians

Venue: Multiple sites in NY & NJ

Duration: 1 day to multiple days

CDE/CEU: Provided

Cost: None

Enroll: Send email to: howard.lavigne2@gmail.com

Addressing the needs of the New York, New Jersey, Puerto Rico & US Virgin Islands oral healthcare community

For more information contact:

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Central Office
Columbia University
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646-774-6978

AETC AIDS Education & Training Center Program Northeast/Caribbean

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AETC AIDS Education & Training Center Program Northeast/Caribbean

NEW YORK STATE Department of Health AIDS Institute

Oral and Oropharyngeal HPV Infection in HIV+ Patients

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NYU | DENTISTRY

Disclosure

I have no real or perceived vested interests that relate to this presentation nor do I have any relationships with pharmaceutical companies, biomedical device manufacturers, and/or other corporations whose products or services are related to pertinent therapeutic areas.

Human Papillomavirus (HPV)

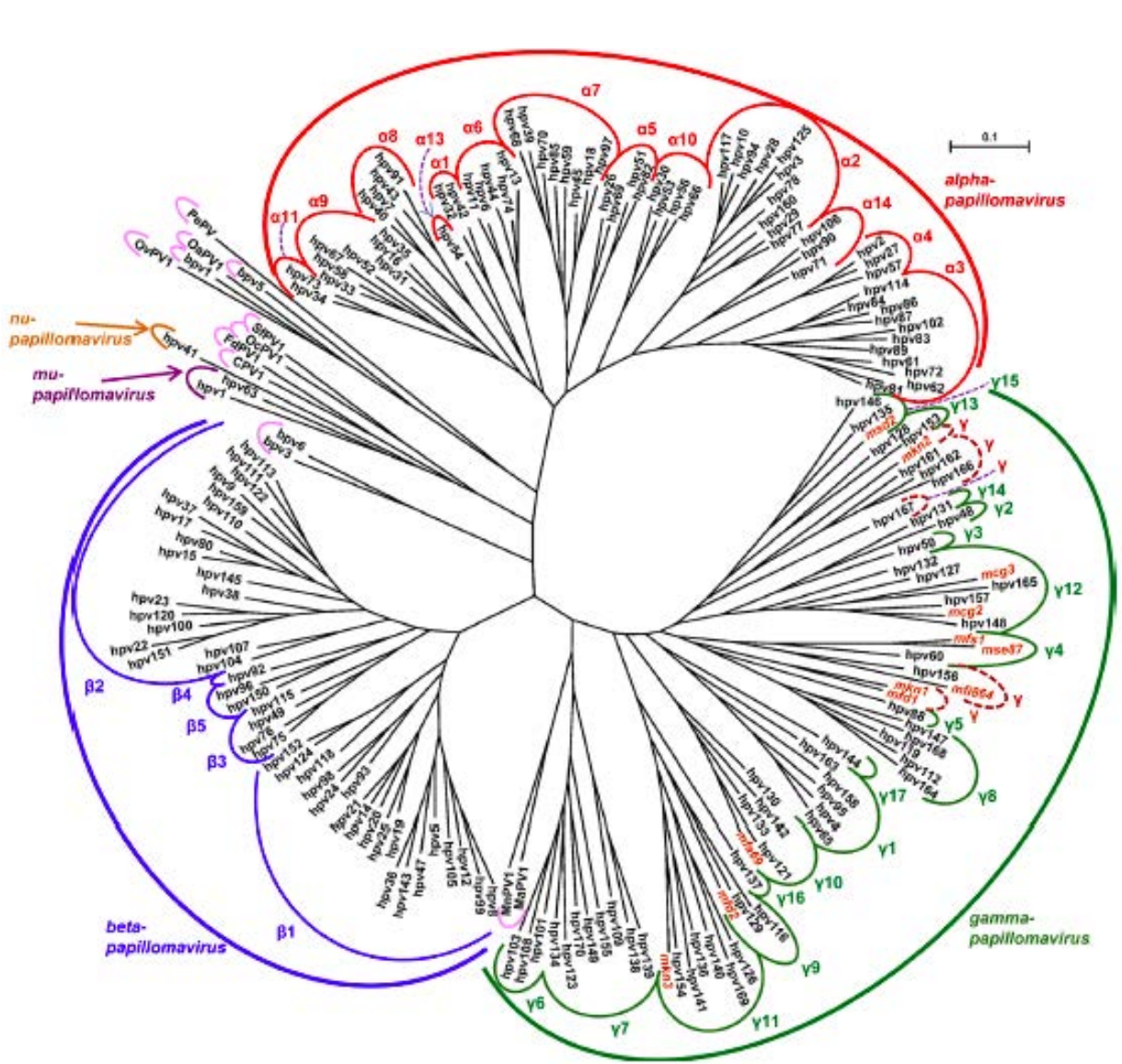
Small, circular DNA viruses

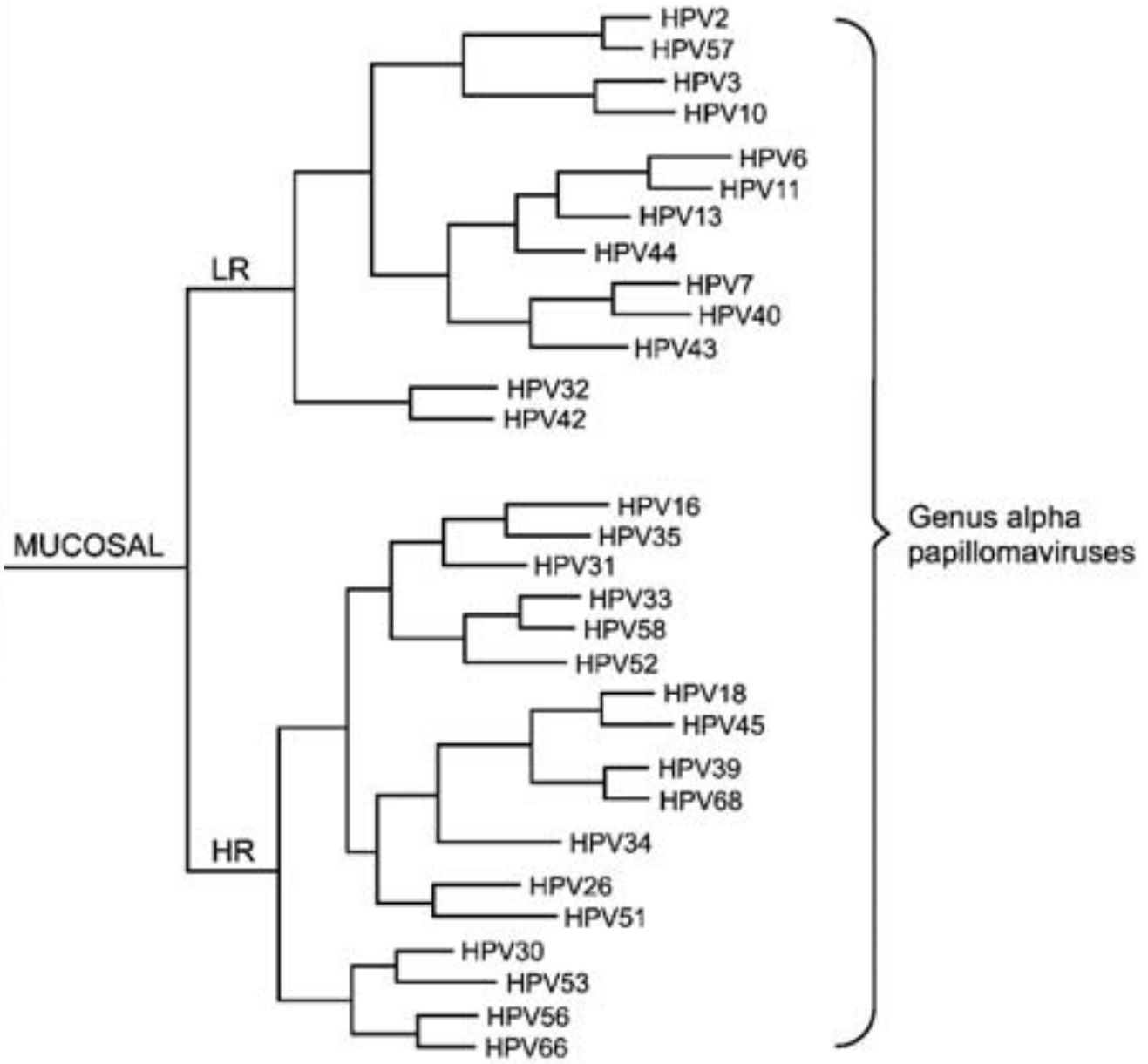
Humans only known host

Over 170 unique types

Cutaneous and mucosal types

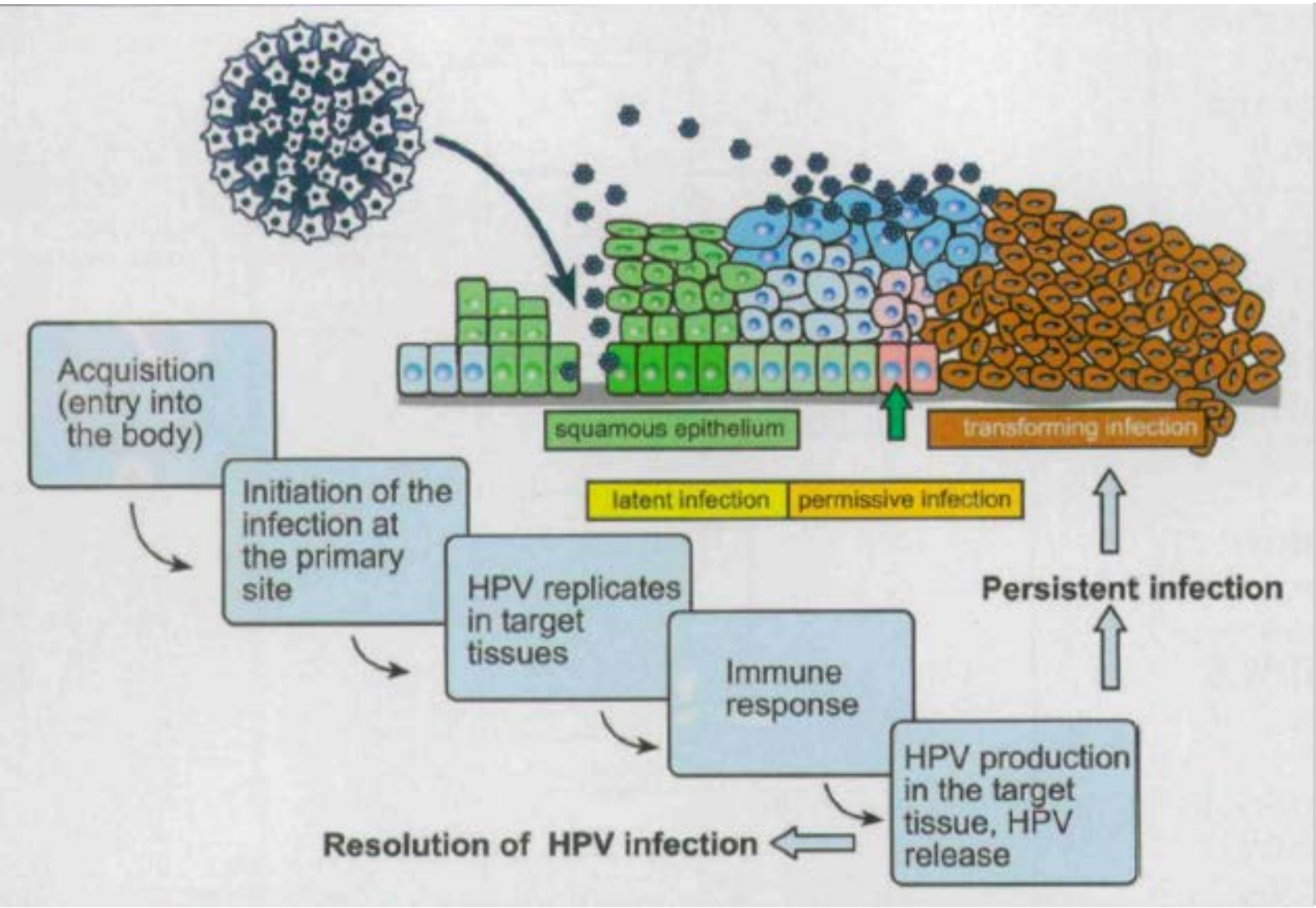
“High” and “low risk” types

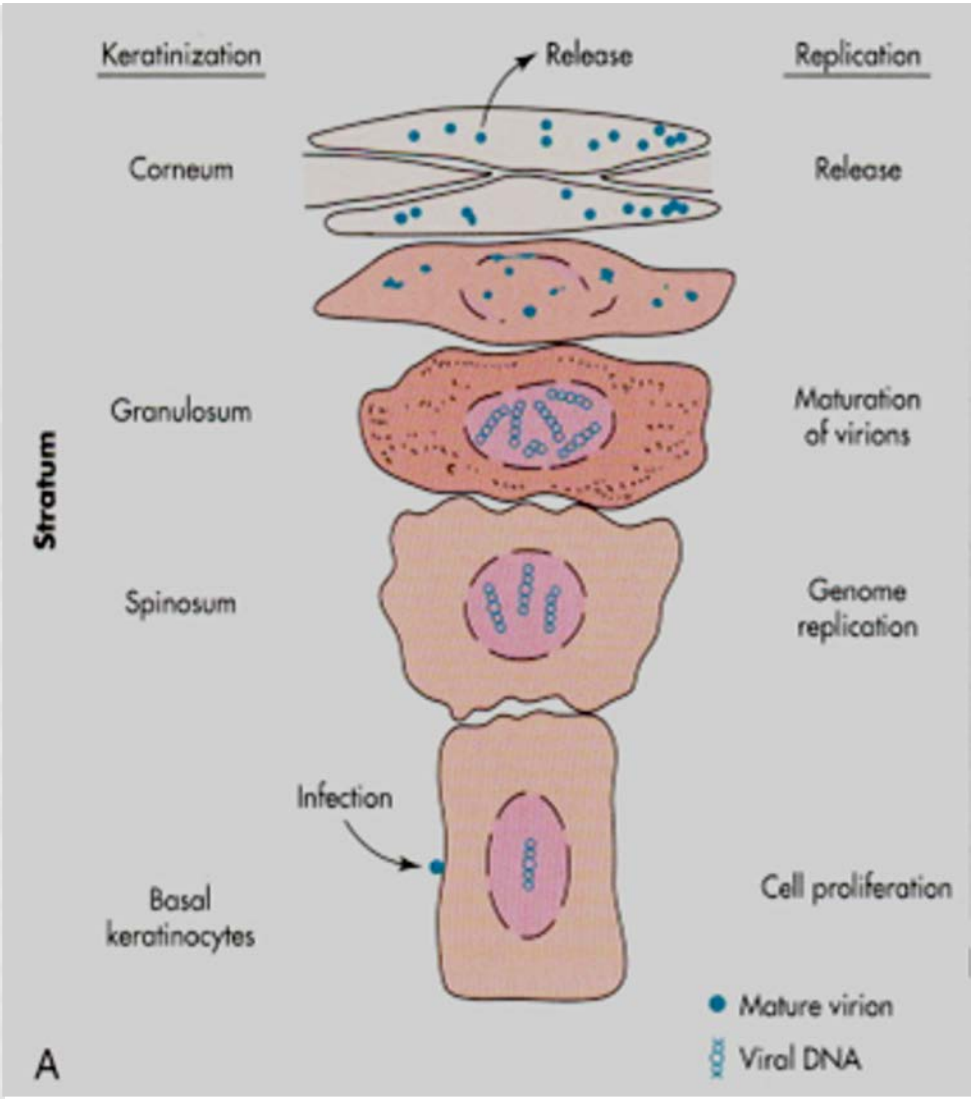




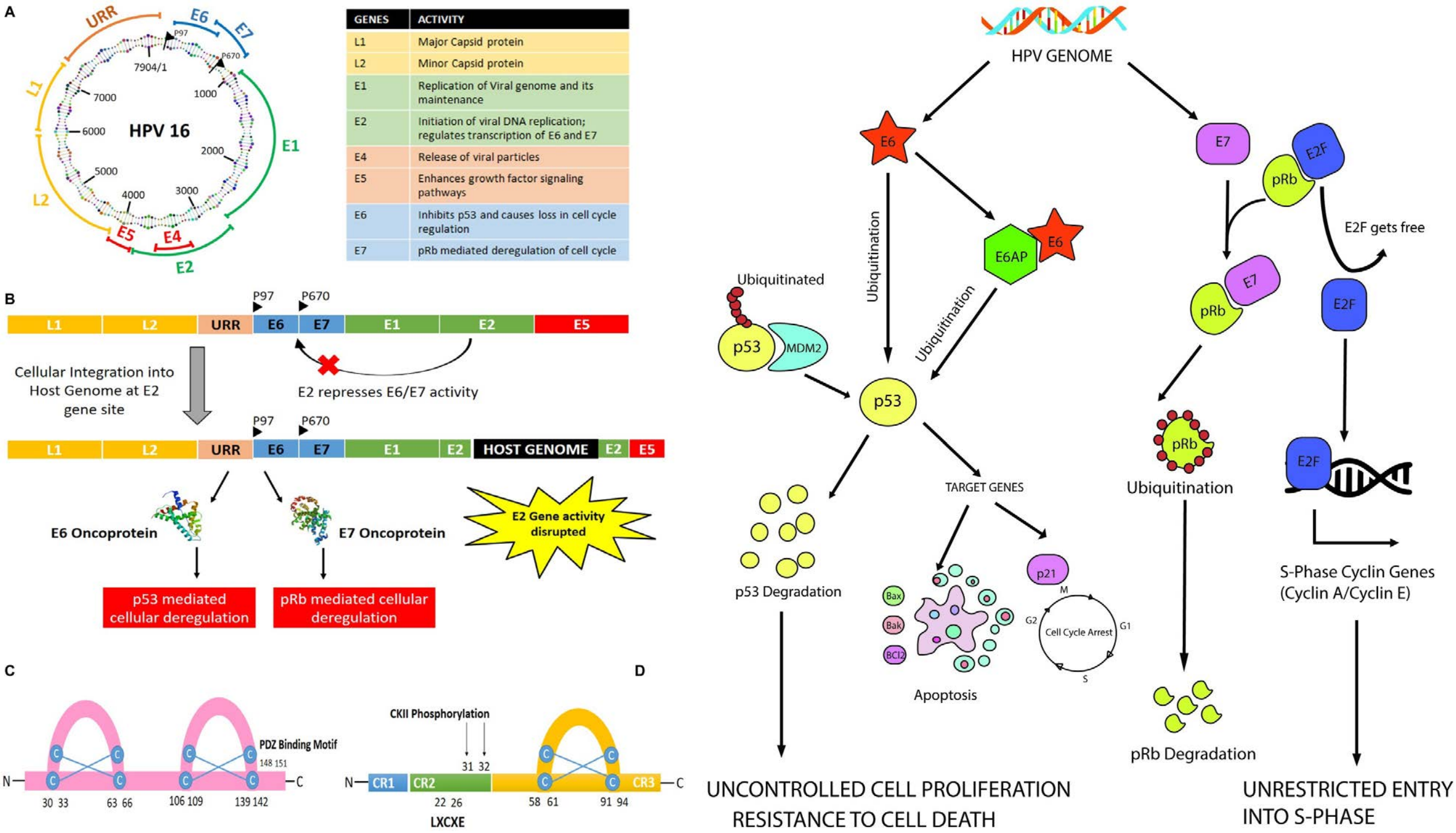
E.-M. deVilliers et al Virology 2013; 445: 2–10
Rautava J. JADA 2011;142(8):905-914

HPV Pathogenesis





How does HIV infection modulate this process?


A
URR
E6
E7
E1
E2
E4
E5
L2
L1
P97
P670
7904/1
1000
2000
3000
4000
5000
6000
7000
HPV 16
GENES
ACTIVITY
L1
Major Capsid protein
L2
Minor Capsid protein
E1
Replication of Viral genome and its maintenance
E2
Initiation of viral DNA replication; regulates transcription of E6 and E7
E4
Release of viral particles
E5
Enhances growth factor signaling pathways
E6
Inhibits p53 and causes loss in cell cycle regulation
E7
pRb mediated deregulation of cell cycle
B
P97
P670
L1
L2
URR
E6
E7
E1
E2
E5
Cellular Integration into Host Genome at E2 gene site
E2 represses E6/E7 activity
HOST GENOME
E6 Oncoprotein
E7 Oncoprotein
E2 Gene activity disrupted
p53 mediated cellular deregulation
pRb mediated cellular deregulation
C
PDZ Binding Motif
148 151
N
C
30 33
63 66
106 109
139 142
D
CKII Phosphorylation
31 32
CR1
CR2
CR3
22 26
58 61
91 94
LXCXE
HPV GENOME
E6
E6AP
Ubiquitination
Ubiquitinated
p53
MDM2
p53 Degradation
TARGET GENES
Bax
Bak
BCl2
Apoptosis
p21
M
G2
G1
S
Cell Cycle Arrest
E7
pRb
E2F
E2F gets free
Ubiquitination
pRb Degradation
S-Phase Cyclin Genes (Cyclin A/Cyclin E)
UNCONTROLLED CELL PROLIFERATION
RESISTANCE TO CELL DEATH
UNRESTRICTED ENTRY INTO S-PHASE

Prevalence of Oral HPV Infections in General Population ("Prevalent Infection")

- Overall: 6.9% (CI 6.7-8.3)*
- Gender: Men (10.1%) > Women (3.6%)
- Age: bimodal distribution
- High risk HPV (3.7%) > Low risk HPV (3.1%)
- HPV-16 infection most prevalent (1% or 2.13 million Americans)

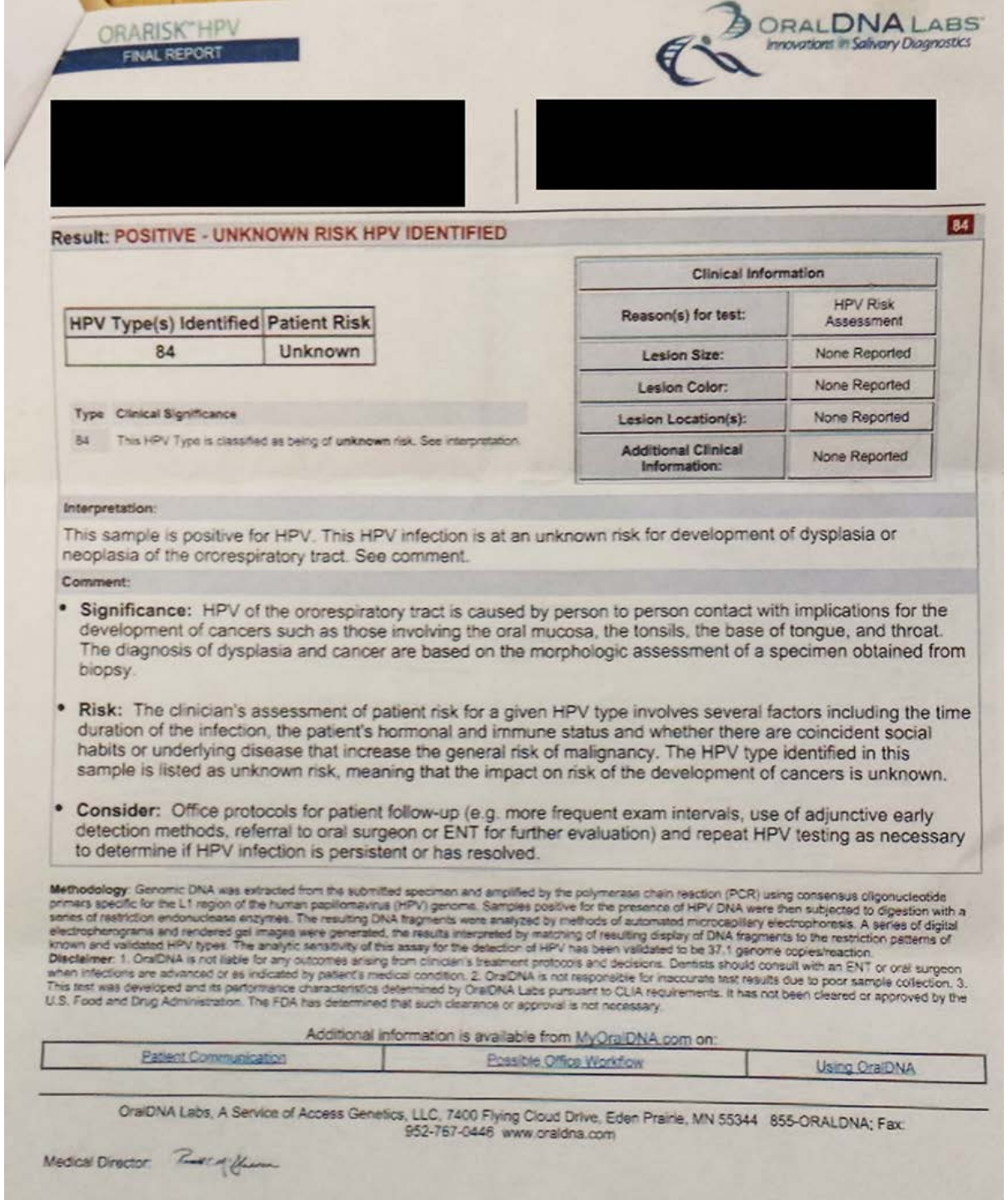
*Based on 5600 NHANES subjects undergoing oral rinse sampling, followed by DNA PCR testing for α HPV

Gillison M. JAMA 2012;307(7):693-703

Commercially available HPV tests:

Approx 200 tests

Most are PCR-based tests, some marketed to dentists



ORARISK™ HPV
FINAL REPORT

ORALDNA LABS
Innovations in Salivary Diagnostics

[redacted]

[redacted]

Result: POSITIVE - UNKNOWN RISK HPV IDENTIFIED 84

HPV Type(s) Identified	Patient Risk
84	Unknown

Type	Clinical Significance
84	This HPV Type is classified as being of unknown risk. See interpretation.

Clinical Information	
Reason(s) for test:	HPV Risk Assessment
Lesion Size:	None Reported
Lesion Color:	None Reported
Lesion Location(s):	None Reported
Additional Clinical Information:	None Reported

Interpretation:

This sample is positive for HPV. This HPV infection is at an unknown risk for development of dysplasia or neoplasia of the ororespiratory tract. See comment.

Comment:

- **Significance:** HPV of the ororespiratory tract is caused by person to person contact with implications for the development of cancers such as those involving the oral mucosa, the tonsils, the base of tongue, and throat. The diagnosis of dysplasia and cancer are based on the morphologic assessment of a specimen obtained from biopsy.
- **Risk:** The clinician's assessment of patient risk for a given HPV type involves several factors including the time duration of the infection, the patient's hormonal and immune status and whether there are coincident social habits or underlying disease that increase the general risk of malignancy. The HPV type identified in this sample is listed as unknown risk, meaning that the impact on risk of the development of cancers is unknown.
- **Consider:** Office protocols for patient follow-up (e.g. more frequent exam intervals, use of adjunctive early detection methods, referral to oral surgeon or ENT for further evaluation) and repeat HPV testing as necessary to determine if HPV infection is persistent or has resolved.

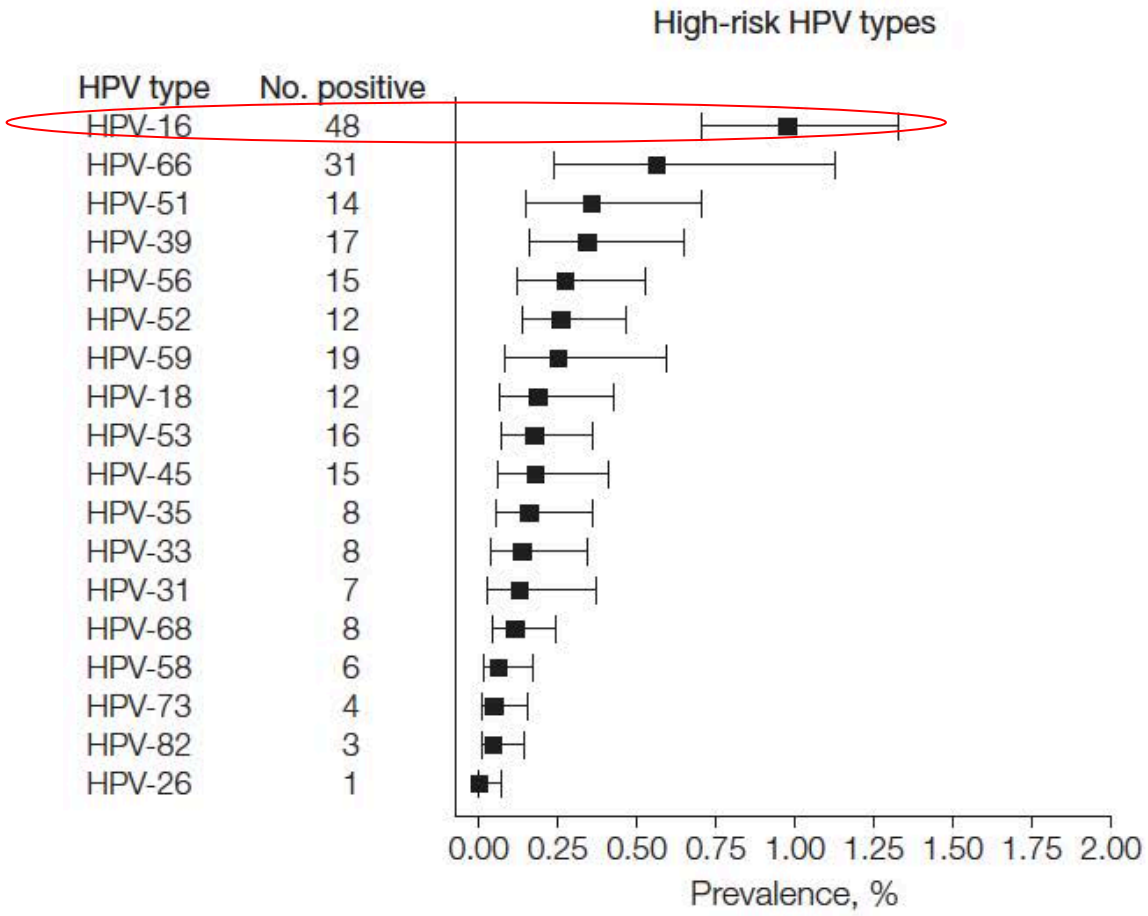
Methodology: Genomic DNA was extracted from the submitted specimen and amplified by the polymerase chain reaction (PCR) using consensus oligonucleotide primers specific for the L1 region of the human papillomavirus (HPV) genome. Samples positive for the presence of HPV DNA were then subjected to digestion with a series of restriction endonuclease enzymes. The resulting DNA fragments were analyzed by methods of automated microcapillary electrophoresis. A series of digital electropherograms and rendered gel images were generated, the results interpreted by matching of resulting display of DNA fragments to the restriction patterns of known and validated HPV types. The analytic sensitivity of this assay for the detection of HPV has been validated to be 37.1 genome copies/reaction.
Disclaimer: 1. OralDNA is not liable for any outcomes arising from clinician's treatment protocols and decisions. Dentists should consult with an ENT or oral surgeon when infections are advanced or as indicated by patient's medical condition. 2. OralDNA is not responsible for inaccurate test results due to poor sample collection. 3. This test was developed and its performance characteristics determined by OralDNA Labs pursuant to CLIA requirements. It has not been cleared or approved by the U.S. Food and Drug Administration. The FDA has determined that such clearance or approval is not necessary.

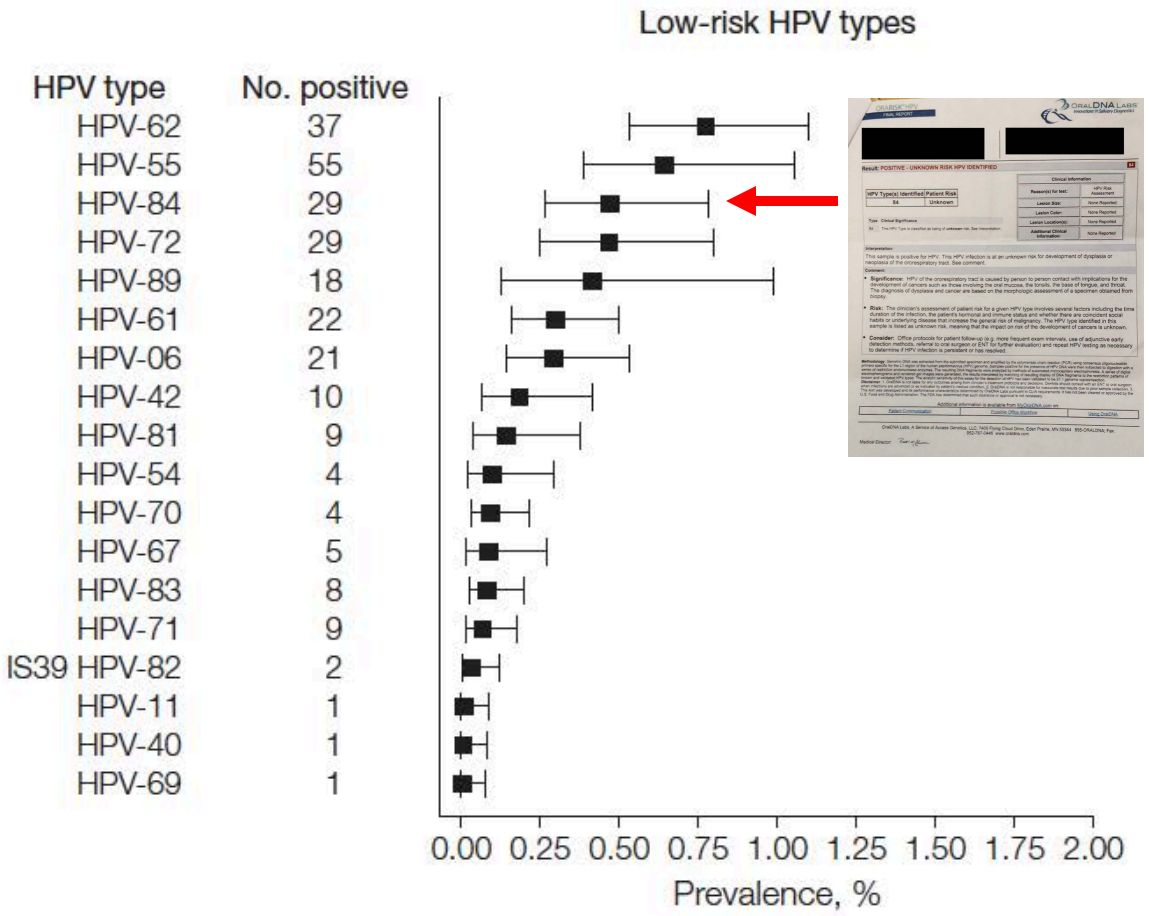
Additional information is available from MyOralDNA.com on:

Patient Communication	Possible Office Workflow	Using OralDNA

OralDNA Labs, A Service of Access Genetics, LLC, 7400 Flying Cloud Drive, Eden Prairie, MN 55344 855-ORALDNA; Fax: 952-767-0446 www.oraldna.com

Medical Director:





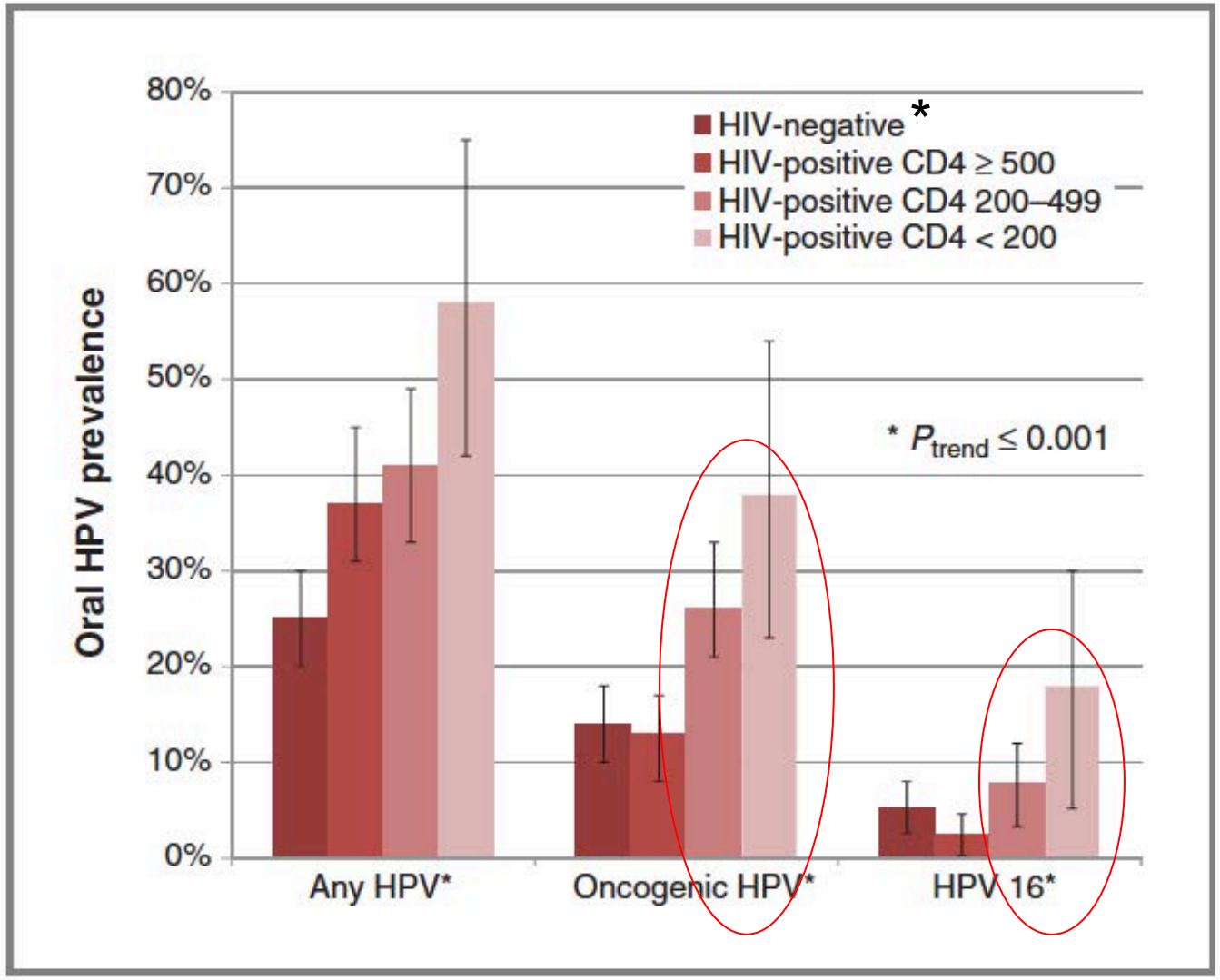
Gillison M. JAMA 2012;307(7):693-703

Risk Factors

- Ever had sex (7.6%) vs never had sex (0.9%)
- Prevalence increases with # of sexual partners and # of recent partners
- If >20 partners, 1 in 5 were infected (21.5%)
- Smokers (>20 cigs/day) had highest prevalence (21.7%)

Gillison M. JAMA 2012;307(7):693-703

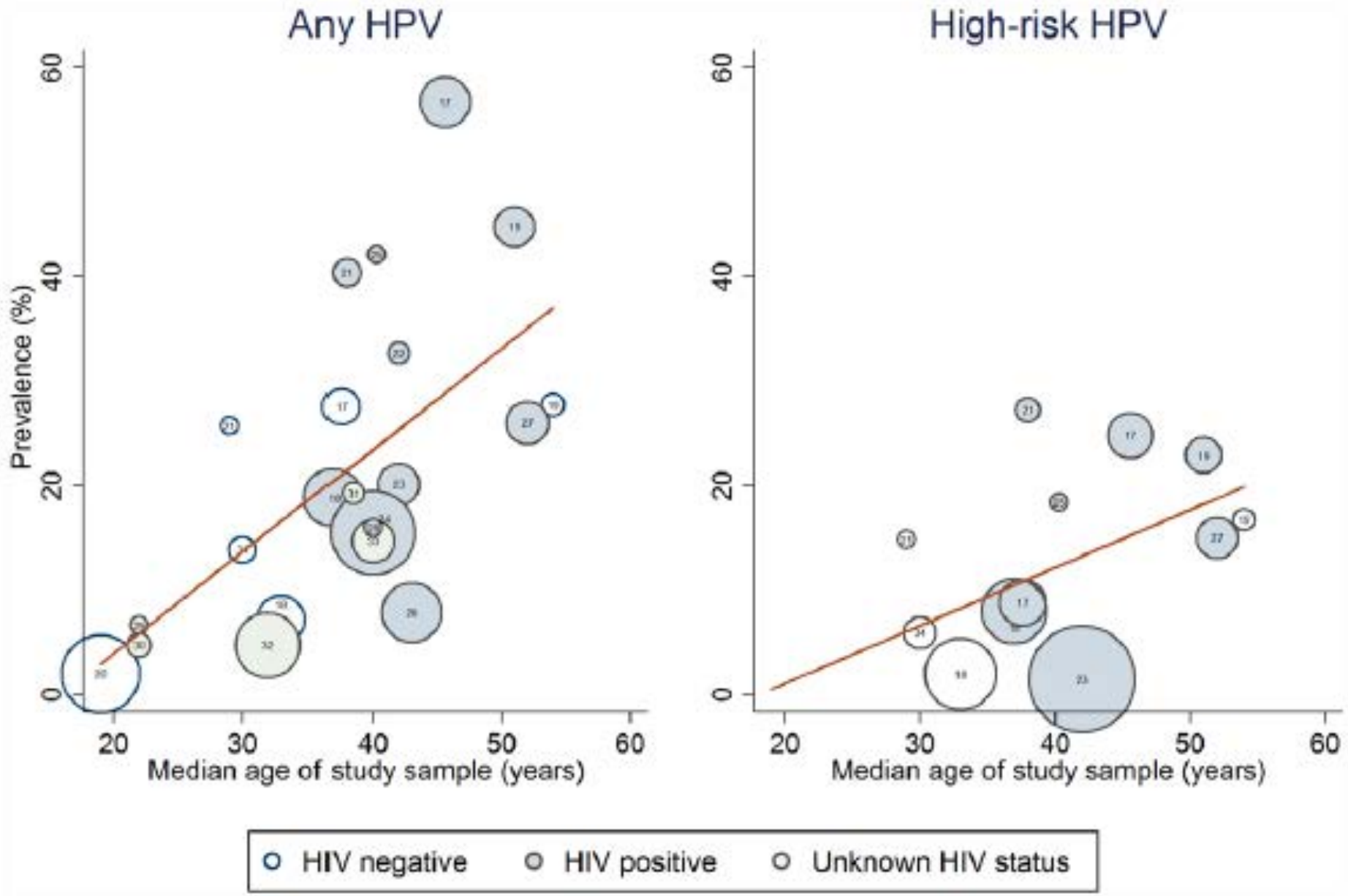
What about prevalent infection in HIV+ populations?



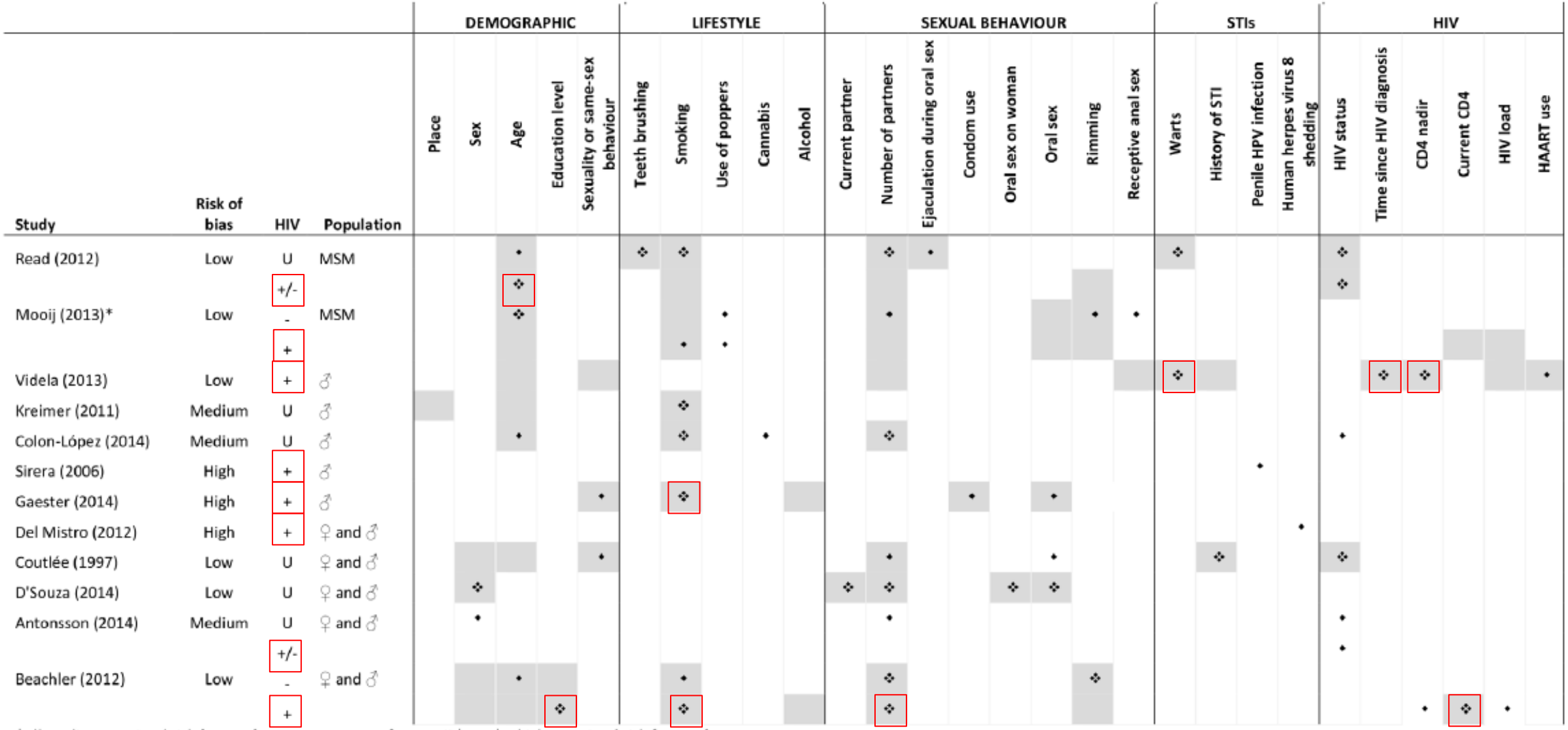
*HIV-negative group were "at risk"

Hi-risk HPV prevalence more affected by immunosuppression

Beachler DC. Cancer Epidemiol Biomarkers Prev 2012;21:122-133



King EM et al. PLOS ONE July 6, 2016



*All studies examined risk factors for any HPV except for Mooij (2013) which examined risk factors for HR-HPV.

Key:

• : Significant (p<0·05) in univariate analyses

❖ : Significant in multivariate analyses

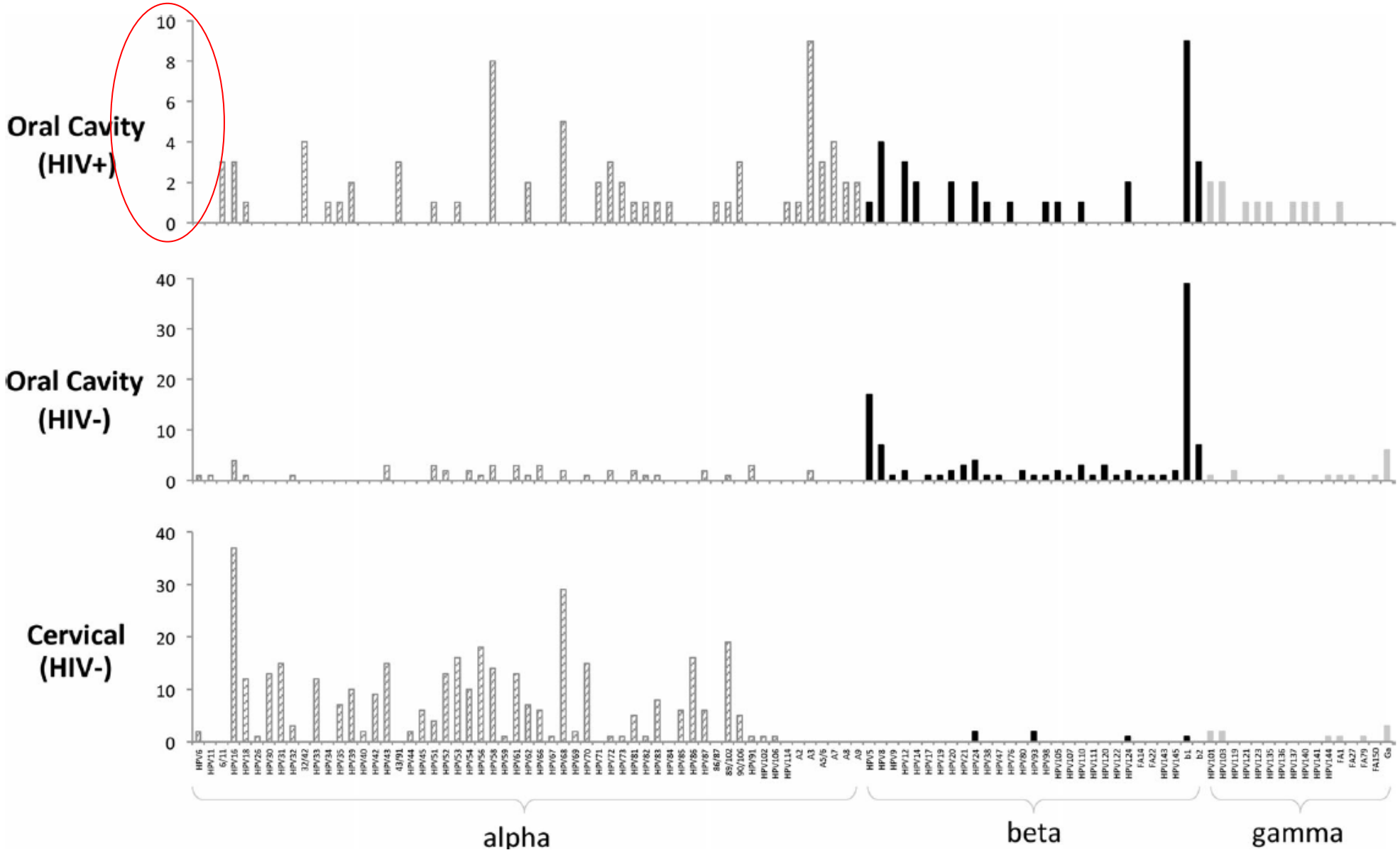
: Covariate in multivariate analyses

U : Unknown HIV status

- : HIV negative

+ : HIV positive

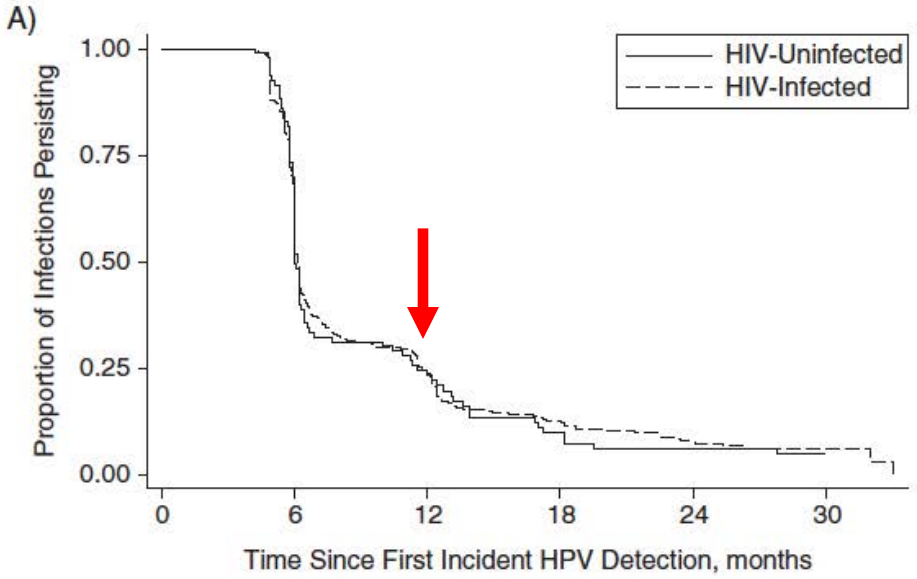
King EM et al. PLOS ONE July 6, 2016

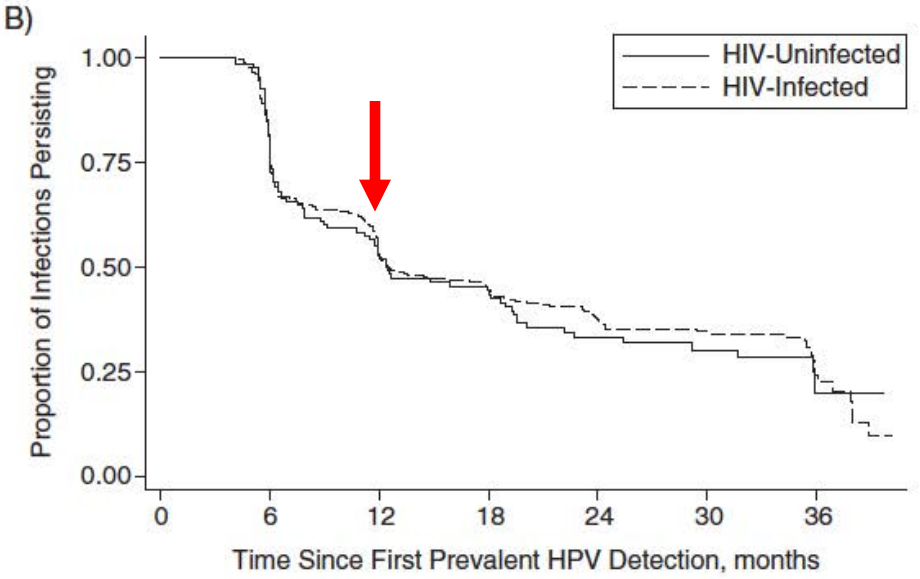


Bottalico D et al. JID 2011;204:787–92
Fatahzadeh M et al. OOOO 2013; 115(4): 505–514

Natural history oral of HPV infection: HIV- vs HIV+

Prevalent vs Incident?
Clearance vs Persistence?
HPV load?

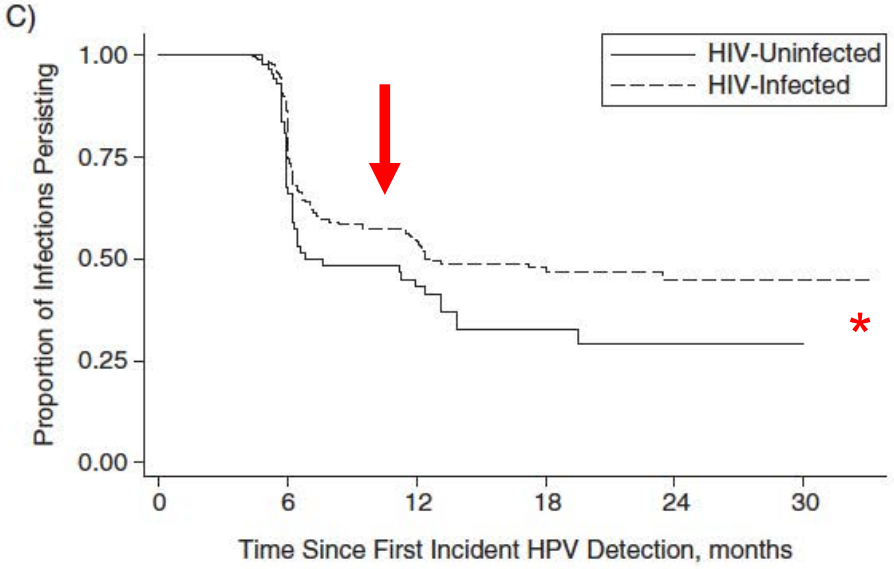


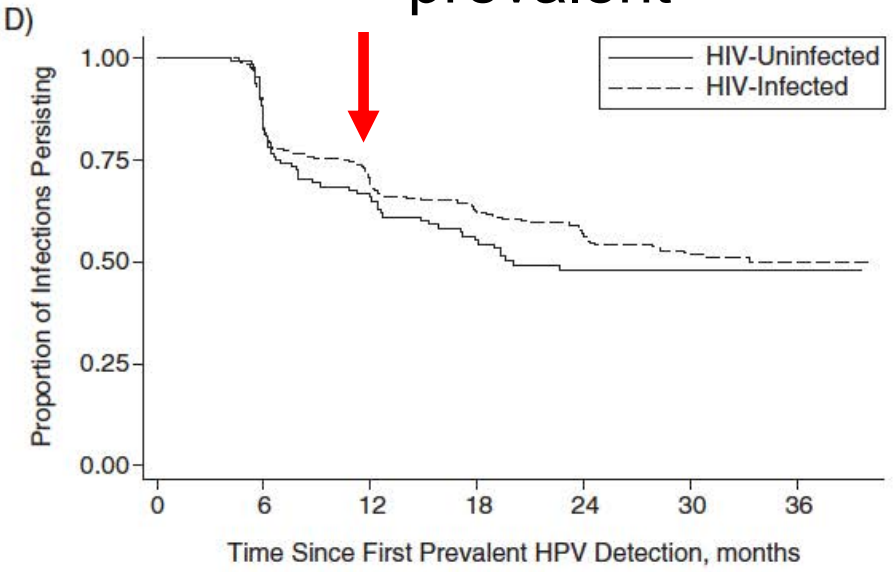


One negative test to show clearance

incident

prevalent





Two negative tests to show clearance

*HIV+ patients cleared incident oral HPV infection significantly slower than HIV- patients

Beachler DC. Am J Epidemiol. 2015;181(1):40–53

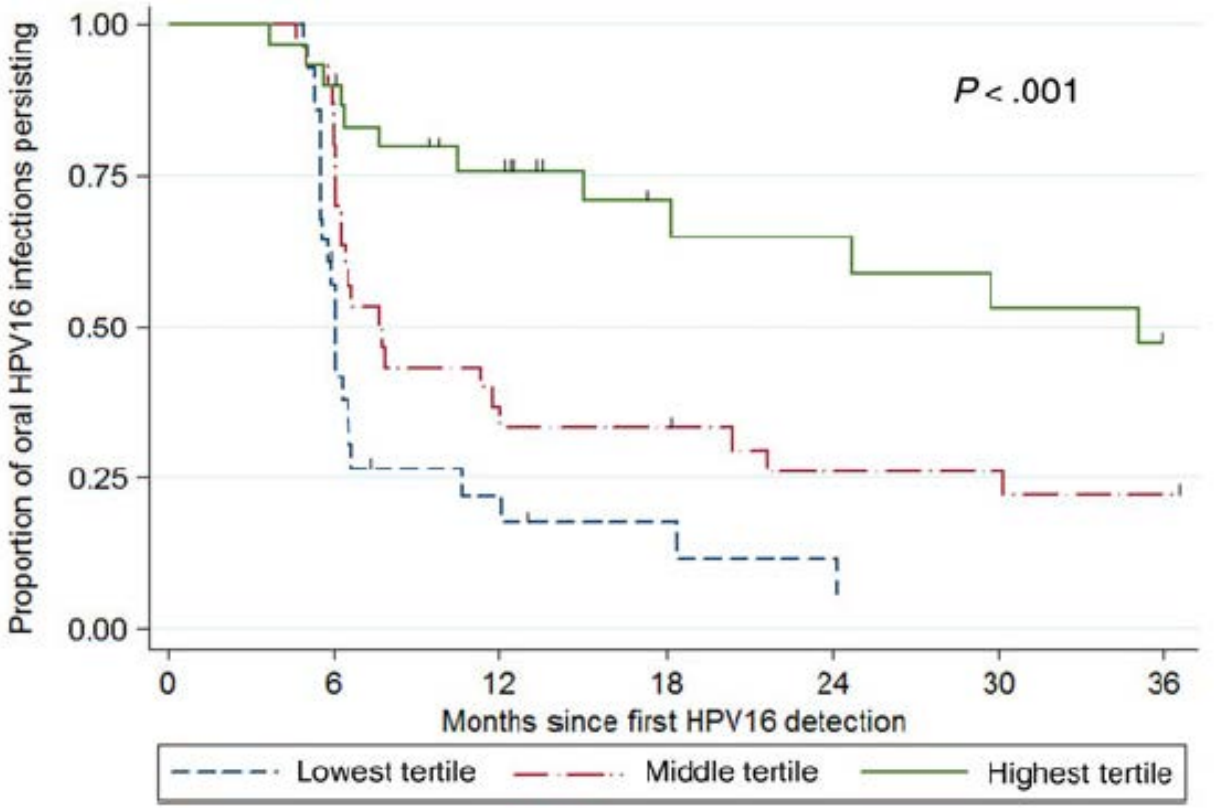
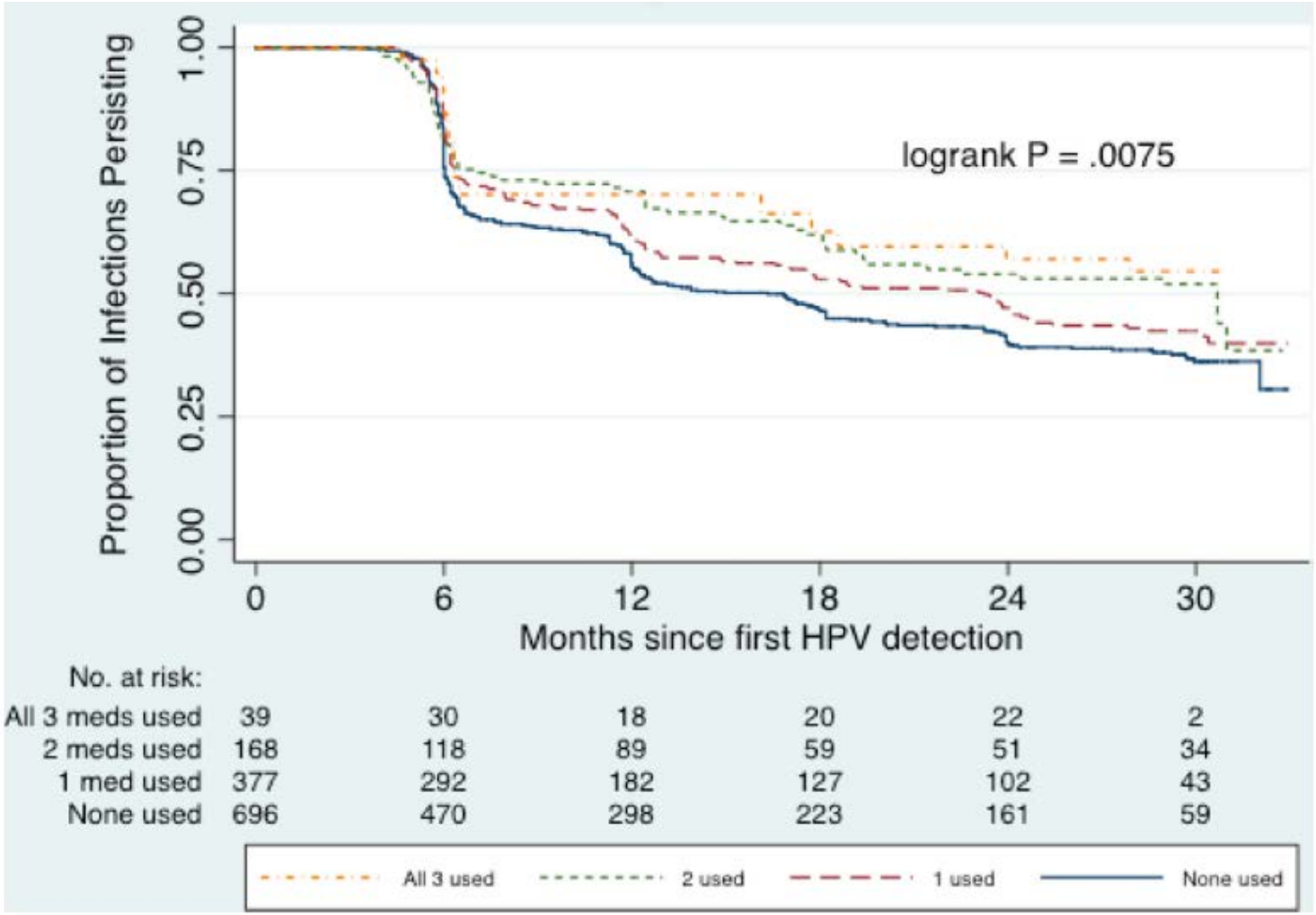


Figure 1. Kaplan–Meier curves for clearance of human papillomavirus virus type 16 (HPV16), by oral HPV16 infection tertile. *Oral HPV clearance defined at first visit when oral HPV16 DNA was not detected. ^Tertile ranges: Lowest tertile 1.0–5.9 copies per 100 000 cells; Middle tertile: 6.0–155.3 copies per 100 000 cells; Highest tertile: >155.3 copies per 100 000 cells. The *P*-trend was calculated using the log-rank test.

HPV-16 load confers a significantly higher risk of persistence

Beachler DC. JID. 2015; 212:1588–91

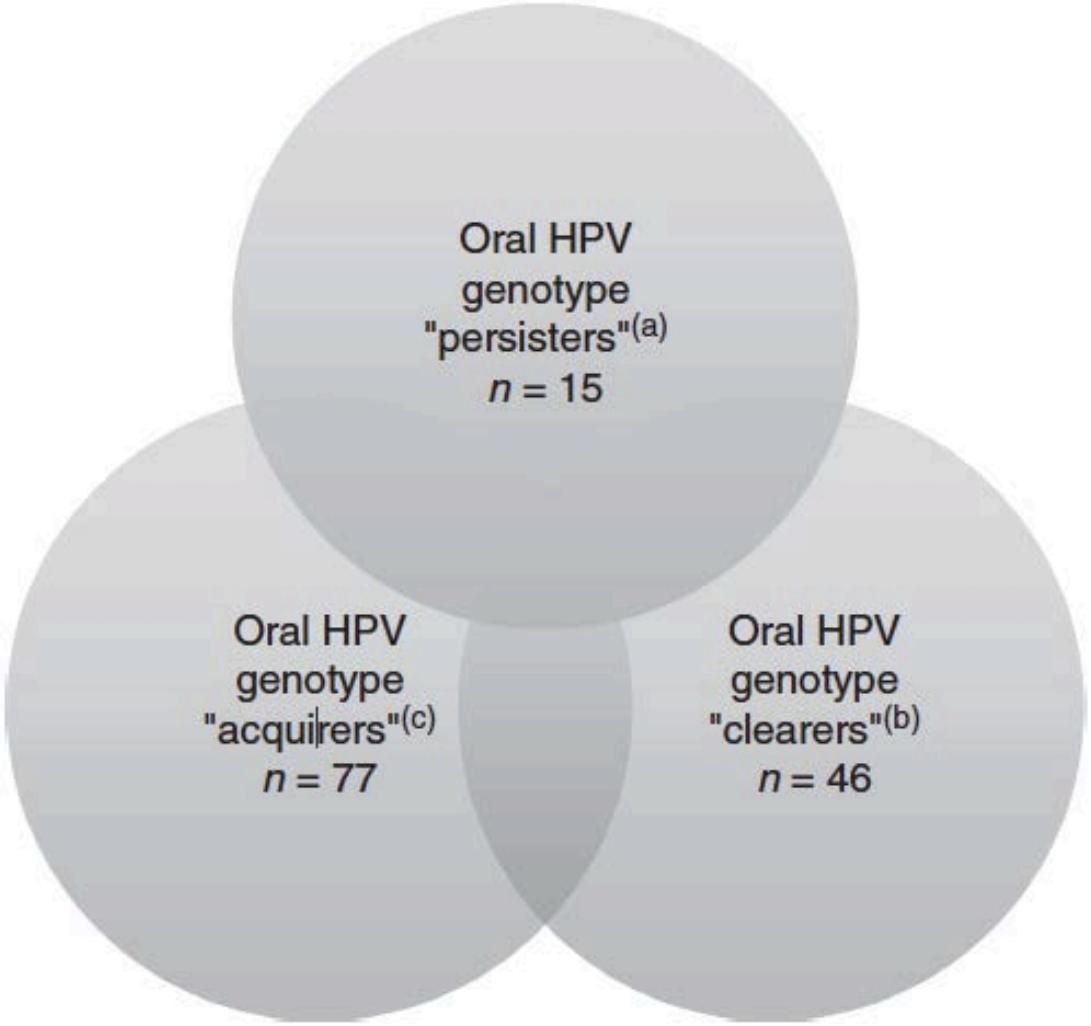
Antipsychotic use as associated with reduced HPV clearance in HIV+ patients (HR .66) but not in HIV- patients



antipsychotics,
antidepressants
and/or
anxiolytics/sedatives

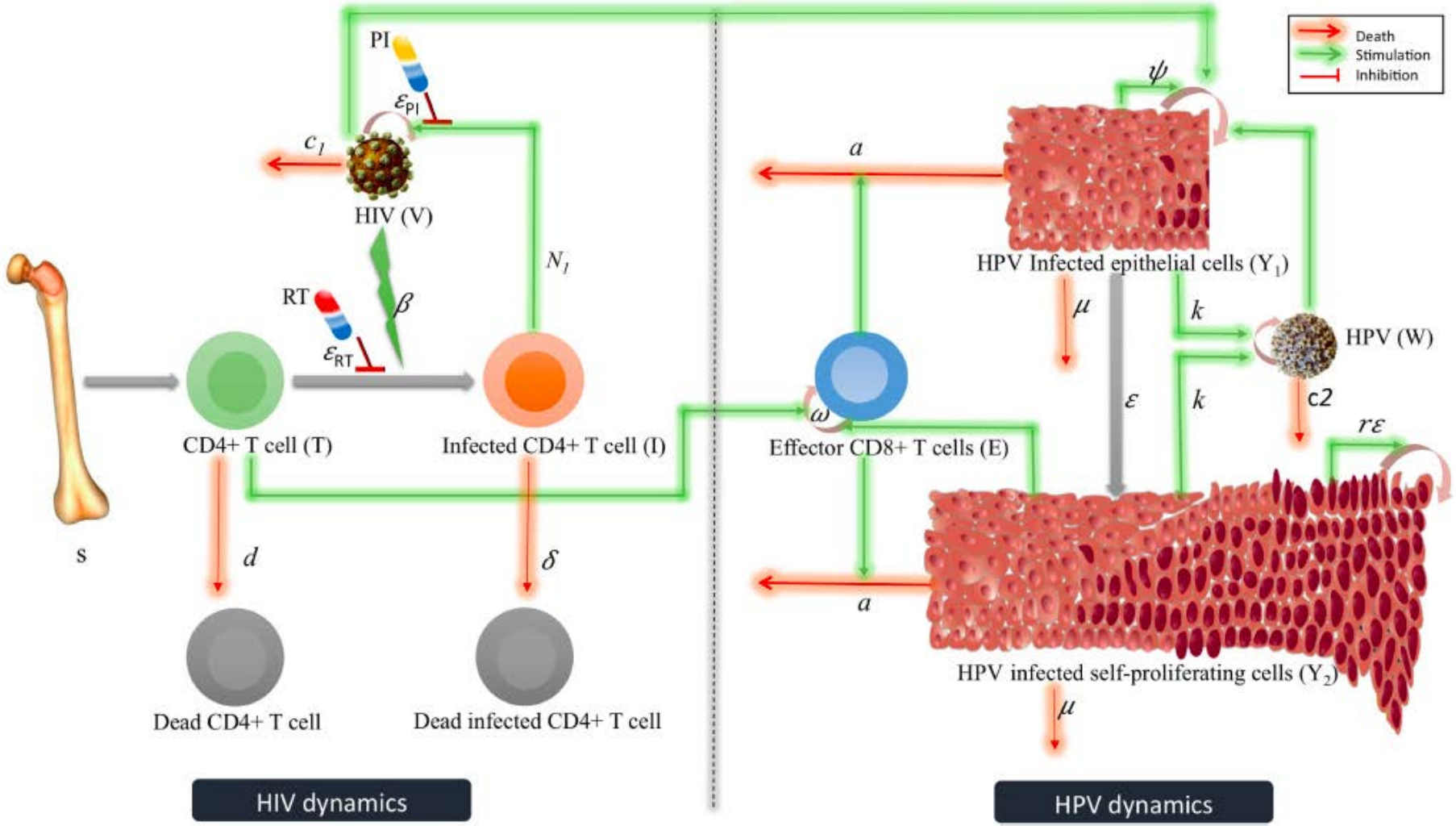
Lam. JO et al. Cancer Causes Control. 2016;27(12):1491–1498

No significant reduction in overall oral HPV DNA prevalence or in the prevalence of oncogenic oral HPV genotypes after 12–24 weeks of ART (n-388)



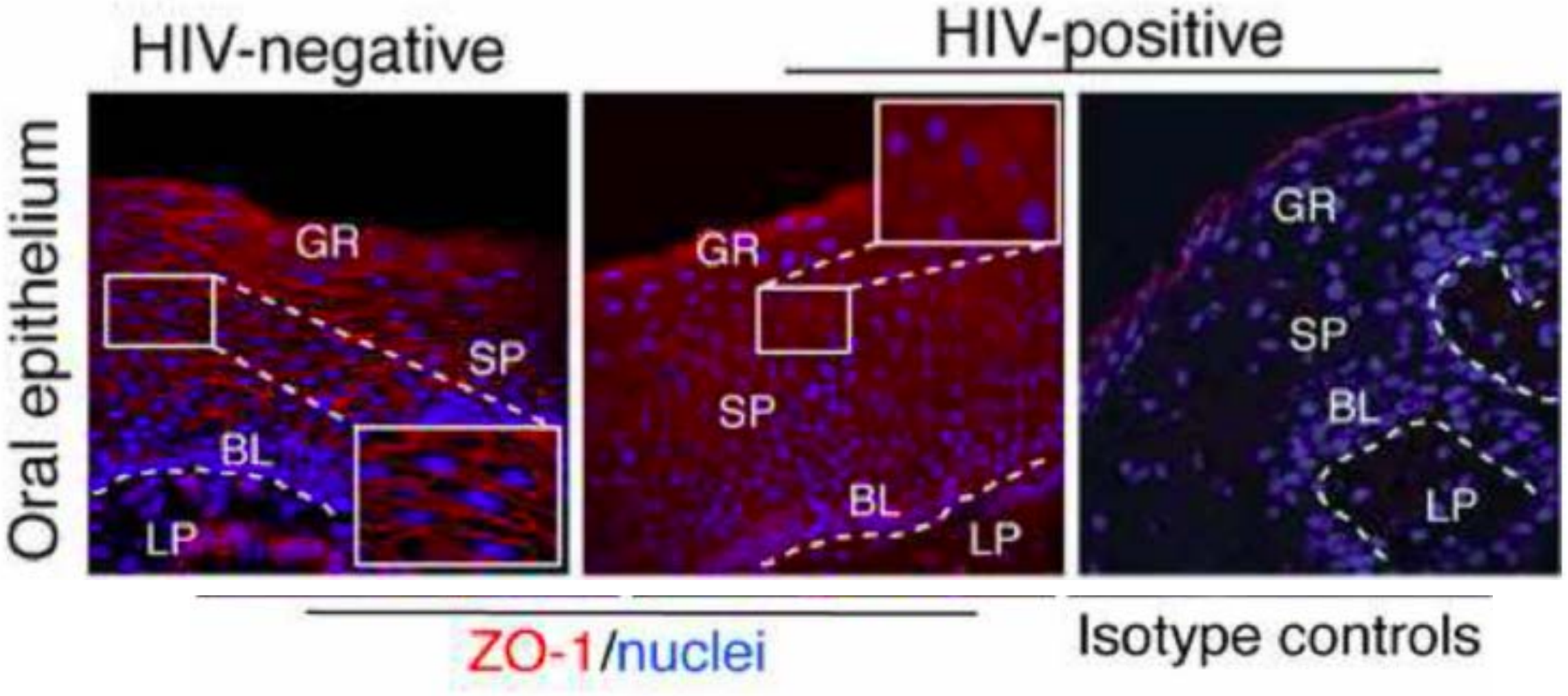
Shiboski C et al. AIDS 2016, 30:1573–1582

How does HIV infection modulate HPV pathogenesis?

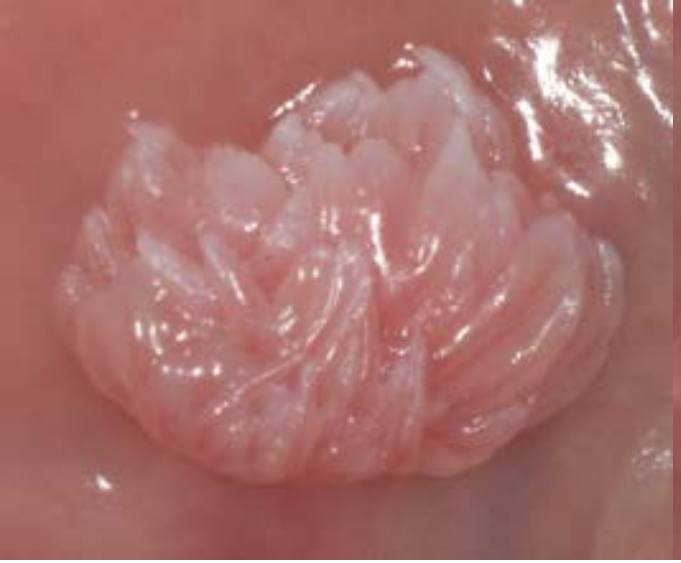
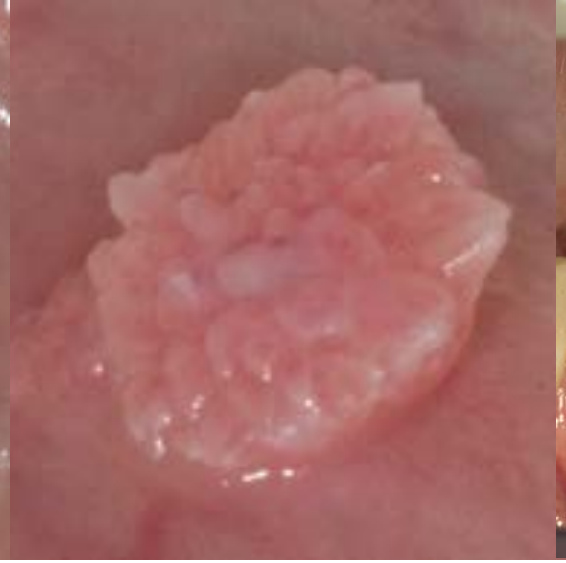
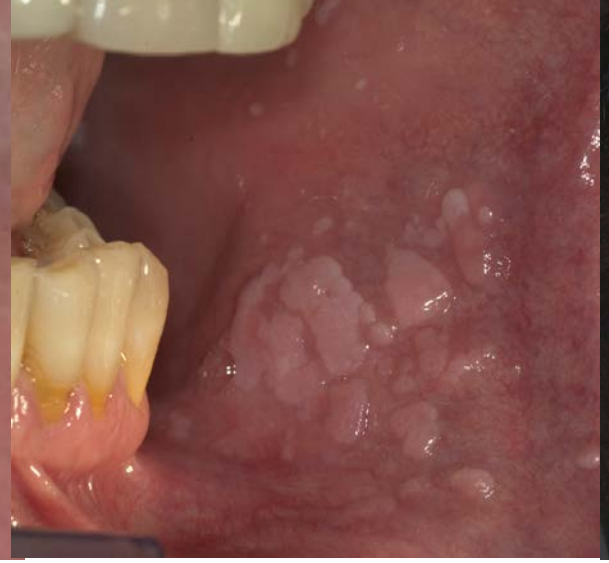
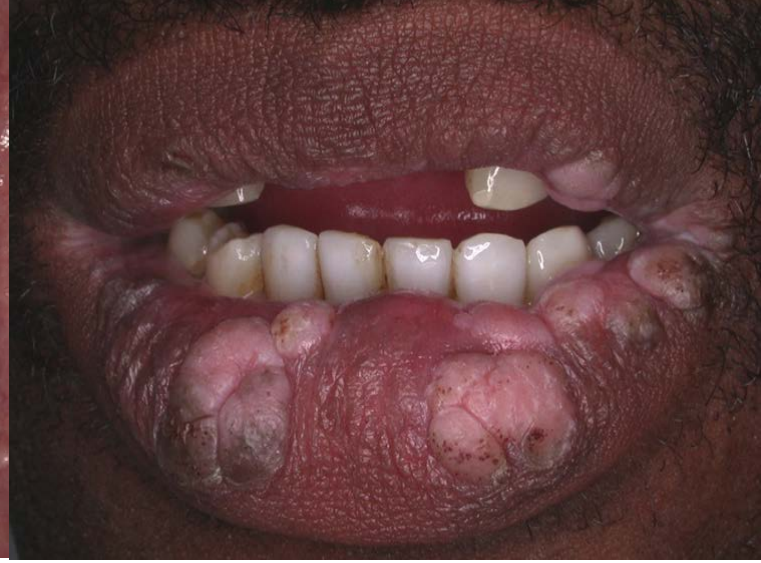


Verma M et al PLOS ONE. January 6, 2017

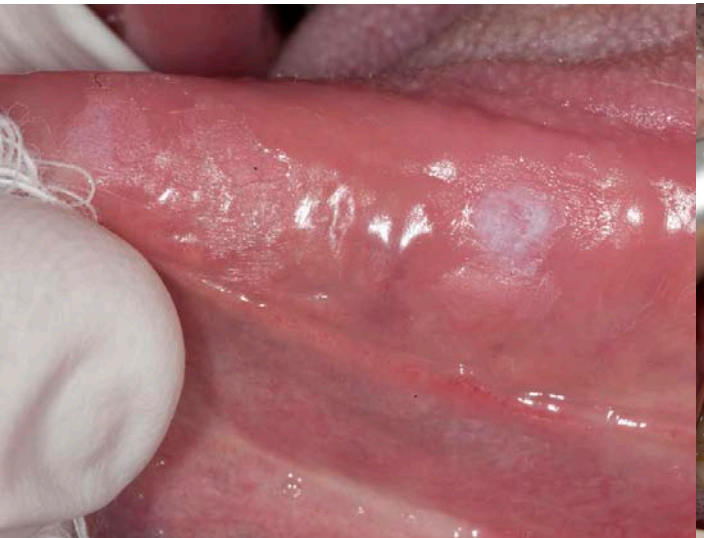
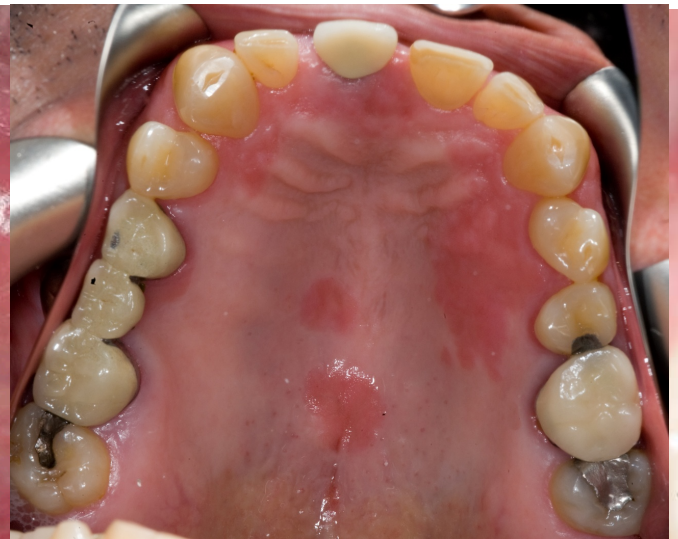
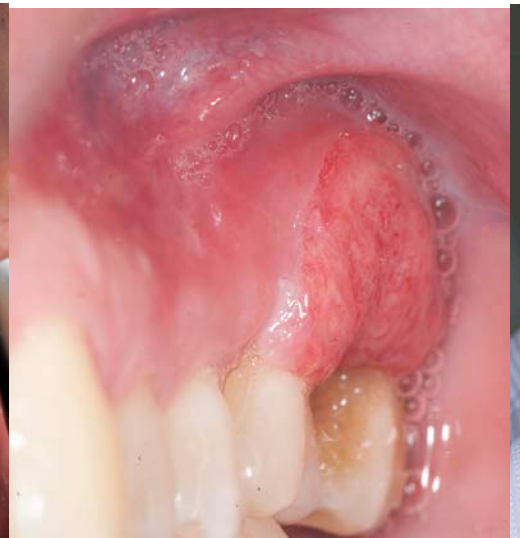
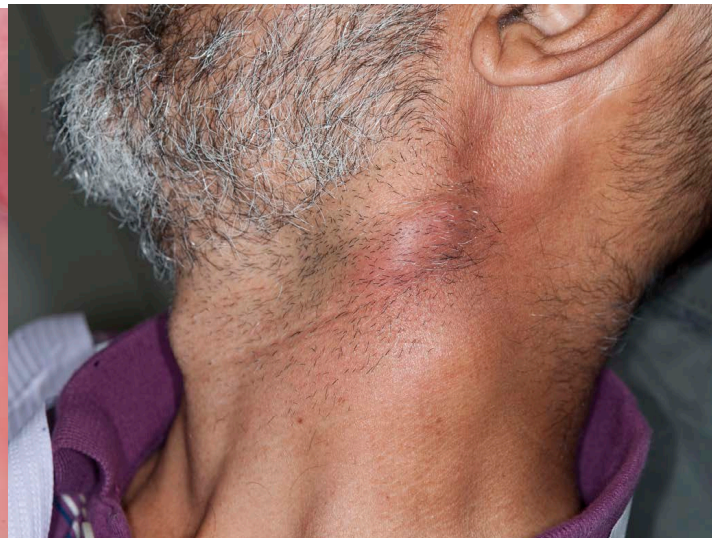
HIV proteins tat and gp120 expression disrupt epithelial tight junctions and may facilitate HPV infection



Tugizov SM et al Virology. 2013;446(0)

Oral HPV-associated lesions/disorders (HPV-OL) in HIV+ patients

Prevalence of HPV-OL in HIV+ patients

Authors & year	N	Gender (%)	Age (years-old)	HAART (%)	HPV-OL	
					Prevalence %	Type
HIV/AIDS adult patients						
(Estrella, 2015)	29	Male (93.1)	32.5–44	(89.6)	3.4	SCP, MEH, VV, CA
(Anaya-Saavedra et al., 2013)	787	Male (93.4)	27–40	(30.9)	6.9	SCP, MEH, VV
(Lourenco et al., 2011)	388	Male (61.6)	Mean: 38	(79.9)	0.6	MEH, CA
(Ortega et al., 2009)	1595	ND	ND	(57.9)	0.5	CA
(Giuliani et al., 2008)	130	Male (54.6)	Mean: 39.6	(79.2)	4.6	HPV-OL
(Kakabadze et al., 2008)	732	Male (82.2)	ND	ND	5.0	Oral warts
(Nunes Mde et al., 2008)	129	Male (100)	31–50	(77.8)	2.3	2 CA, 1 VV
NON HIV/AIDS adult patients						
(Robledo-Sierra et al., 2013)	6,448	Male & female	Adults	ND	<0.1	SCP
(Castellanos & Diaz-Guzman, 2008)	23,785	Male (31.2)	15–97	ND	0.29	SCP

Speicher DJ et al. Oral Dis. 2016;22(Suppl 1):181–192.

HPV Genotypes?

Age[a]	Localization	Clinical Aspect	HPV Type
30	Retroangular (right)	Condyloma	32
35	Gingiva reg 44	Verruca vulgaris	7, 16
25	Gingiva reg 15-17	Condyloma	32
37	Gingiva reg 31/41	Verruca vulgaris	16
42	Lower lip	Condyloma	6
26	Gingiva reg 44	Condyloma	6
32	Lingual gingival, lower jaw	Condyloma	6
58	Upper lip, left	Condyloma	32
35	Gingiva reg 44	Verruca vulgaris	32

	HPV-OL (n = 26)		
	n	*(%)*	*Types of HPV-OL*
Low-risk HPV types			
HPV-1	1	(3.8)	1 MEH+OW
HPV-6	2	(7.7)	2 OW
HPV-11	3	(11.5)	2 SCP, 1 VV
HPV-13	8	(30.8)	5 SCP, 1 OW, 1 MEH, 1 MEH+OW
HPV-32	2	(7.7)	2 MEH
HPV-74	1	(3.8)	1 SCP
Low- and high-risk HPV types			
HPV-13 & 16	1	(3.8)	1 MEH+OW
HPV-13 & 18	1	(3.8)	1 SCP
High-risk HPV types			
HPV-16	1	(3.8)	1 MEH
HPV-31	2	(7.7)	2 OW
Multiple HPV infections			
	4	(15.4)	2 SCP, 2 MEH

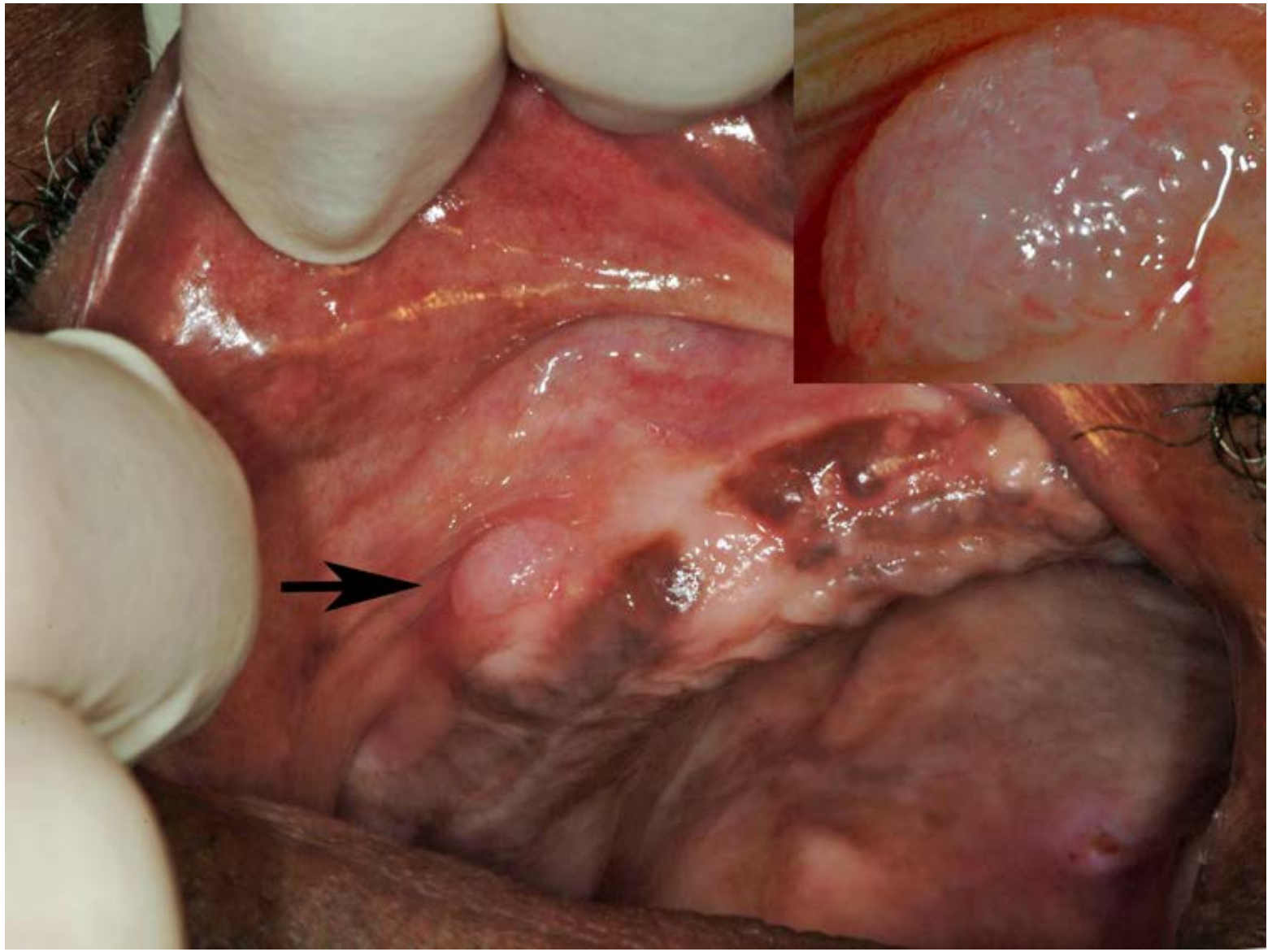
Syrjänen S. Adv Dent Res 23(1) 2011
Anaya-Saavedra G et al. J Oral Pathol Med. 2013;42:443–449

ART and HPV-OL

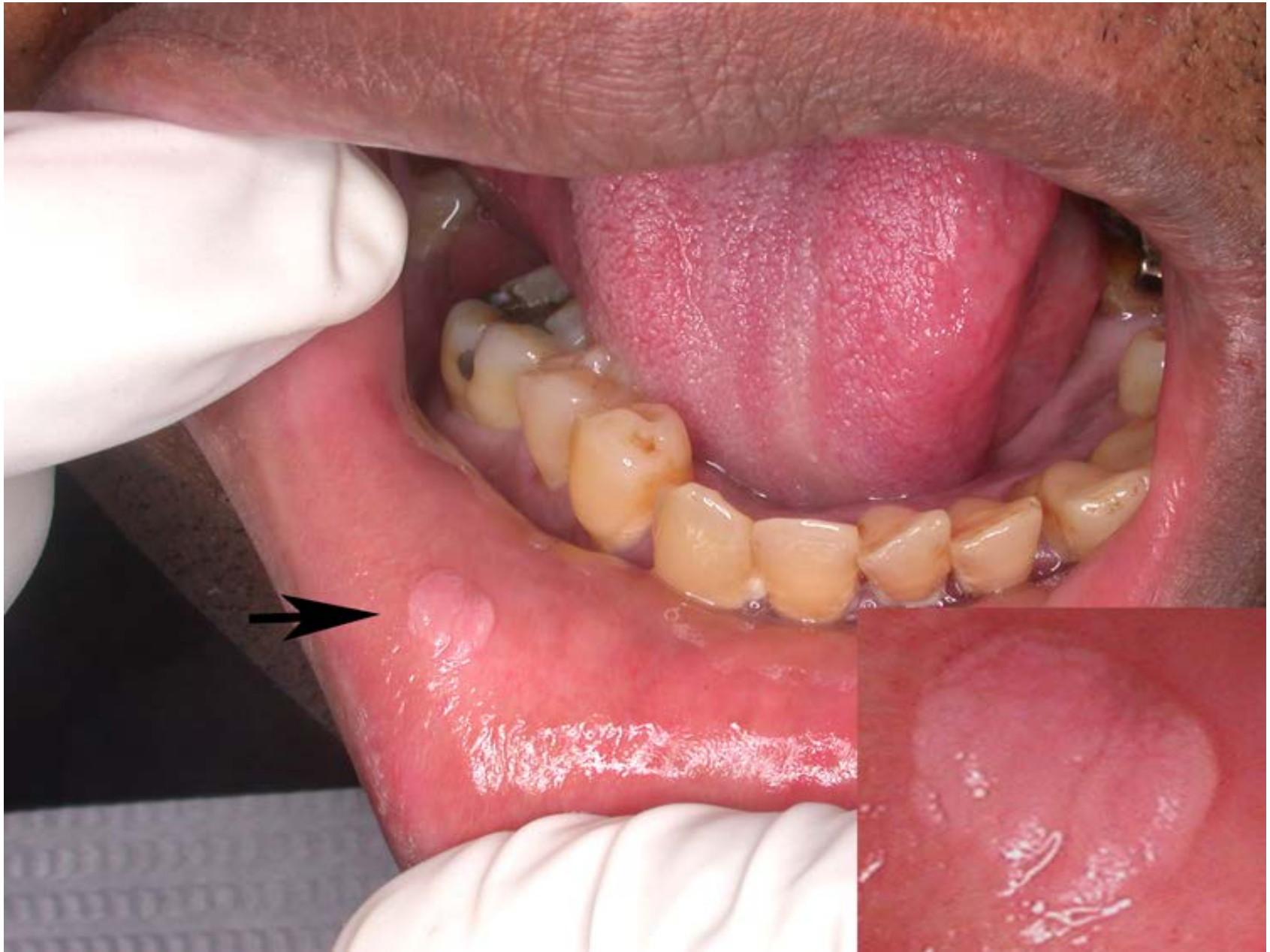
Multivariate analysis			
Age			
Reference: ≤40 years	1.00		
>40 years	2.51	(1.38–4.56)	0.002
CDC stage[26]			
Reference: no-AIDS	1.00		
AIDS	1.09	(0.98–1.22)	0.096
Time under HAART			
Reference: ≤12 months	1.00		
>12 months	3.14	(1.72–5.74)	<0.001

Anaya-Saavedra G et al. J Oral Pathol Med. 2013;42:443–449

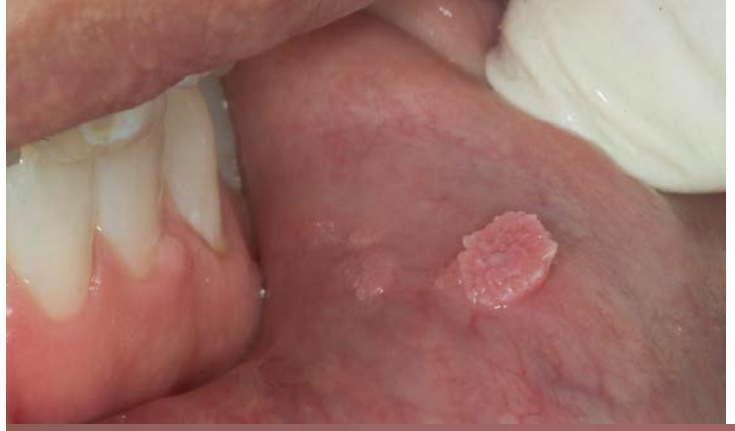
Viral papilloma/Squamous papilloma

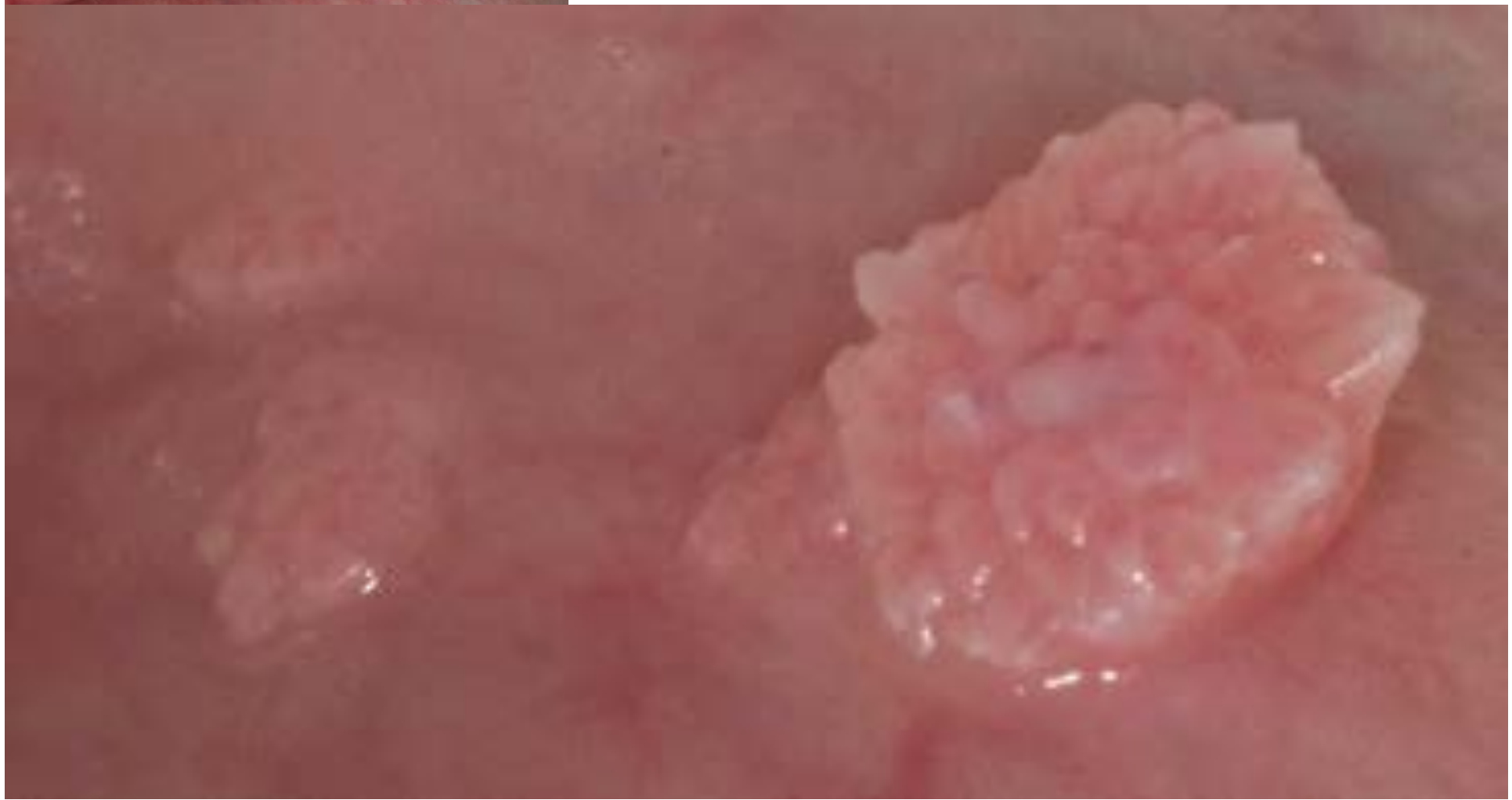


Verruca Vulgaris



Condyloma Acuminatum (Venereal Wart)



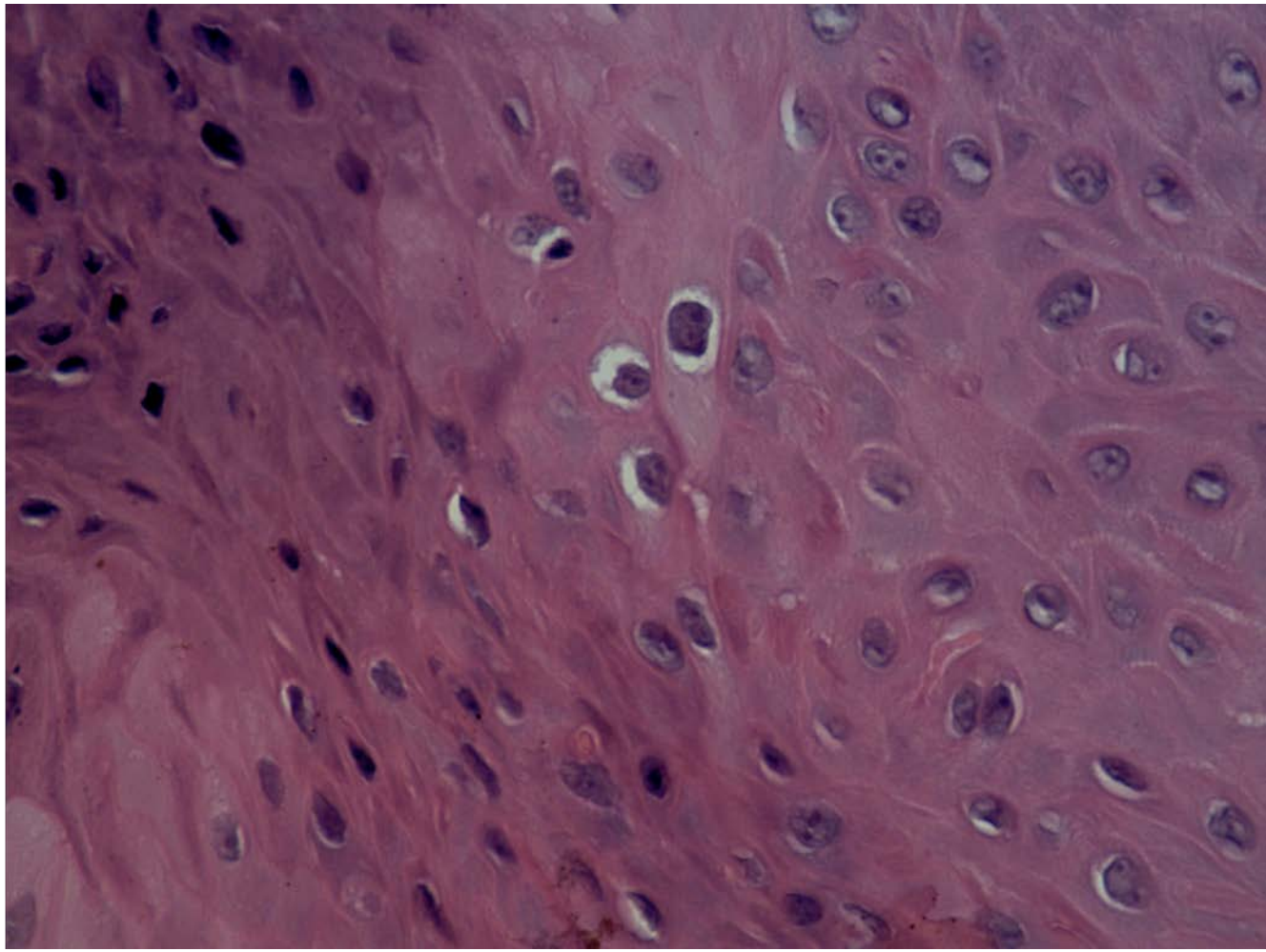


Histopathologic Features HPV-OL (benign)

- Acanthosis
- Koilocytosis
- Bi and multinucleated keratinocytes
- Dyskeratosis
- Mitosoid figures
- Basilar hyperplasia

Koilocytes

- The characteristic cells of HPV infected lesions
- Enlarged, squamous epithelial cells with clear halos around shrunken nuclei
- Produced when a portion of the HPV genome encodes a protein that binds to and disrupts the cytoplasmic keratin network



Not all papillary lesions harbor HPV

Mucosal

HPV genotype	Oropharynx $n = 43$	Oral cavity $n = 31$
Low-risk	1 (2.3)	9 (29.0)
6	1 (2.3)	9 (29.0)
11	0 (0.0)	0 (0.0)
High-risk	1 (2.3)	5 (16.1)
16	0 (0.0)	3 (9.7)
18	0 (0.0)	1 (3.2)
35	0 (0.0)	1 (3.2)
51	1 (2.3)	0 (0.0)
Undetermined	1 (2.3)	1 (3.2)
74	1 (2.3)	1 (3.2)
Overall	3 (6.9)	15 (48.3)

Cutaneous

HPV genus and genotype	Oropharynx $n = 41$	Oral cavity $n = 31$
Alpha	0 (0.0)	0 (0.0)
Beta	4 (9.8)	5 (16.1)
5	0 (0.0)	1 (3.2)*
12	2 (4.9)	1 (3.2)*
23	1 (2.4)	2 (6.4)
93	0 (0.0)	0 (0.0)
96	0 (0.0)	1 (3.2)
98	0 (0.0)	0 (0.0)
110	0 (0.0)	1 (3.2)†
120	1 (2.4)	0 (0.0)
Gamma	2 (4.9)	3 (9.7)
121	0 (0.0)	1 (3.2)†
123	0 (0.0)	1 (3.2)
130	0 (0.0)	1 (3.2)‡
131	1 (2.4)§	1 (3.2)‡
156	1 (2.4)	0 (0.0)
SD2	1 (2.4)§	0 (0.0)
Mu	0 (0.0)	0 (0.0)
Overall	6 (14.7)	7 (22.6)‖

N=72+ papillary lesions tested for HPV
n=18 mucosal HPV+ (8 hi risk)
n=13 cutaneous HPV+
Rest were negative

Dona MG et al. Head and Neck 2016

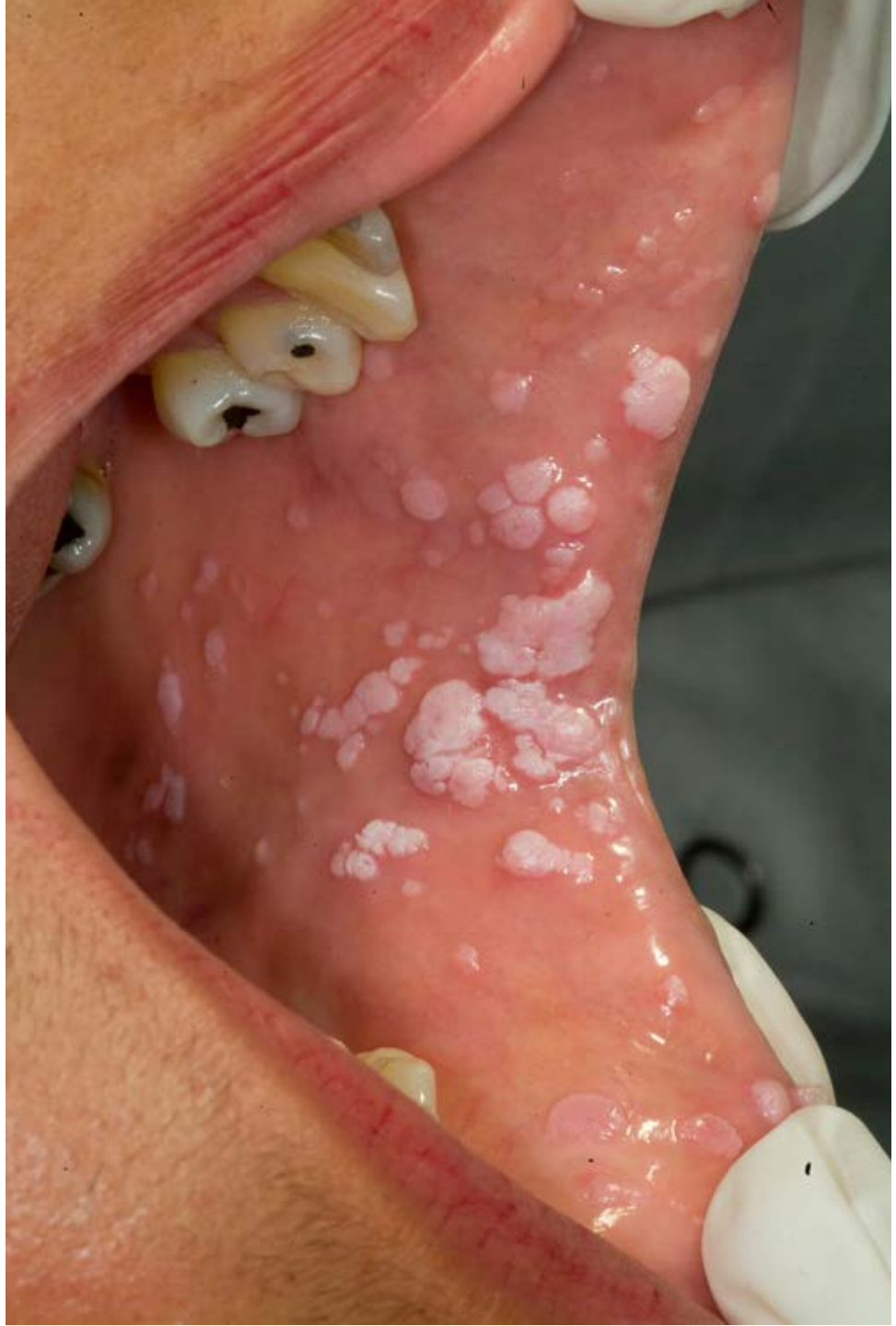
HPV lesions in Pediatric Population

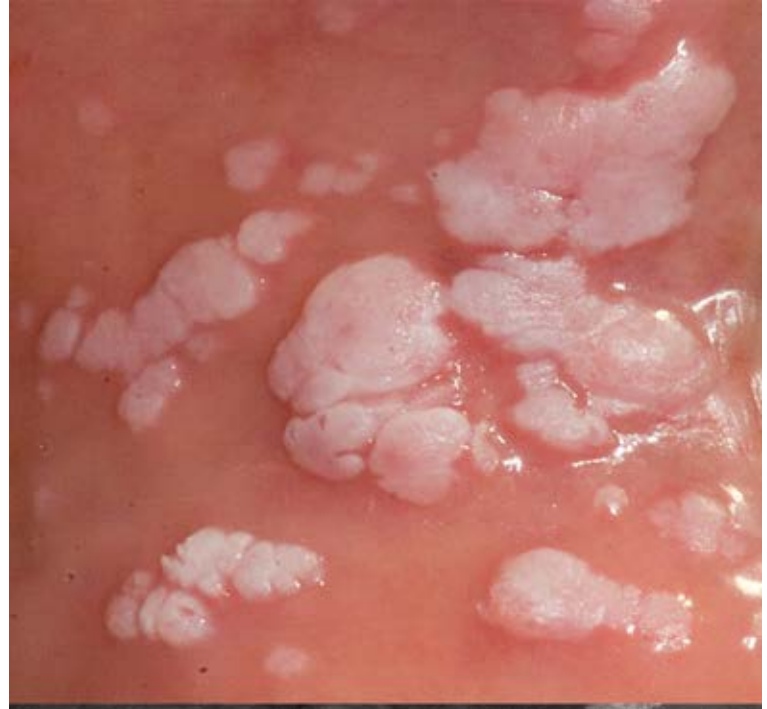
- Oral HPV infection is relatively common in infants and children due to transmission from parents (perinatal, breast milk, auto/hetero-inoculation, or possibly by sexual abuse).
- Rates are higher in HIV+ children (approx 10% prevalence, 2x that of HIV- children).
- Most infections are transient and rarely result in clinical lesions.

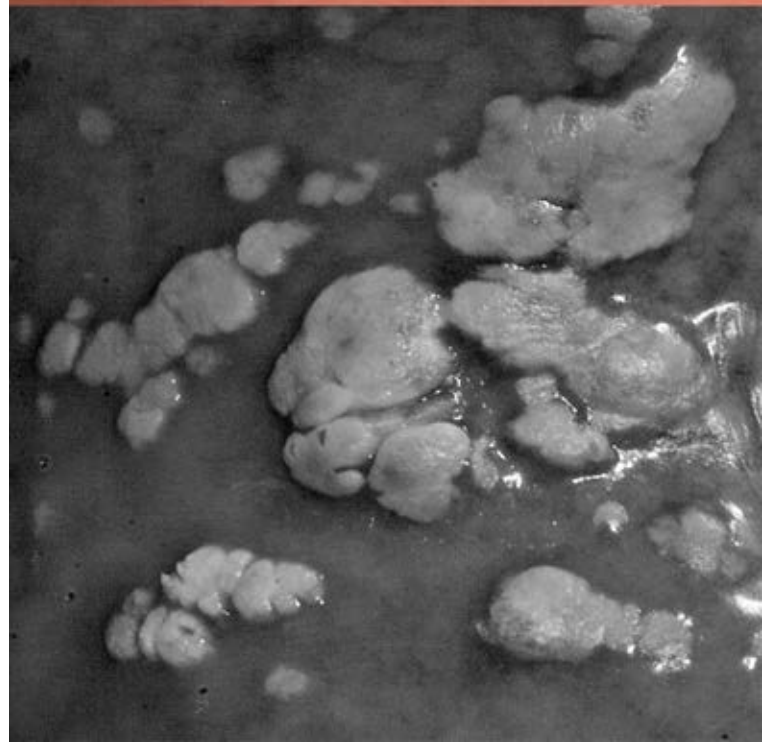
Pinheiro RS et al. J Clin Pathol 2011;64; Moscicki AB et al AIDS Patient Care and STDs 2014.

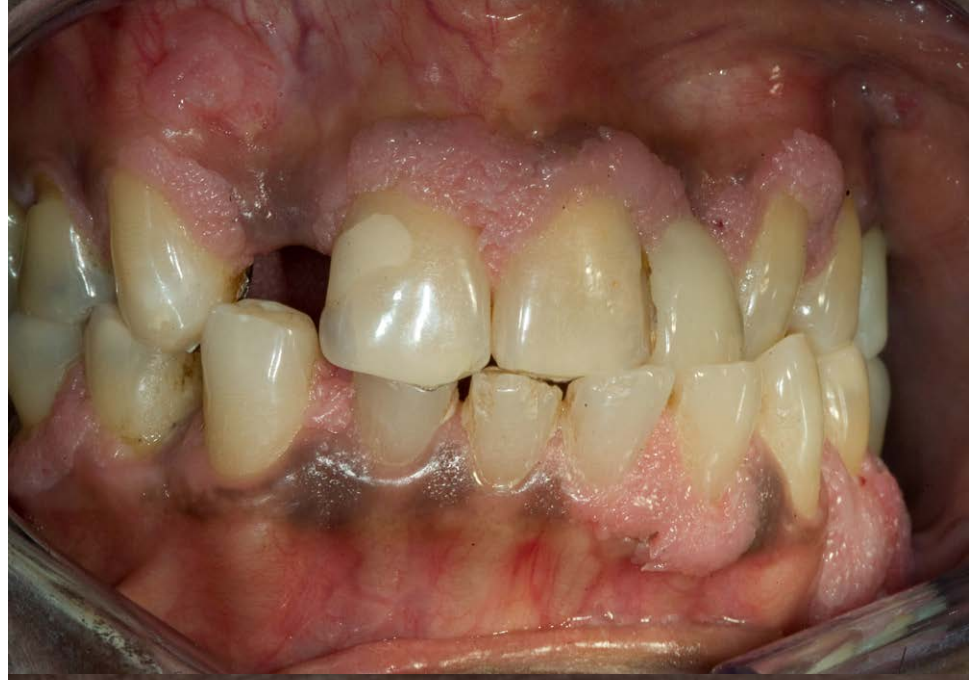
Florid Papillomatosis
Oral HPV-Associated Papillomatosis
Multifocal Epithelial Hyperplasia

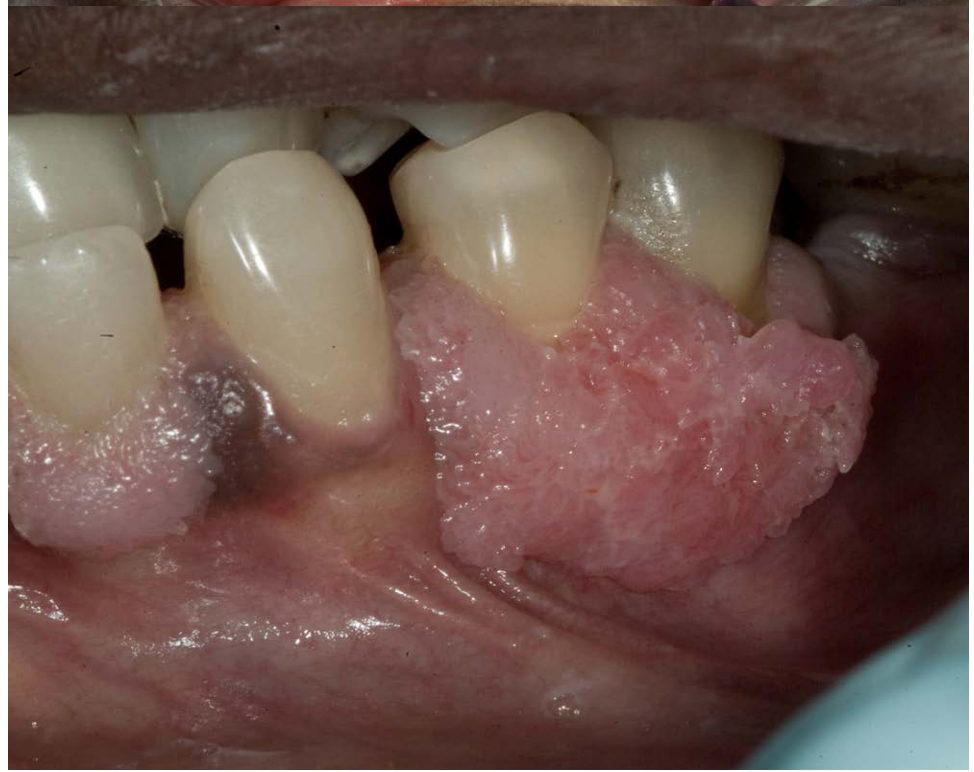
– Increased prevalence since advent of ART therapy
– Multiple HPV types

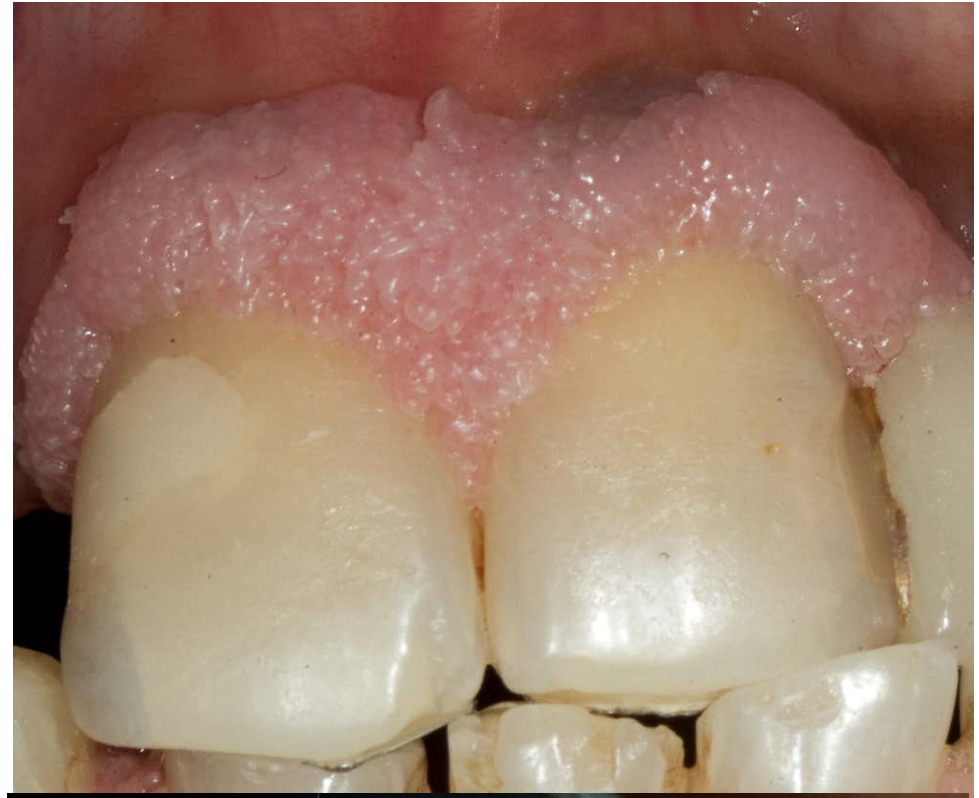


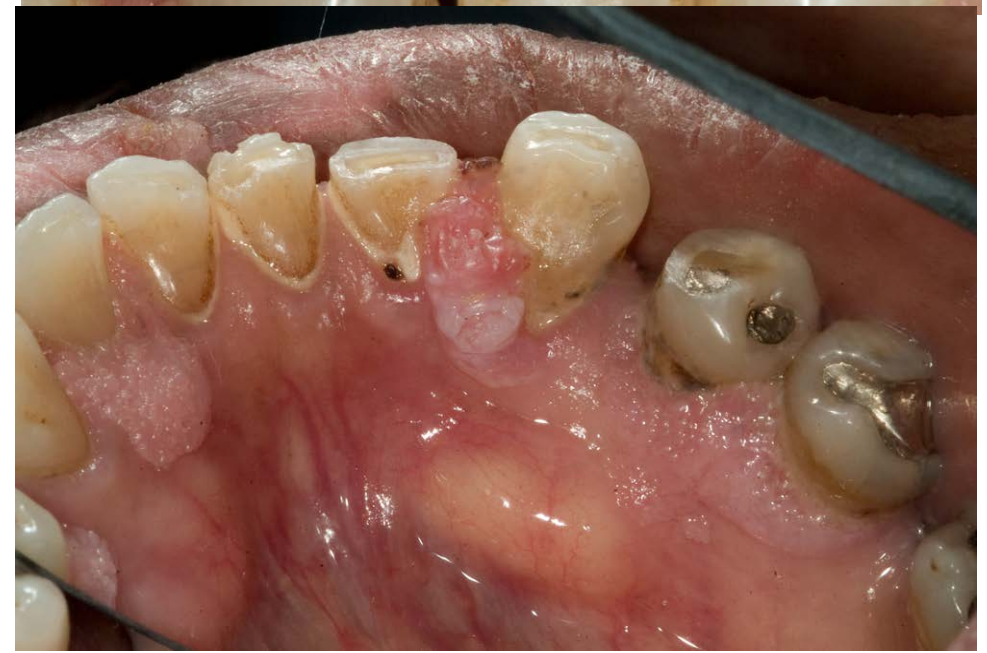


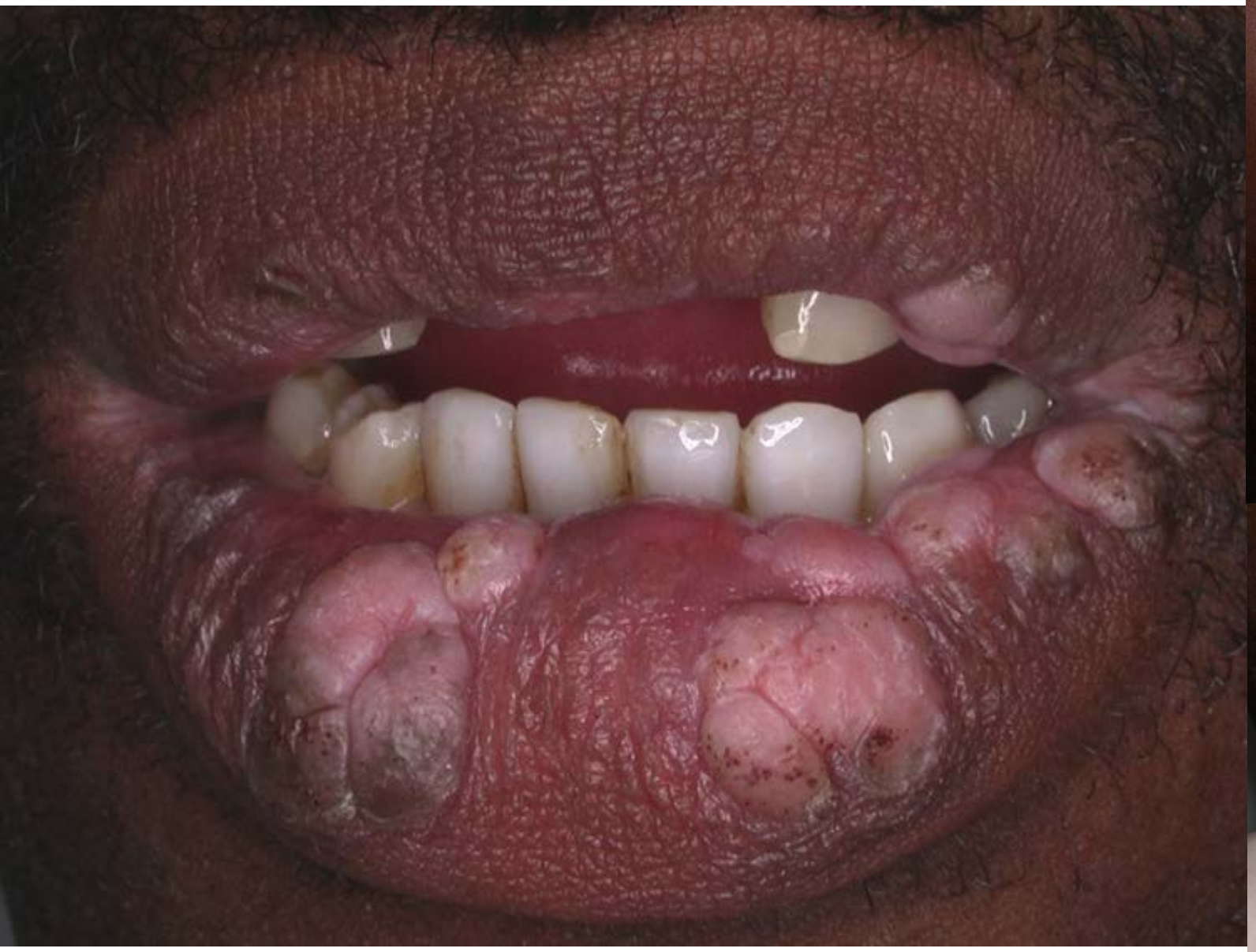


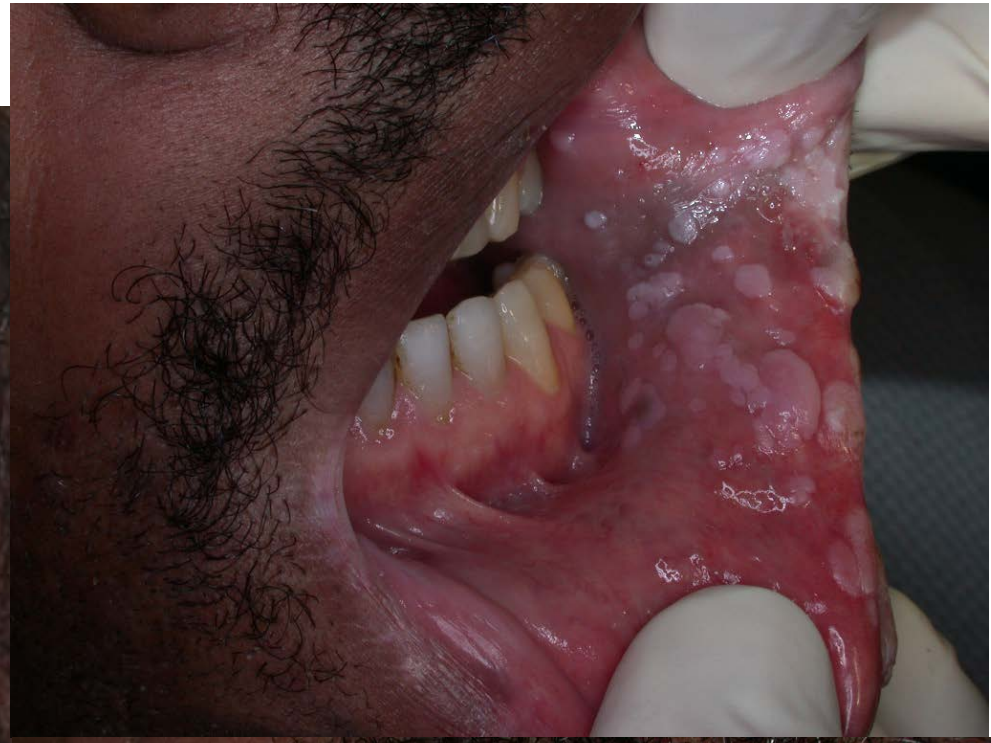


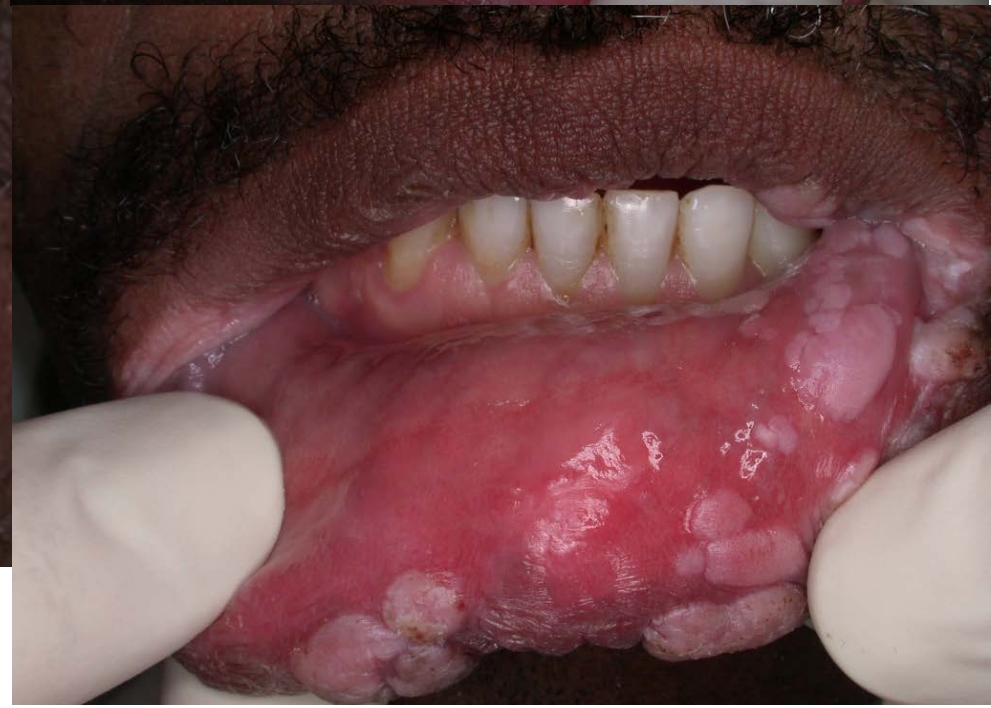








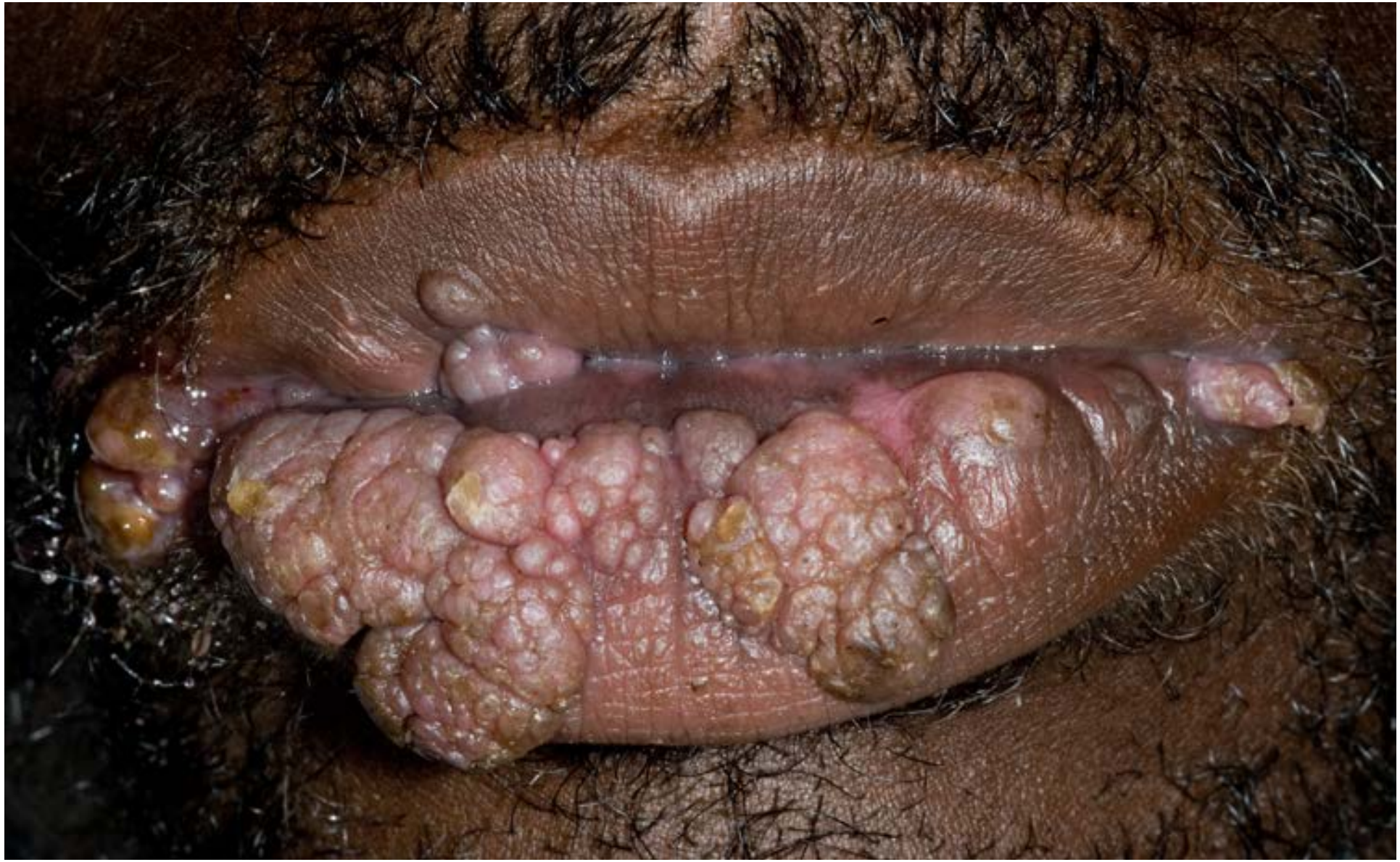


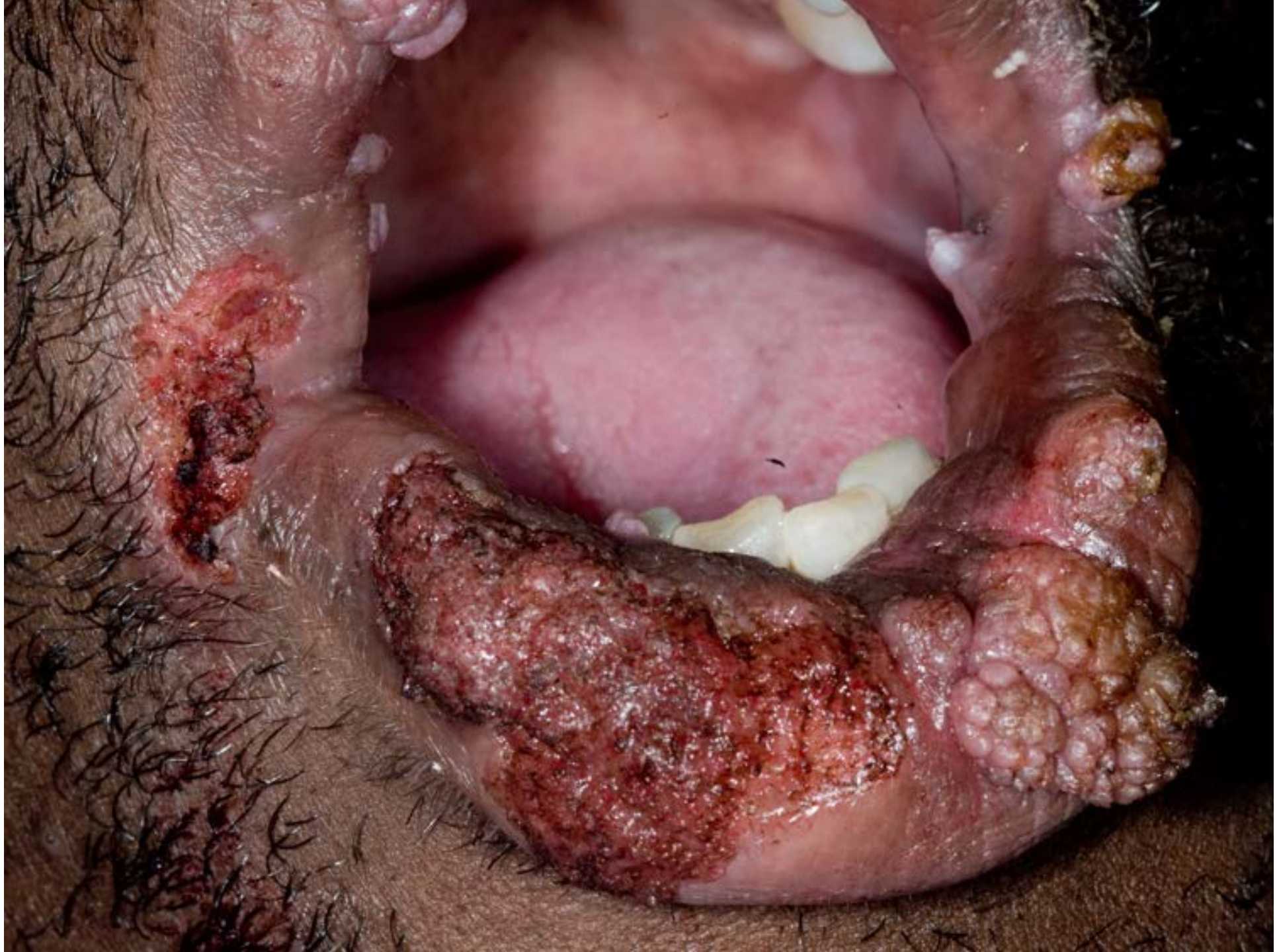


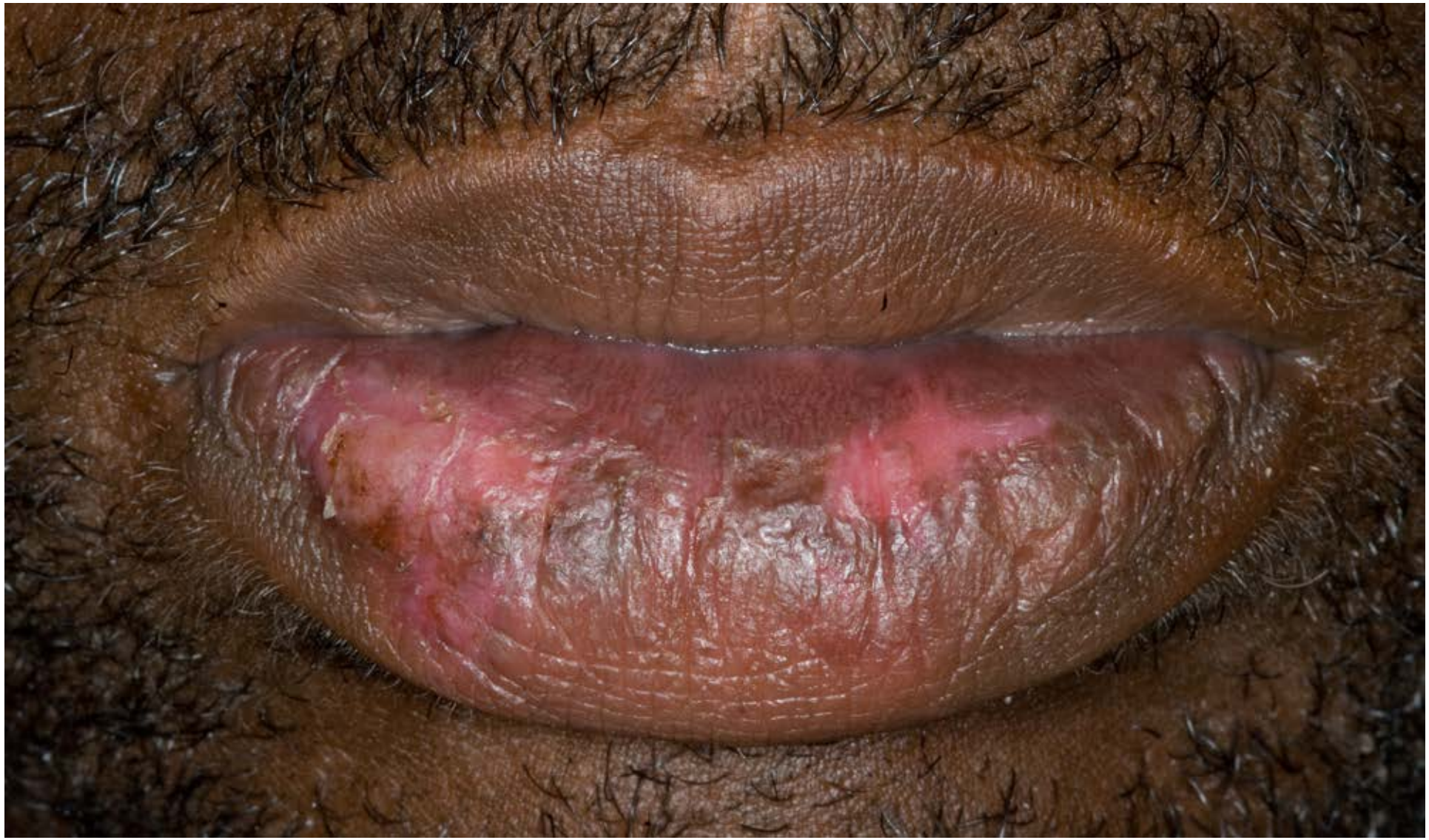
Management of Benign HPV-OLs

- Solitary Lesions
  - Excision is warranted.
  - Recurrence is possible
- Multiple Lesions (no evidence-base)
    - High-power evacuation is imperative to prevent transmission of HPV.
  - Controversial treatment
    - Excision/ablation vs topical vs intralesional therapy (or combination)
  - Recurrence more likely
  - Excision/Ablation
    - Carbon dioxide laser, electrosurgery, scalpel removal
  - Topical therapy
    - Podophyllin resin
    - Imiquimod (extra-oral use only)
    - Cidofovir
    - Interferon
  - Intralesional therapy
    - Interferon

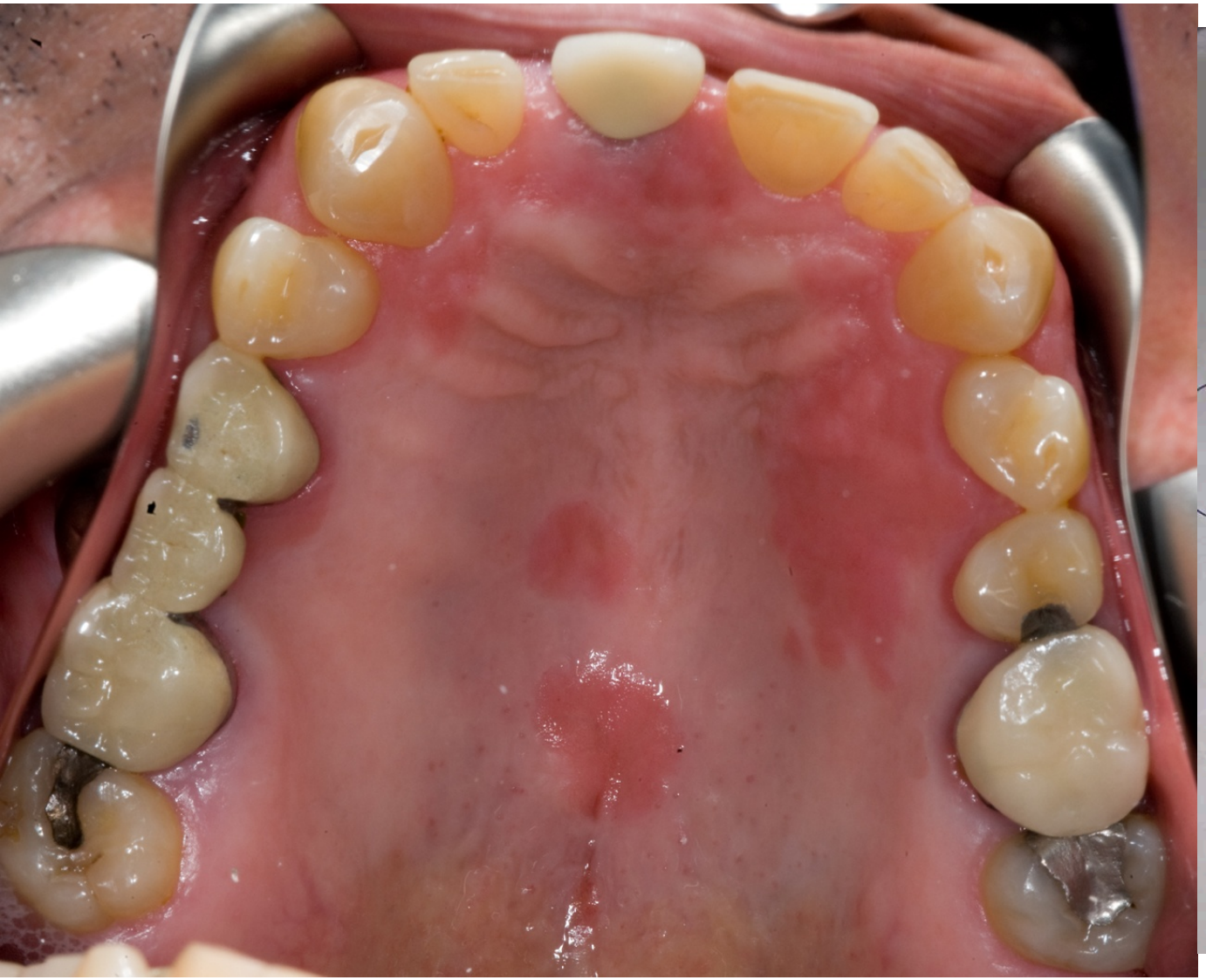
Baccaglini L. OOOO 2007;103(suppl 1):S50.e1-S50.e23)







HPV-Associated Oral Potentially Malignant Disorders and Cancers in HIV-infected Patients



GENPATH

Final Report

DOCTOR [redacted]

PATIENT [redacted]

SAMPLE

Specimen ID: [redacted]
Date of Report:
Date Collected:
Date Received:
Source: 1) Palatal gingiva, tooth #4 2) Palatal gingiva, tooth #6
Clinical Information: Epithelial dysplasia severe.

HPV (Human Papillomavirus) In Situ Hybridization

RESULTS

HPV Low Risk: Negative

HPV High Risk: Positive

p16: Positive

COMMENT

Both blocks stained.

INTERPRETIVE INFORMATION

Human Papillomavirus is an epidermotropic papillomavirus associated with an increased risk of both genital warts (condyloma acuminata) in low risk viral genotypes, and cervical and throat squamous carcinoma in high risk viral genotypes.

Low risk HPV: PathoGene HPV Type 6/11 (ENZ-32885, Enzo Life Sciences, Farmingdale, NY) has affinities to HPV genotypes 6 and 11. These types are associated with development of condyloma acuminata.

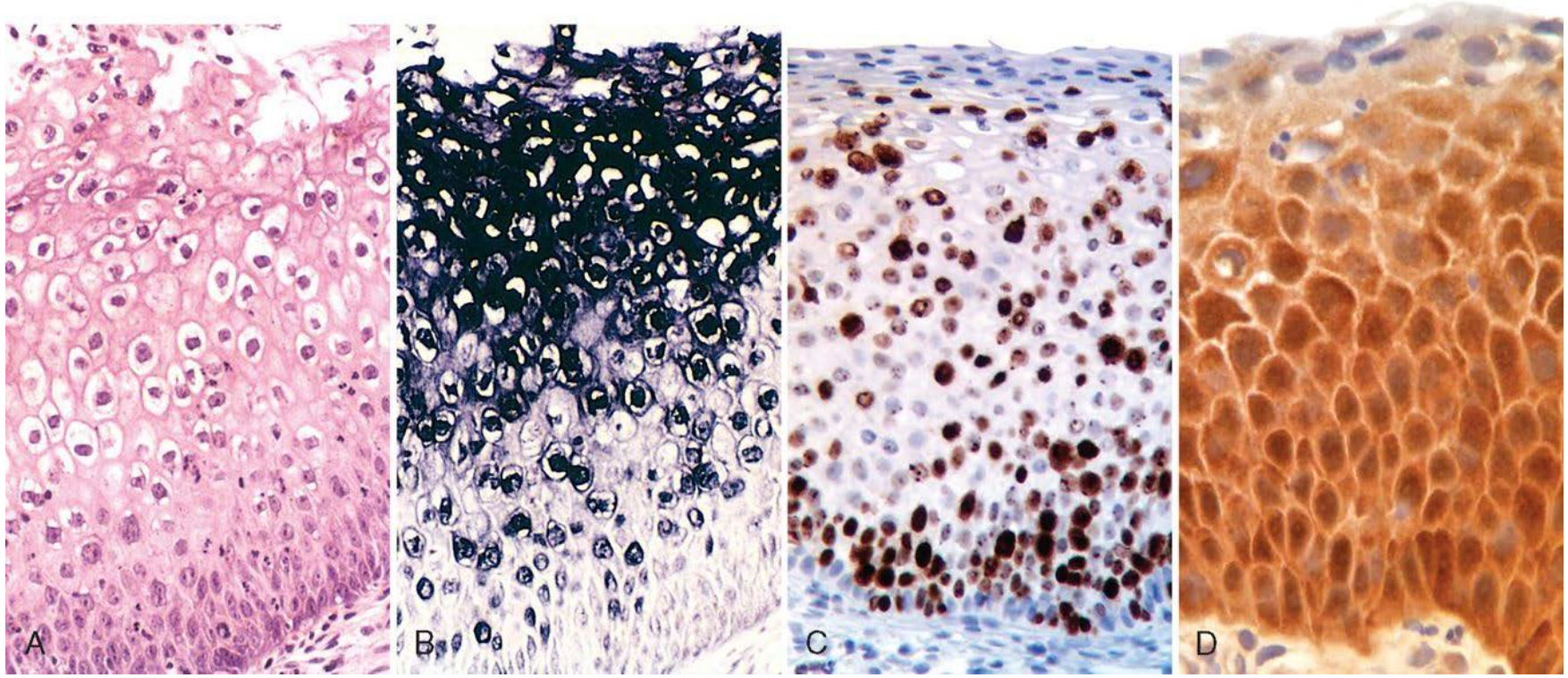
High risk HPV: PathoGene HPV Type 16/18 Probe (ENZ-32886, Enzo Life Sciences, Farmingdale, NY) has affinities to HPV genotypes 16 and 18. These types are associated with an increased risk of oropharyngeal carcinoma and a majority of uterine cervical carcinoma and associated high grade SIL.

These ISH probes are used in conjunction with a signal amplifying reagent (rabbit antibiotin linker, ENZ-32892, Enzo Life Sciences, Farmingdale, NY)

Gardasil® (Human Papillomavirus Quadrivalent Vaccine, Merck & Co., Inc.) protects against low risk types 6 and 11 and high risk types 16 and 18. Vaccine administration must predate exposure to HPV to be effective.

A negative control and a positive control for each ISH stain have been reviewed and accepted.

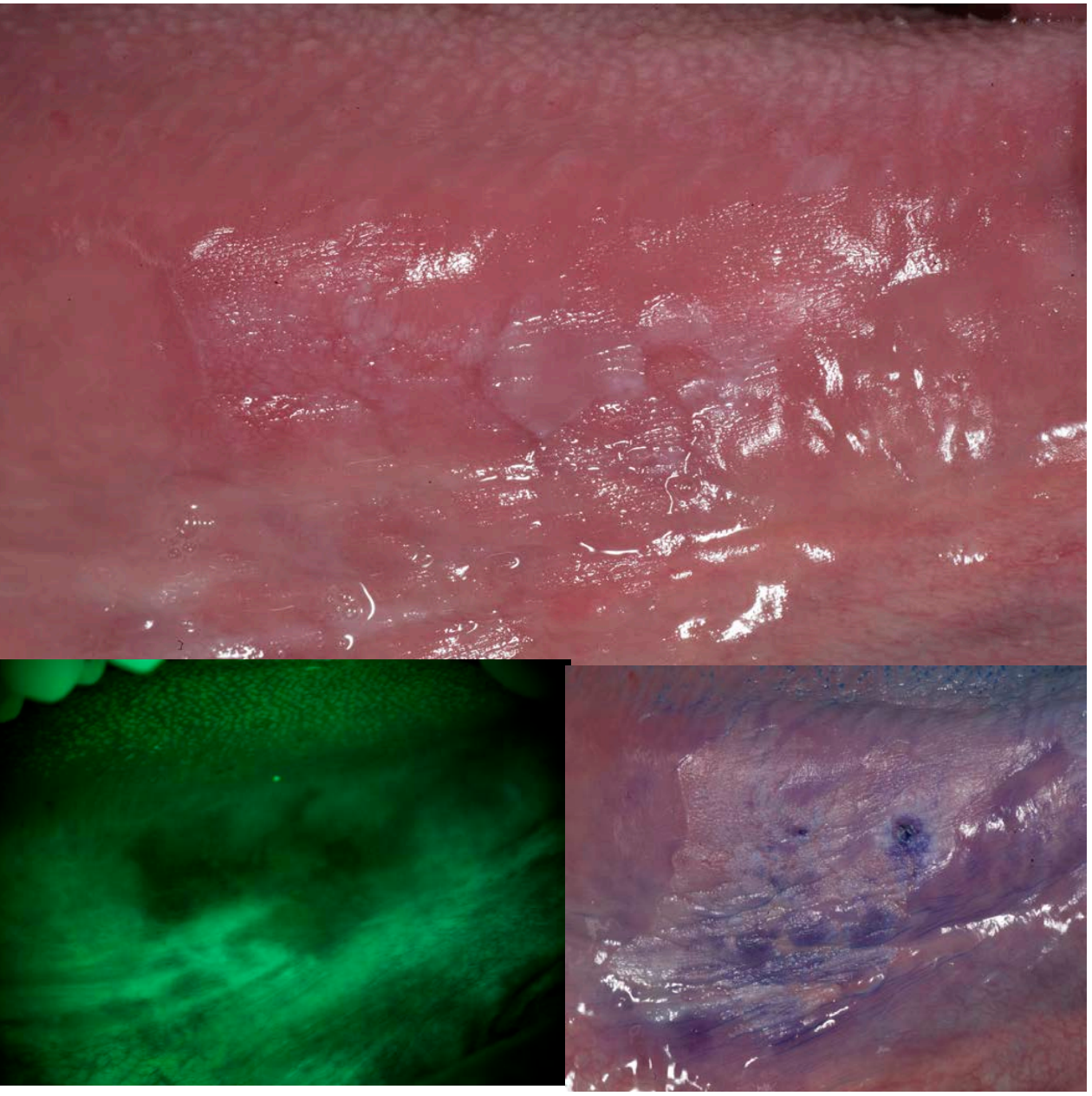
Erythroplakia, candida negative. Biopsy: severe dysplasia, HPV16+, p16+

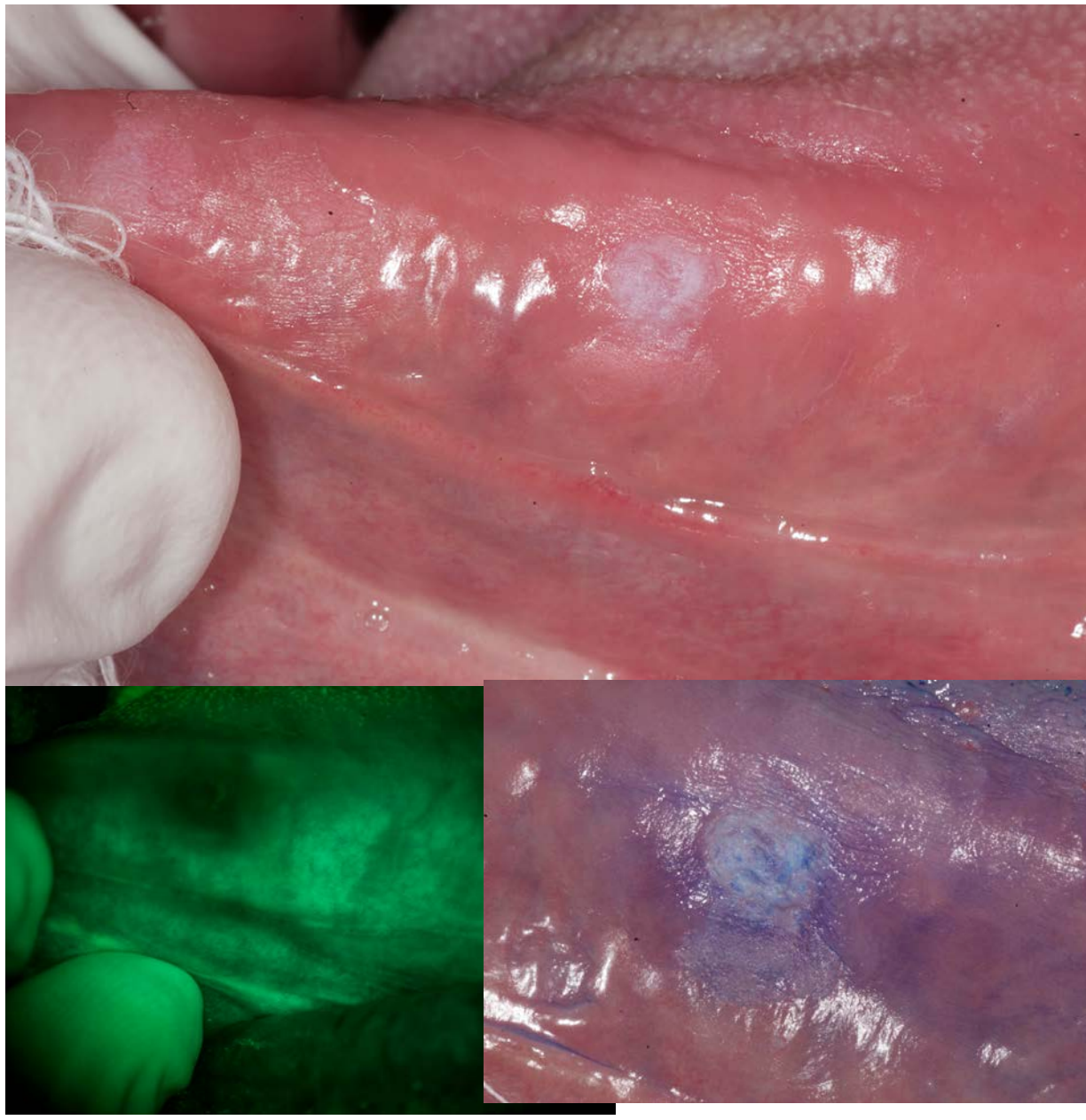


H & E HPV DNA ISH Ki-67 P16INK4 upregulation

HPV-in-situ hybridization tests (and more recently RNA ISH) can reveal viral integration

p16 (not to be confused with HPV 16) immunohistochemistry is a reliable surrogate for HPV+ oropharyngeal cancer. However, it isn't a good surrogate for HPV in oral cavity cancers (ie a significant proportion of oral cavity cancer are p16+ yet HPV negative)



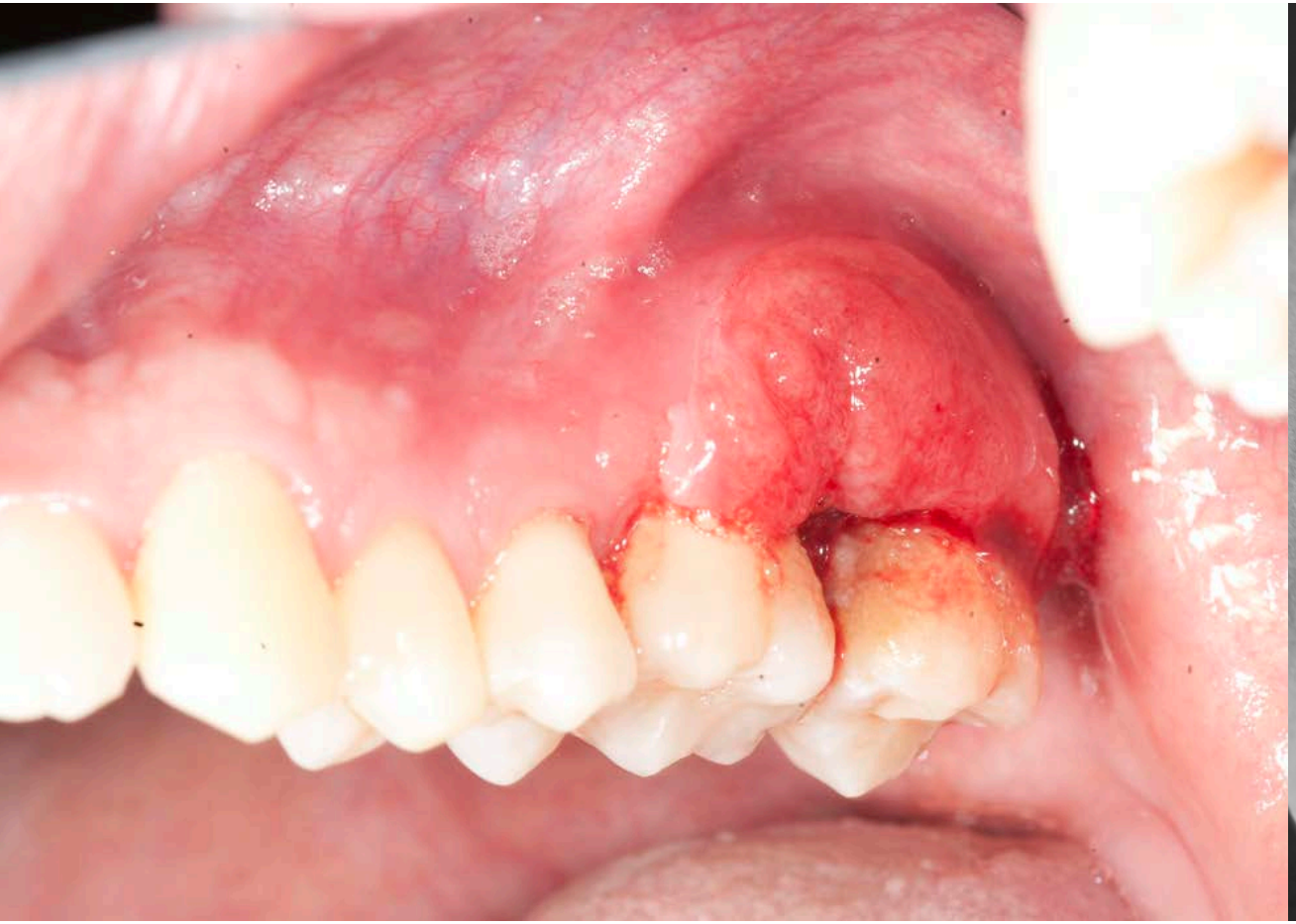


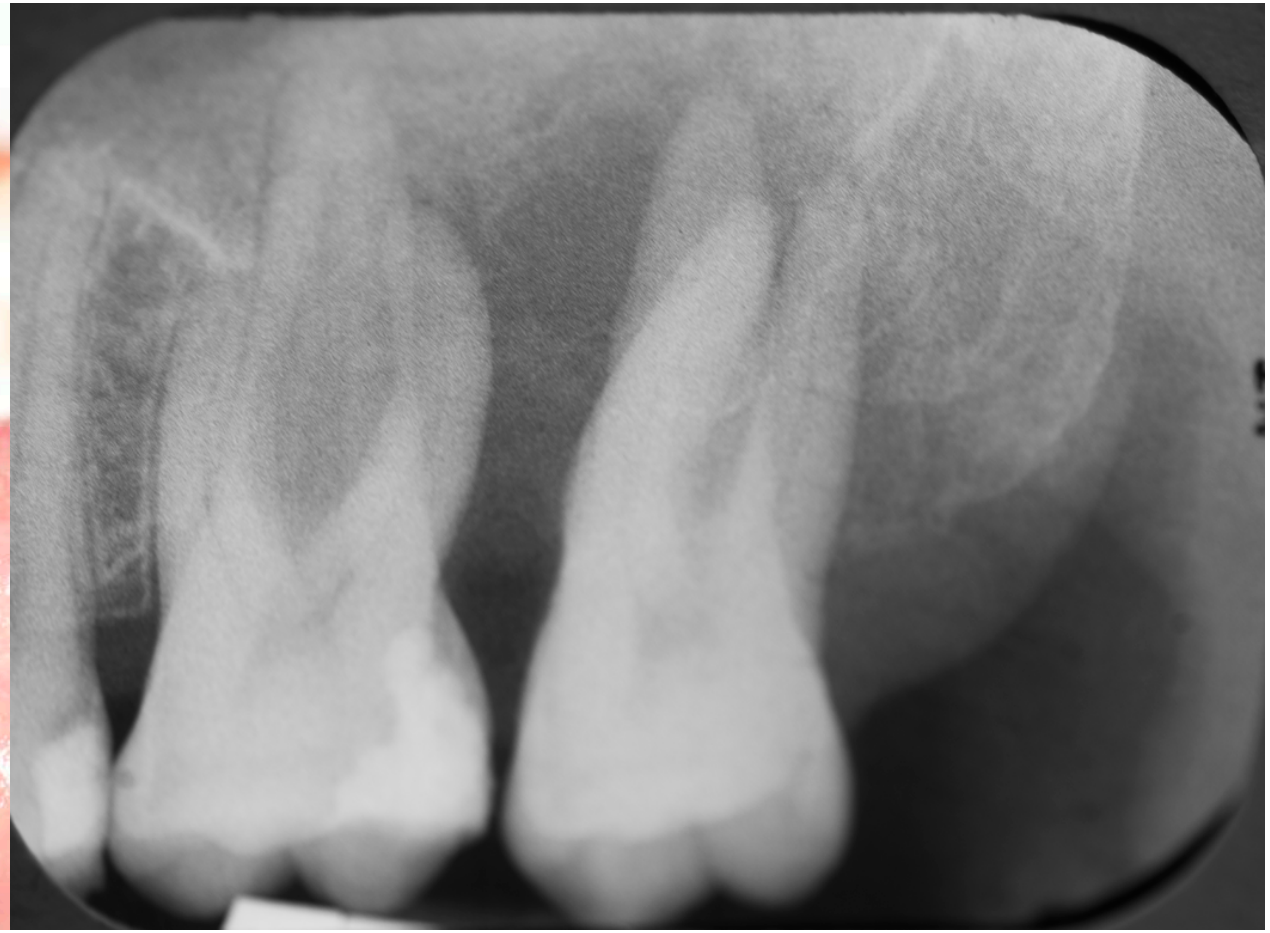
Leukoplakia, candida negative. Biopsy: severe epithelial dysplasia, HPV16+, p16+

	All sites HNSCC ($n=40$)	Oropharynx ($n=12$)	Oral cavity ($n=12$)
IHC: p16+	15 (38%)	7 (58.3%)	5 (41.7%)
ISH+	11 (28%)	4 (33.3%)	3 (25.0%)
PCR+for HPV HR	12 (30%)	5 (41.7%)	3 (25.0%)
HPV+	12 (30%)	5 (42%)	4 (33.3%)

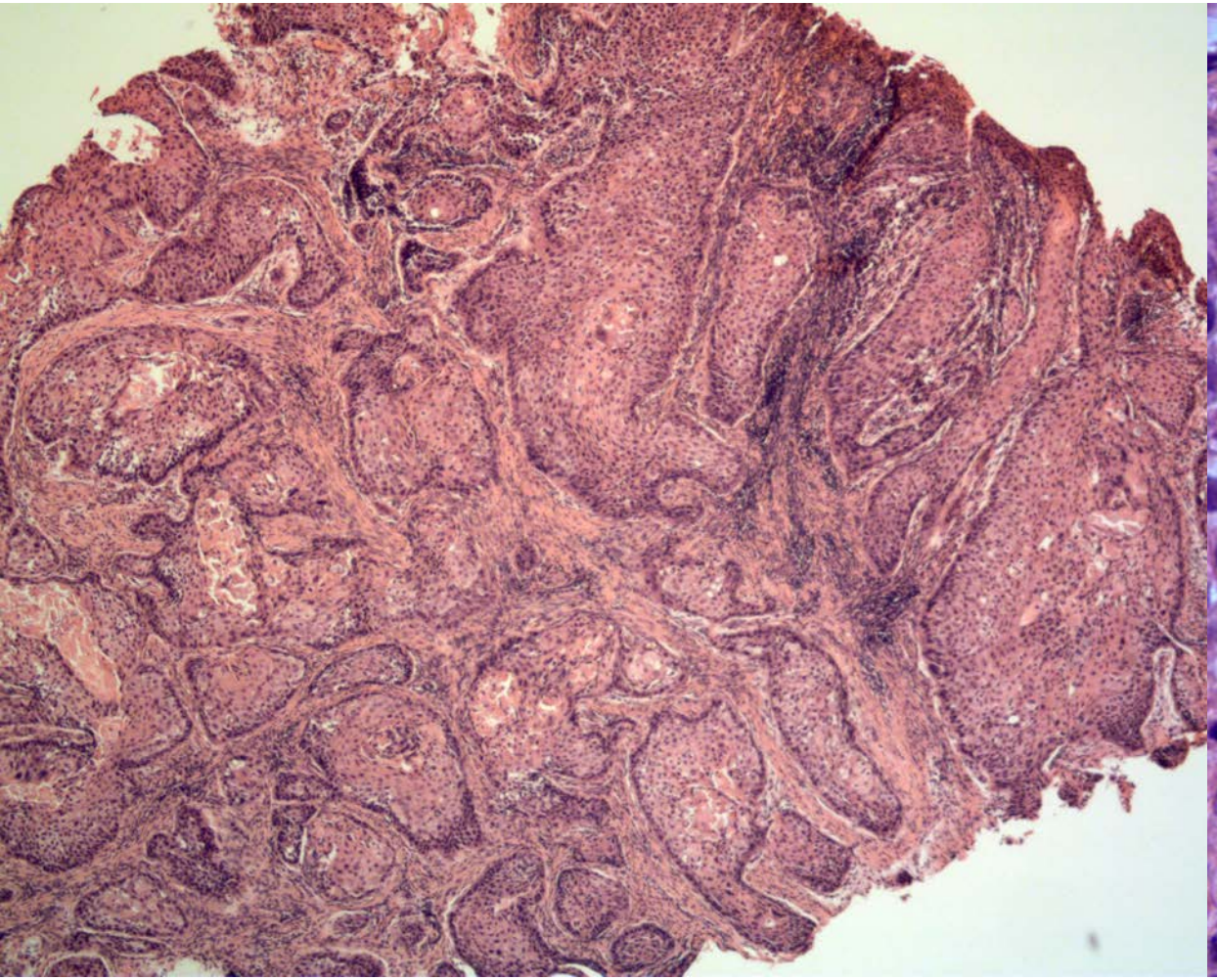
Picard et al AIDS 2016, 30:1257–1266

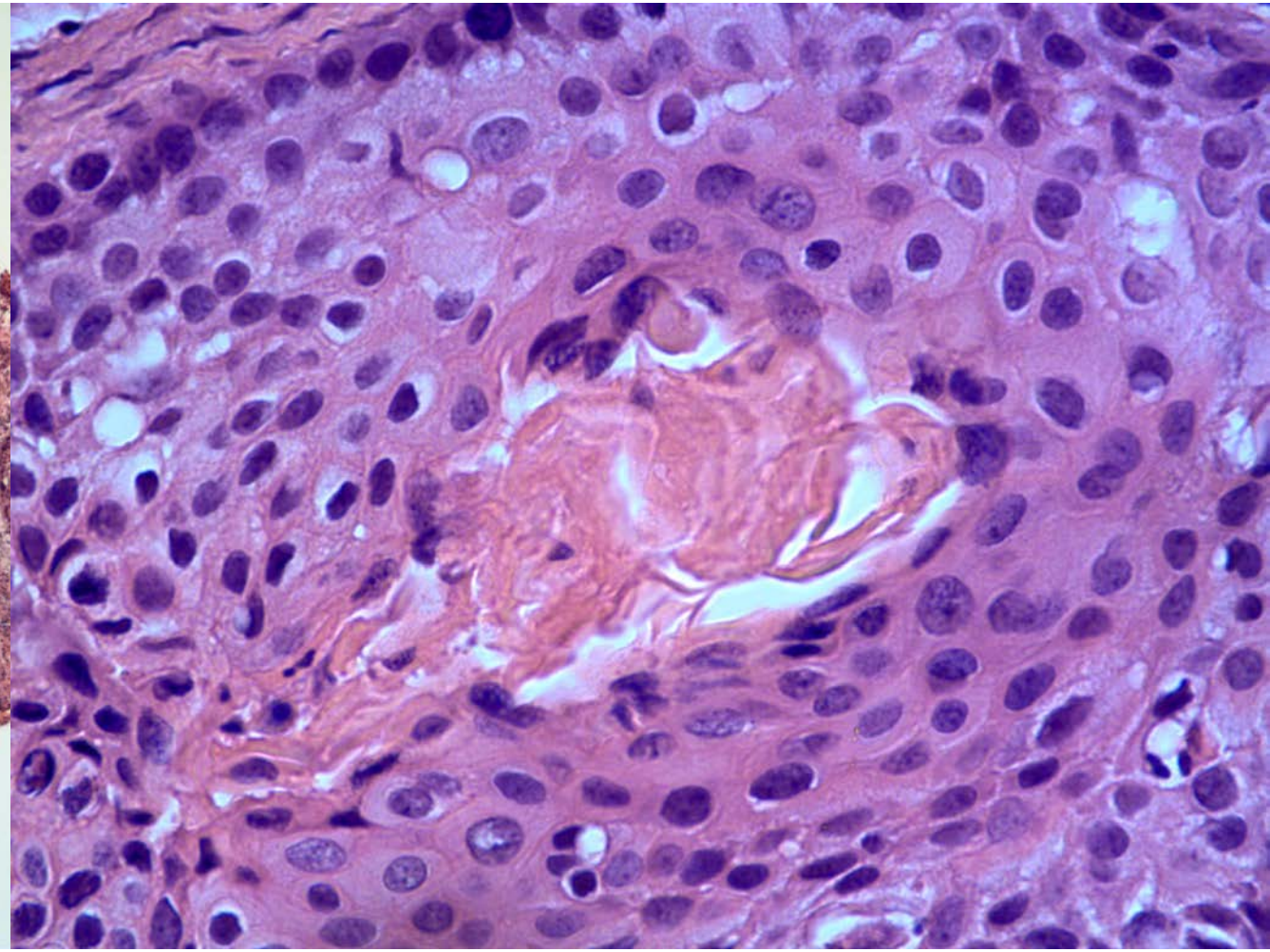
- 43-year-old male patient presents with persistent periodontal disease and bone loss in posterior left maxilla despite SRP/perio tx and antibiotics
- Medical history: HIV+ (undetectable VL/CD4>400), high cholesterol
- Medications: abacavir, tenofovir, raltegravir, ritonavir, simvastatin, Allergy to amoxicillin
- Social history: Non-smoker, etoh+

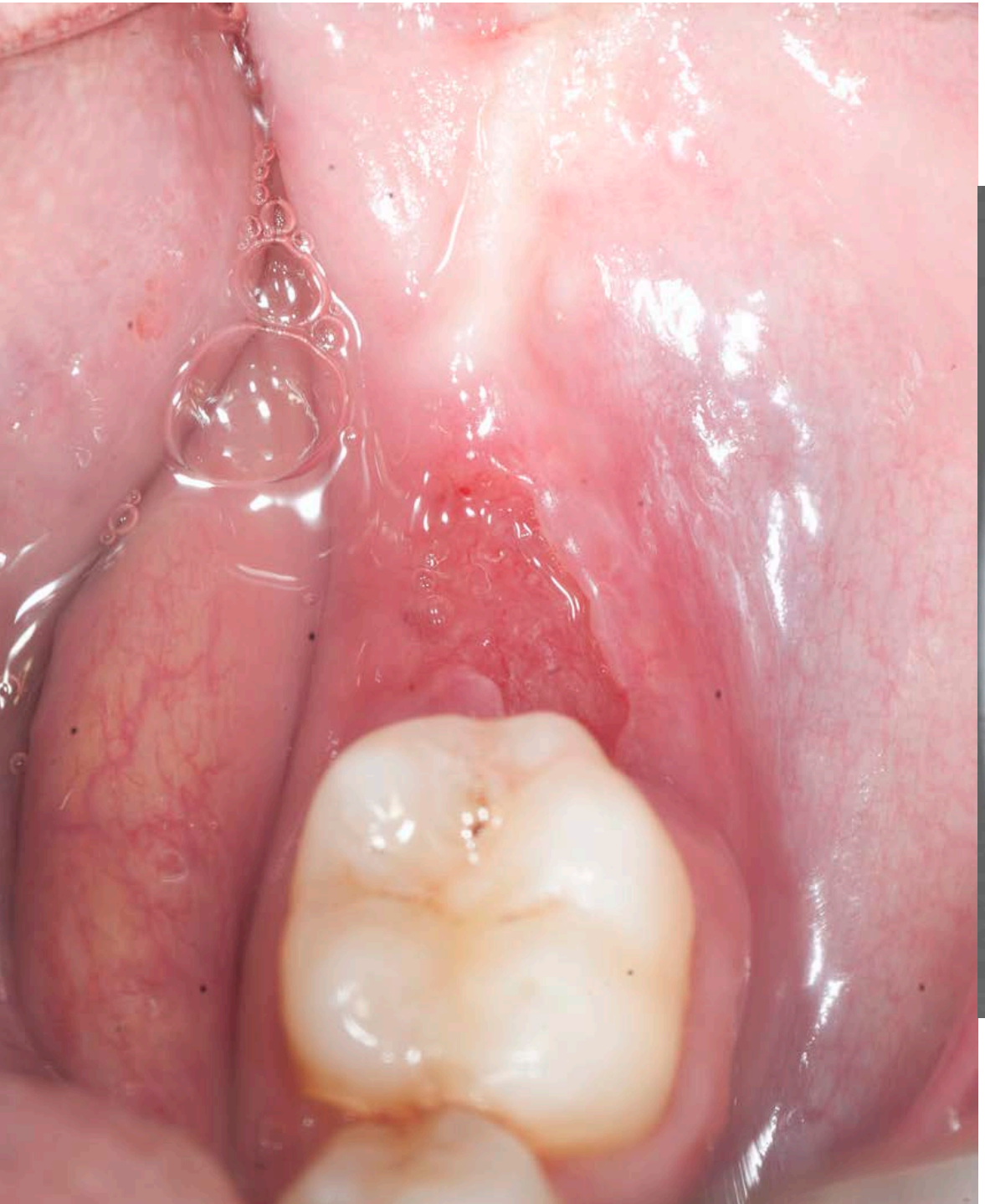


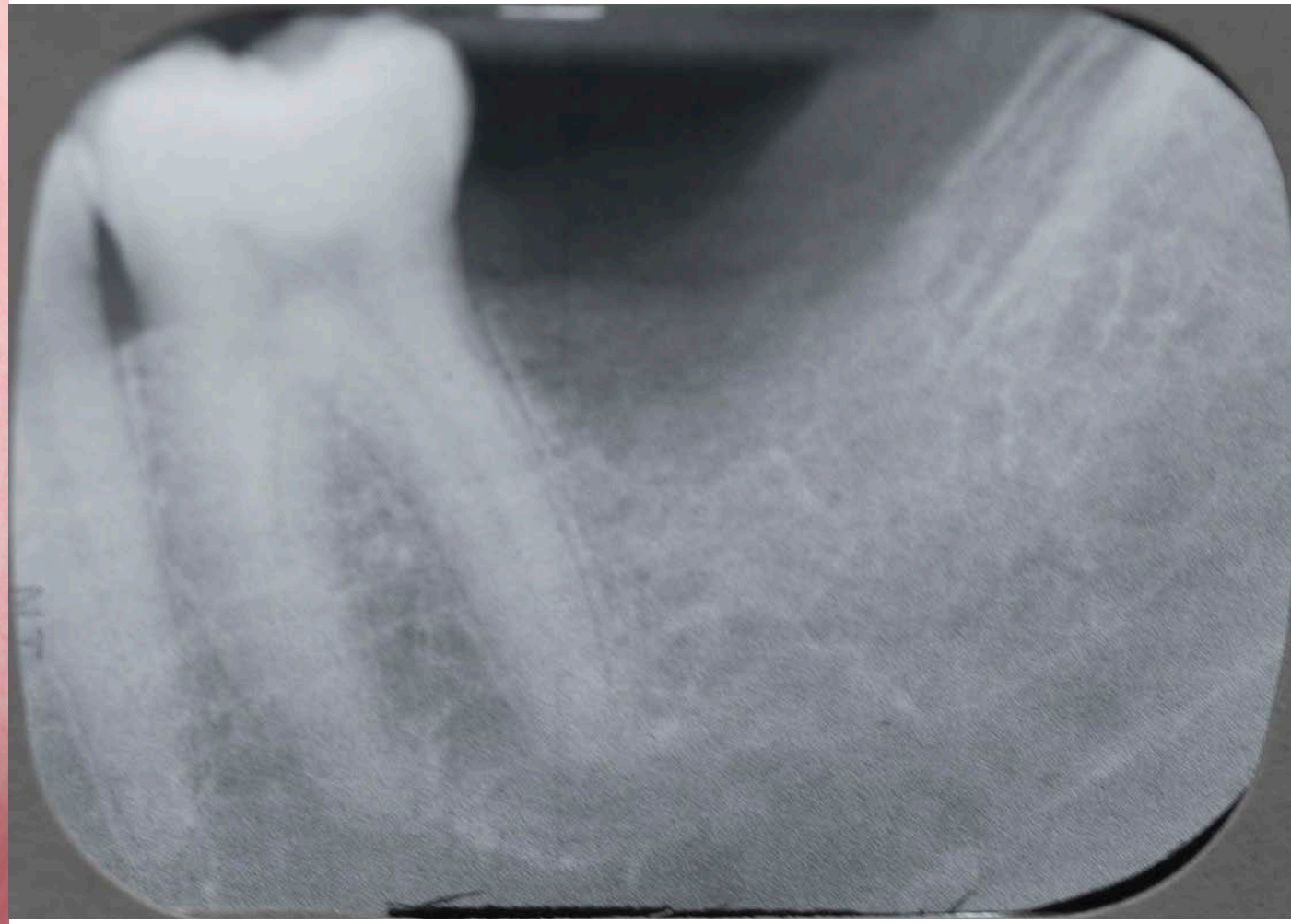


Squamous cell carcinoma HPV16/p16+

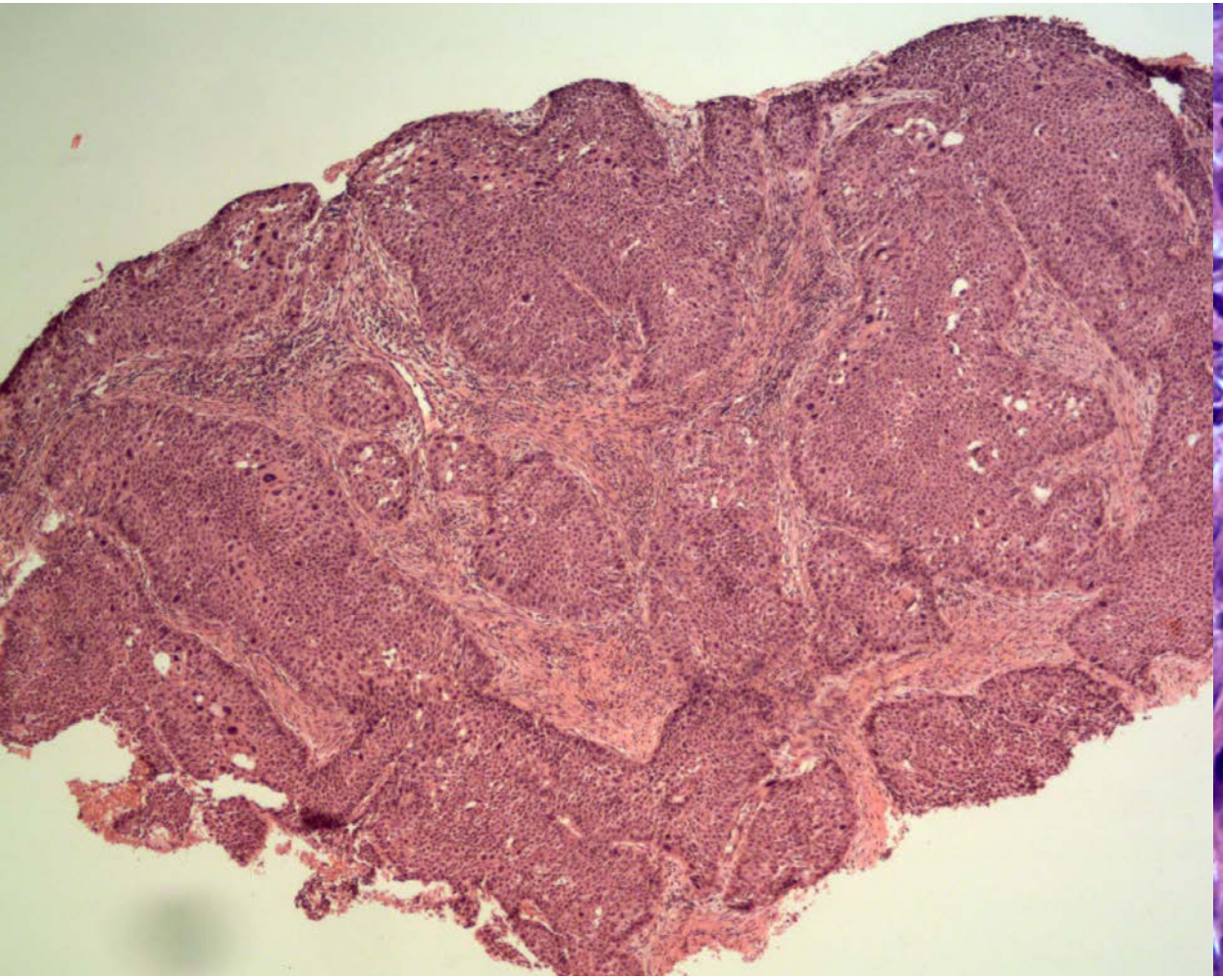


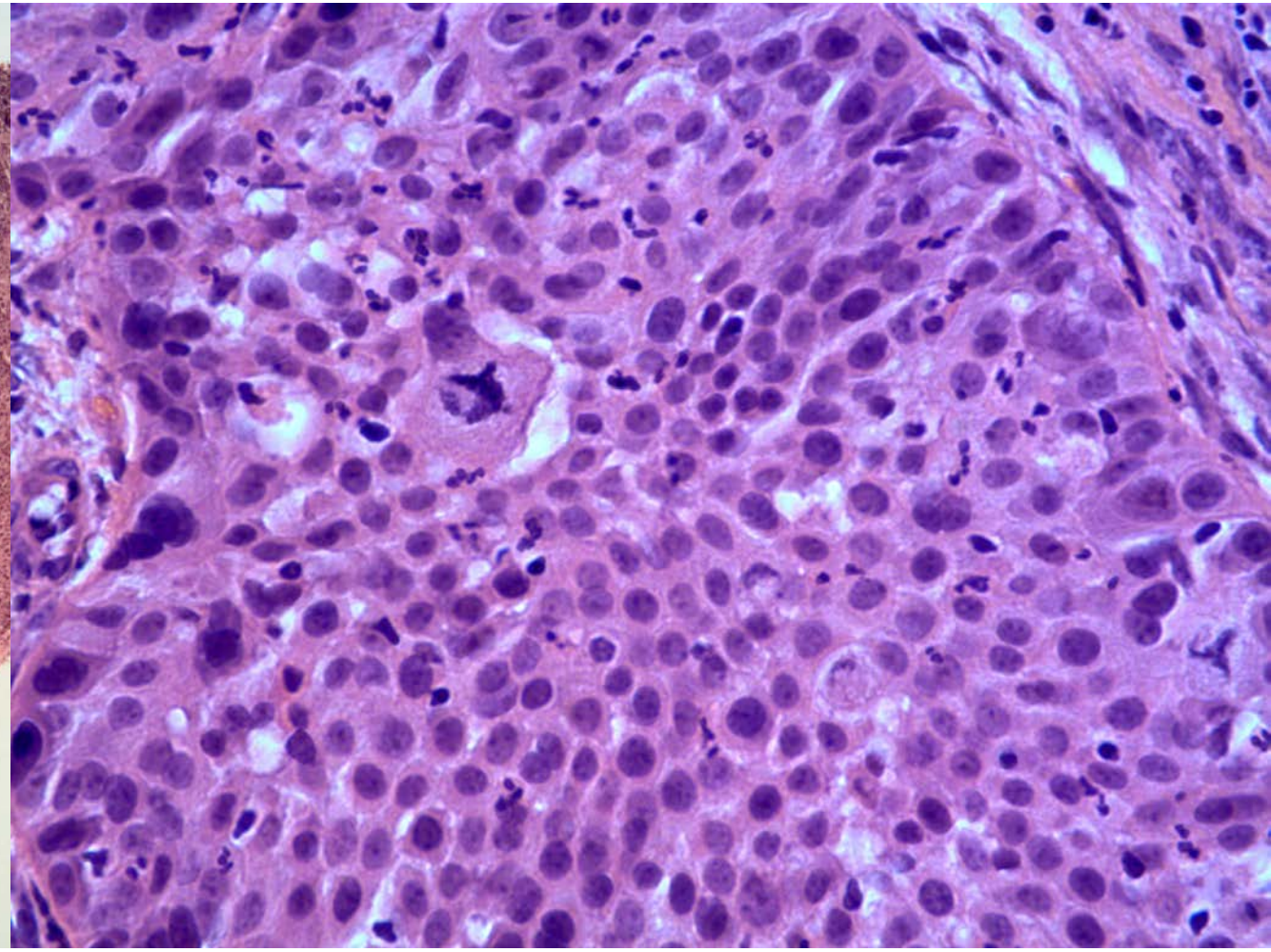






Squamous cell carcinoma HPV16/p16+

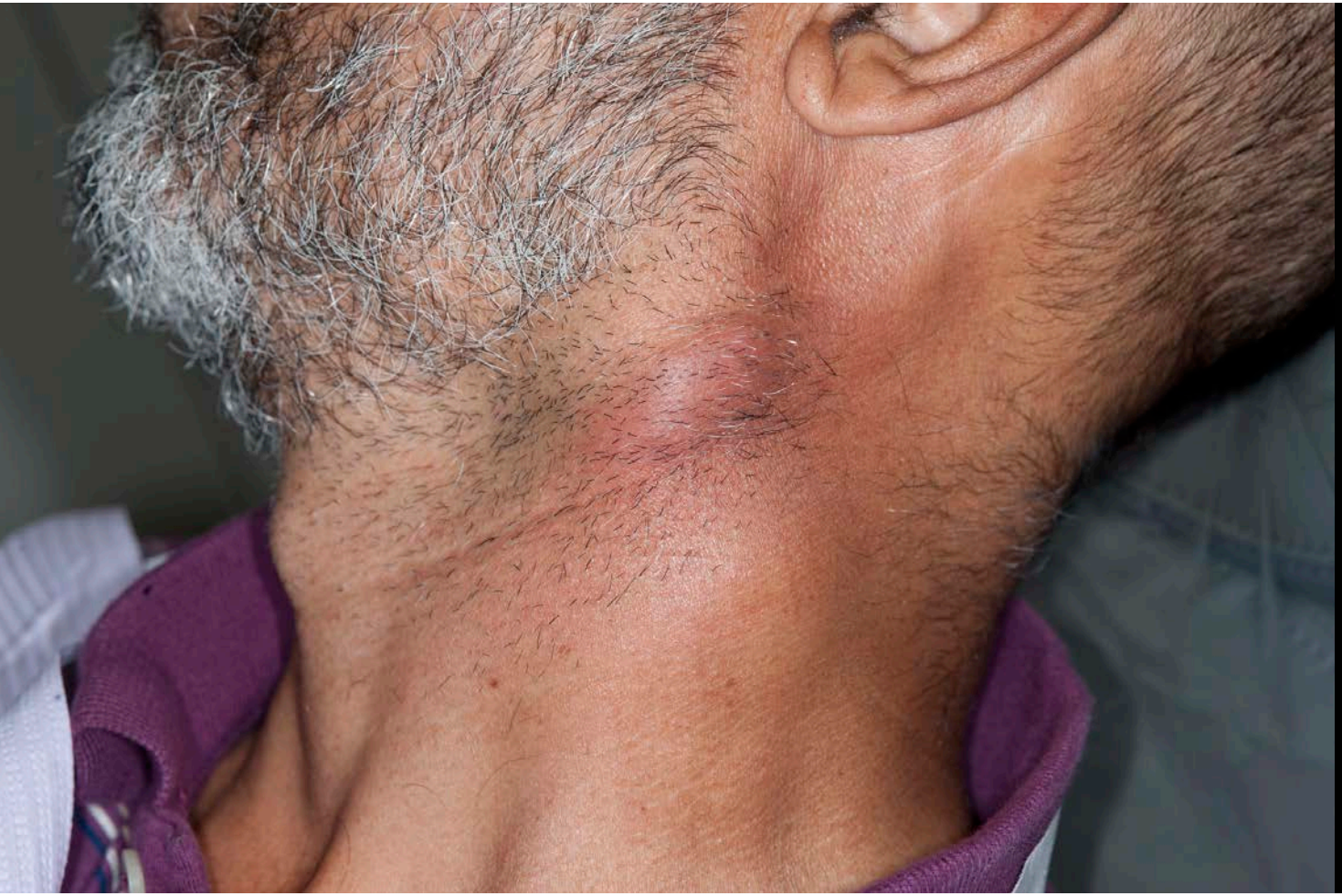


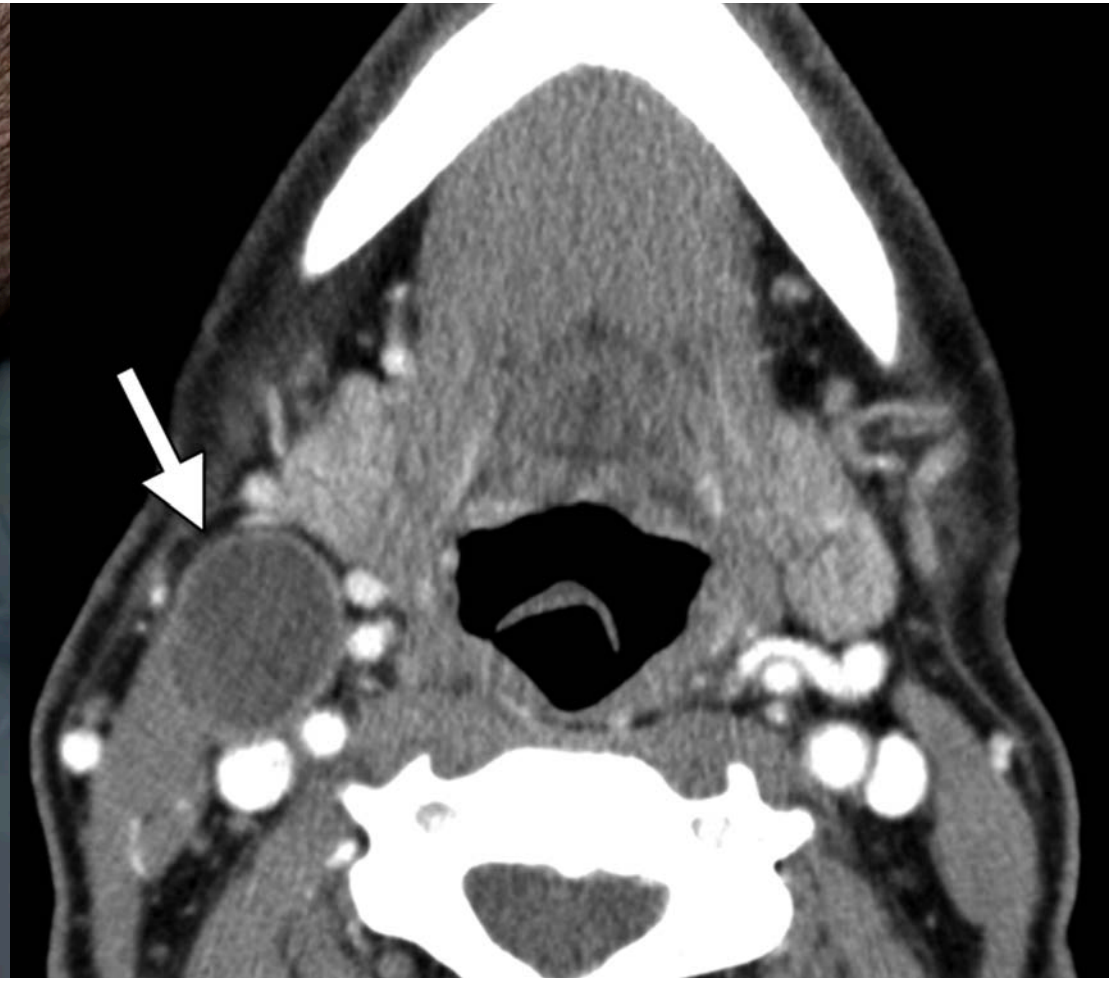


HPV-related Oropharyngeal SCC in HIV+ patients

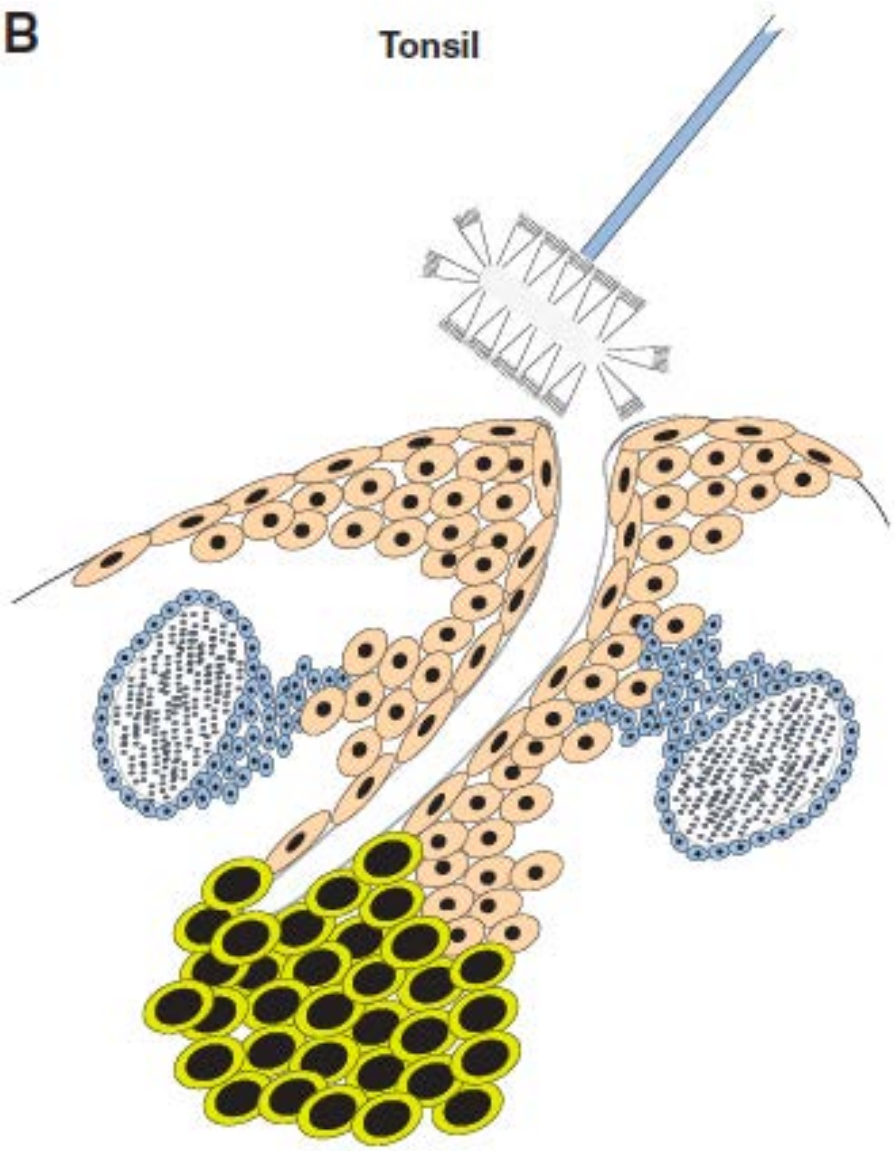
- Data from >85,000 patients pooled from 17 prospective cohort studies in North America (1996-2009)
- 3-fold higher among individuals with HIV vs general population
- Higher rates correlated to a trend of immunosuppression (CD4<200) prior to cancer diagnosis

Beachler DC, et al. Oral Oncol. 2014; 50(12): 1169–1176





cytopathology to detect oncogenic HPV subtypes in oropharynx?



Lingen MW. Cancer Prev Res 2011

HPV Vaccine: Now approved

- Gardisil 9 (2016)
- 6, 11. 16, 18, 31, 33, 45, 52, and 58
- HPV naïve males/females age 9-12, with "catch-up" up to age 26, and up to age 45 i selected patients
- HPV non-naïve HIV+ patients
  - Cervical cancer: Yes
  - Anal cancer: No
  - Oral/Ororpharynx: ??

Taking a sexual history in a dental setting?

Guidelines

INTERNATIONAL JOURNAL OF STD & AIDS

International Journal of STD & AIDS
0(0) 1–14

DOI: 10.1177/0956462413512807
std.sagepub.com
SAGE

2013 UK national guideline for consultations requiring sexual history taking

Clinical Effectiveness Group
British Association for Sexual Health and HIV

Gary Brook[1], Lesley Bacon[2], Ceri Evans[3], Hugo McClean[4], Colin Roberts[5], Craig Tipple[6], Andrew J Winter[7] and Ann K Sullivan[8]

….the content and detail of the sexual history will depend on the setting in which it takes place, the role of the clinical service and the needs of the individual patient.

Begin with less intrusive questions regarding presenting concerns, symptoms, or examination findings before asking more sensitive questions regarding sexual behavior.

Take home messages

- Oral HPV infection is prevalent in HIV+ patients
- Most infections are transient, but some remain persistent
- Most lesions are benign and few are at risk for malignant transformation.
- Florid papillomatosis remains a treatment challenge
- The percentage of HPV+ oral cavity squamous cell carcinomas is higher in the HIV+ population

Referrals

- NYU College of Dentistry Oral Mucosal Disease Clinic
- Kathy Gutierrez (212) 998-9743

Dr. Kerr: ark3@nyu.edu