

The Current Model of HPV Carcinogenesis



Division of Virology, Department of Pathology
University of Cambridge, UK



Men, Cancer & HPV

Webinar

11AM, 30th October 2025



John Doorbar
Department of Pathology,
University of Cambridge

NOMAN Is an Island: Race

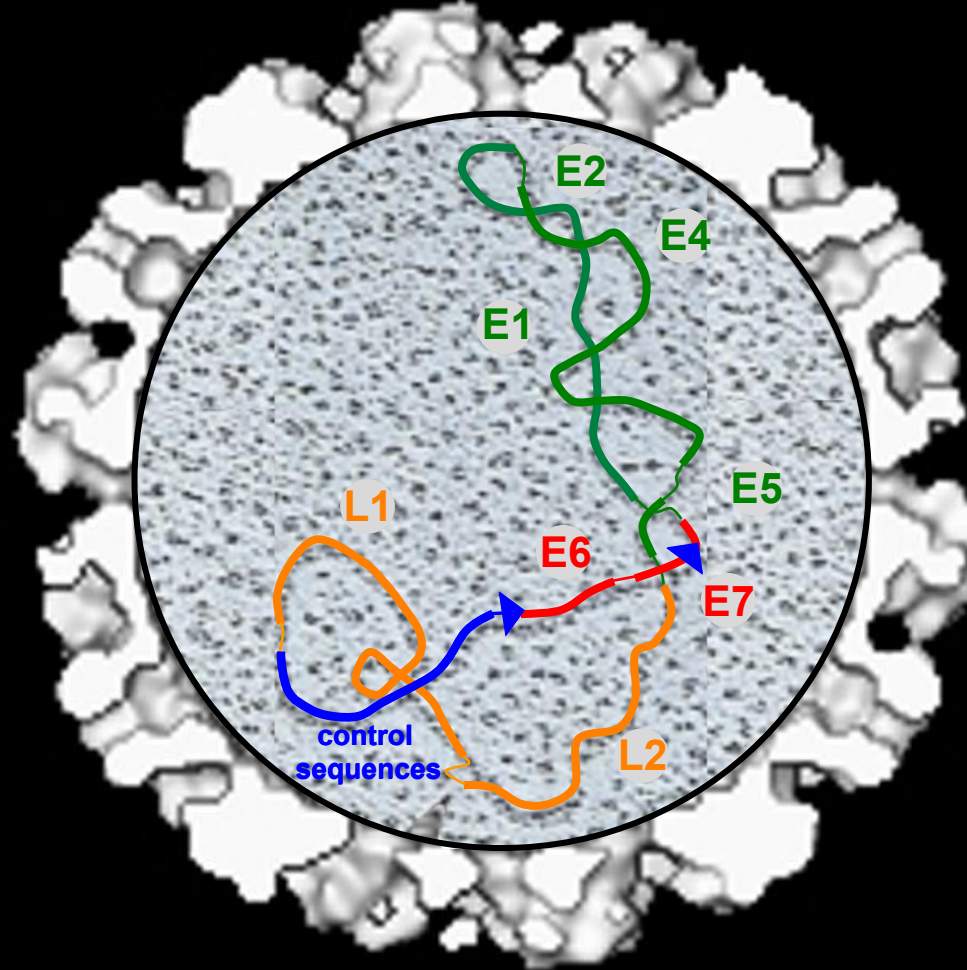


GLOBAL
MEN'S

GAMH is a UK-based charity. Register



viruses are
obligate
intracellular
parasites



Viral DNA

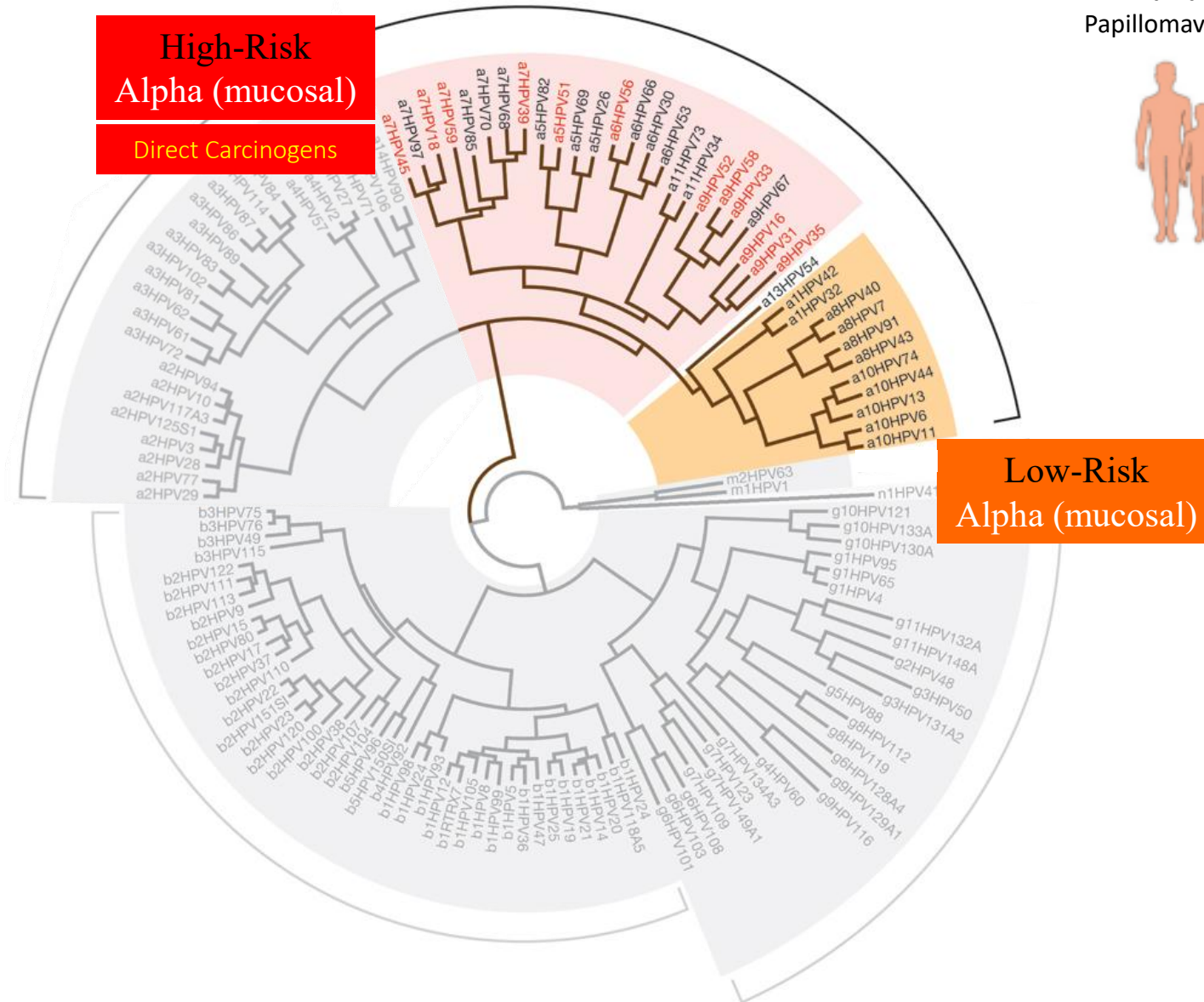
Disease Outcome depends on **SPECIFIC** viral **GENE FUNCTION**



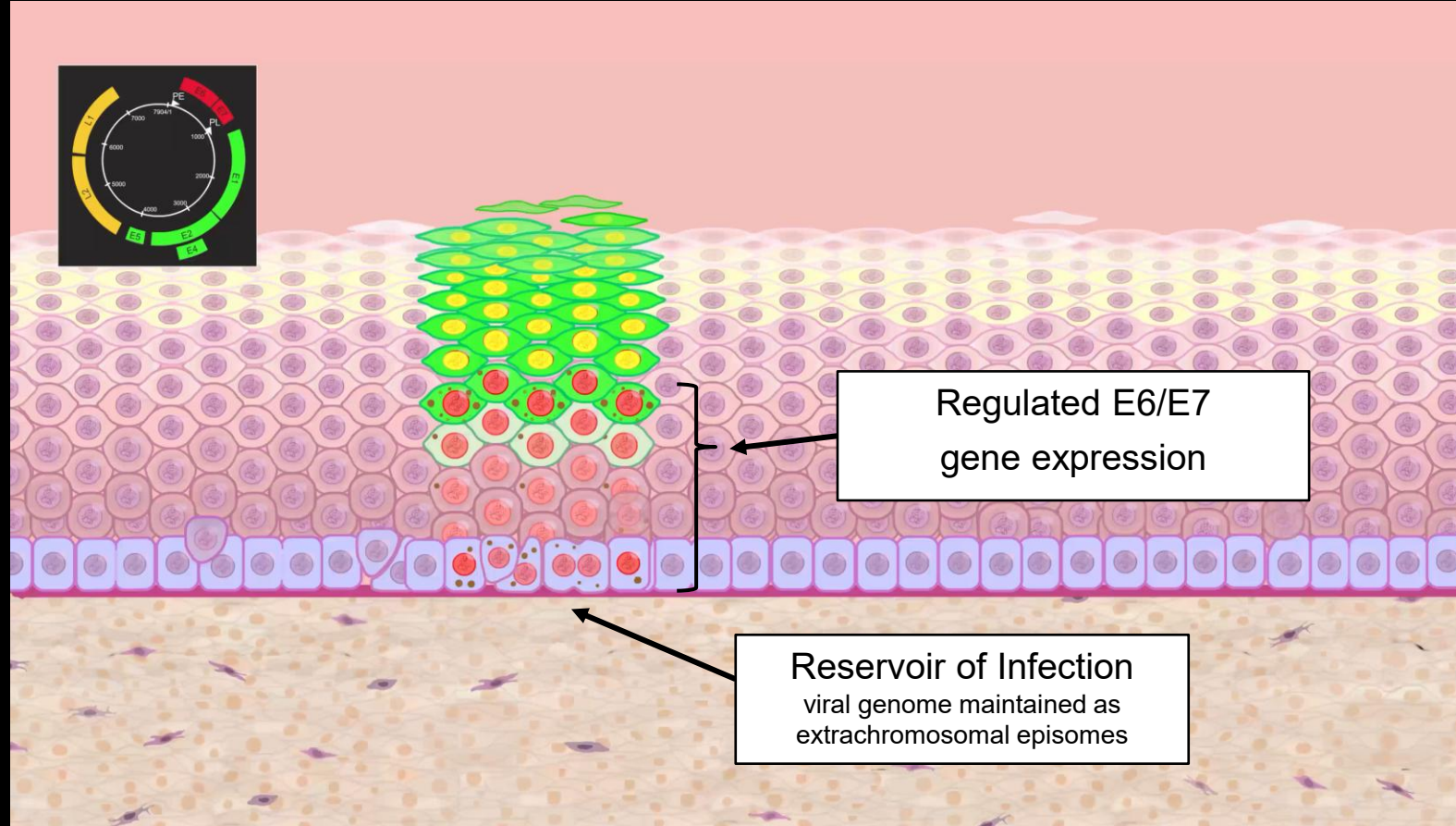
Human
Papillomaviruses



Principles

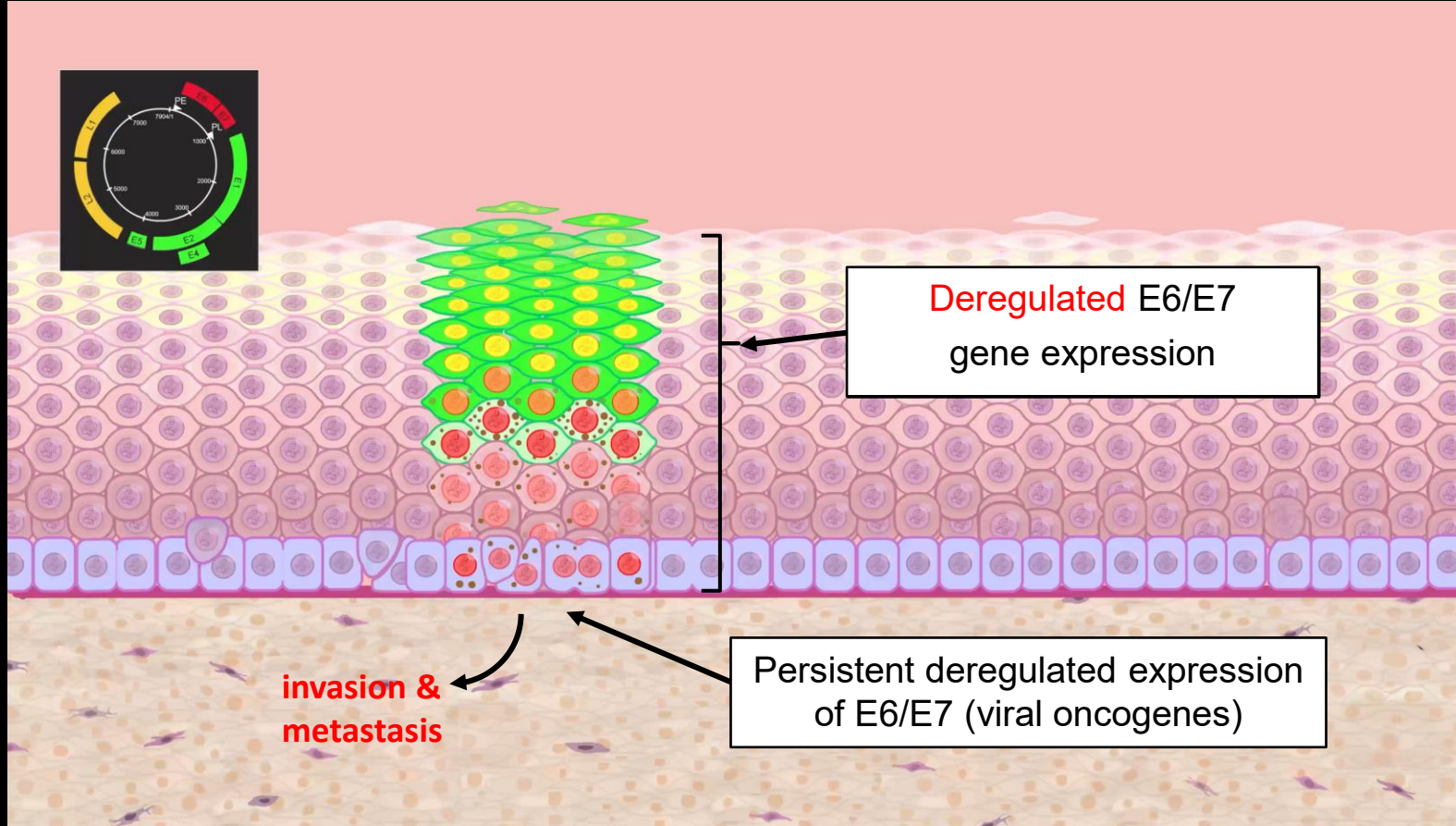


Papillomavirus types that infect humans



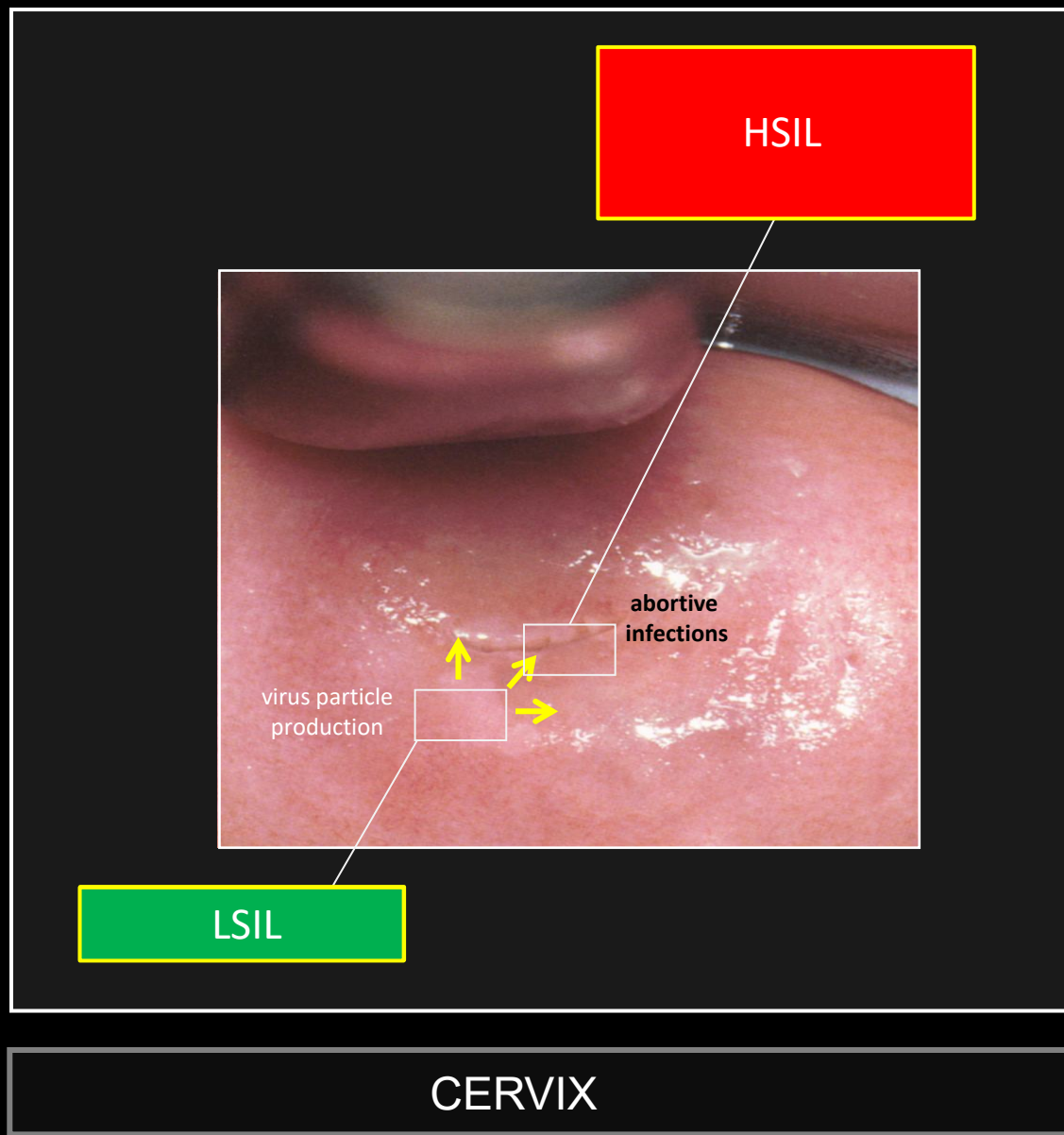
HPV E6/E7 RNA – viral gene expression





HPV E6/E7 RNA – viral gene expression





Conceptual Similarities in Carcinogenic Mechanisms

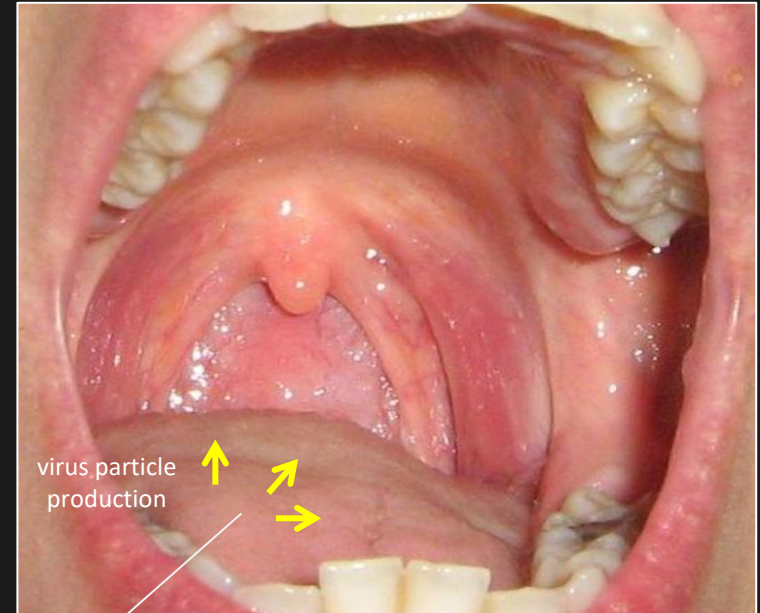


Disease is brought
under control by the
host immune system

virus particle
production

productive

CERVIX



virus particle
production

productive

OROPHARYNX

Cancer Progression is a rare outcome of a Common Infection



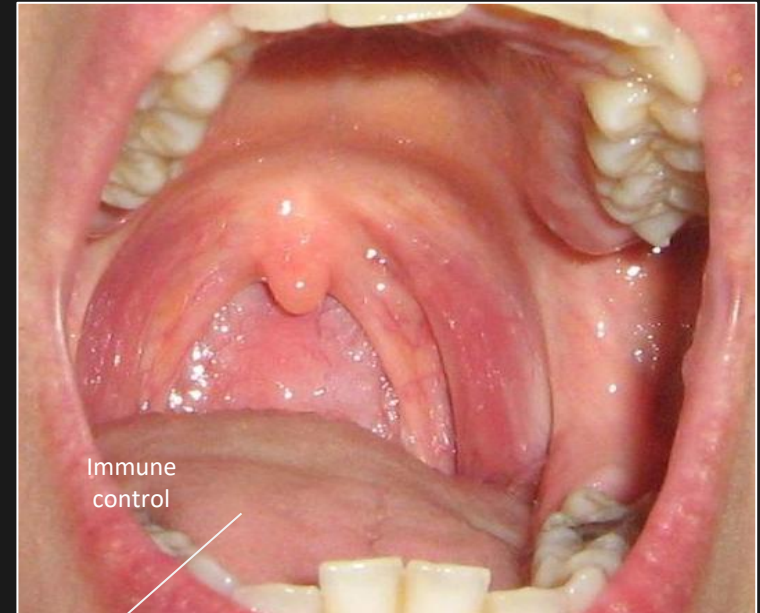
Disease is brought
under control by the
host immune system

Immune
control

Poorly or
non productive

subclinical

CERVIX



Immune
control

subclinical

OROPHARYNX

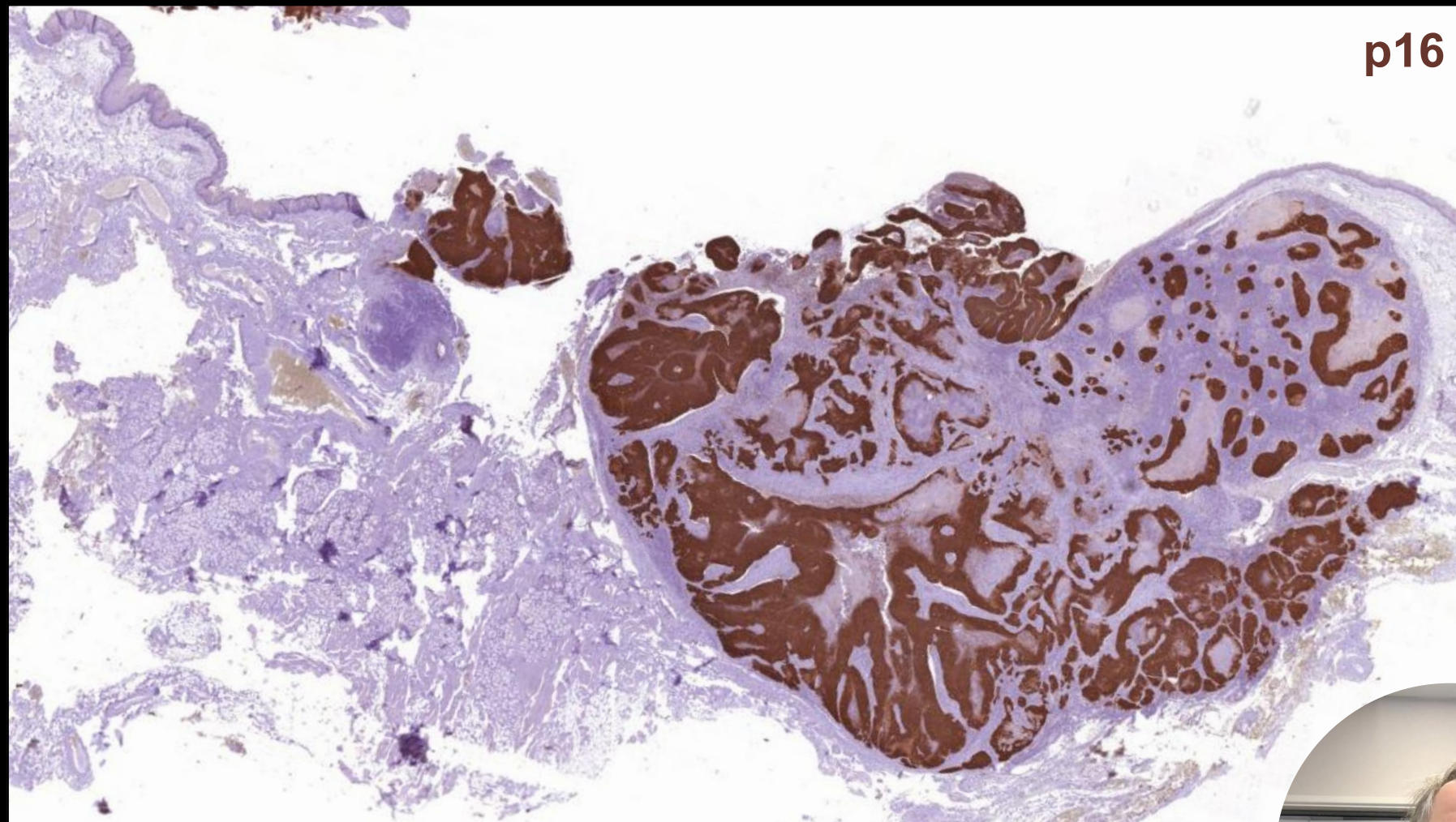
Disease Clearance – Control of Viral Gene Expression



Conceptual Similarities in Carcinogenic Mechanisms

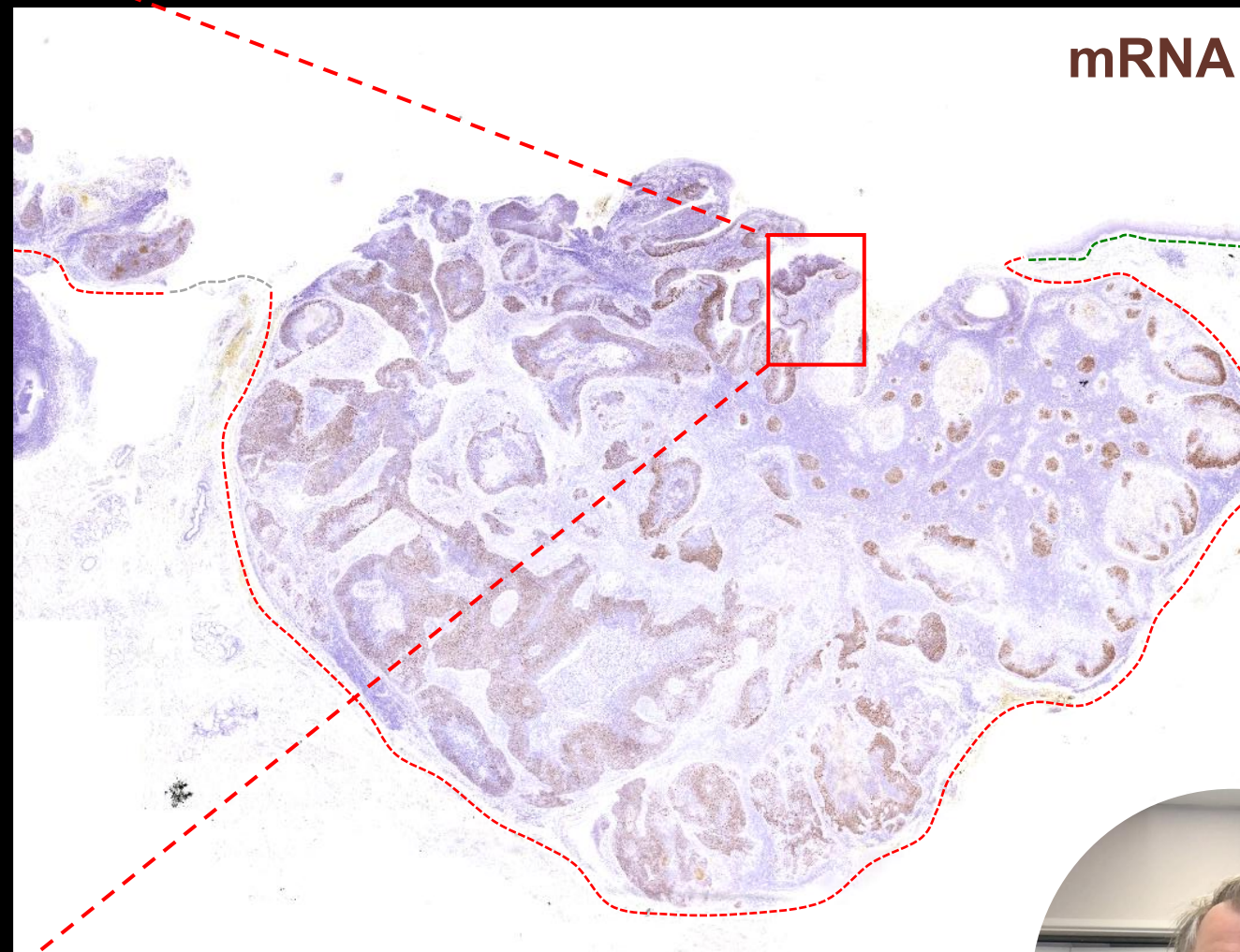
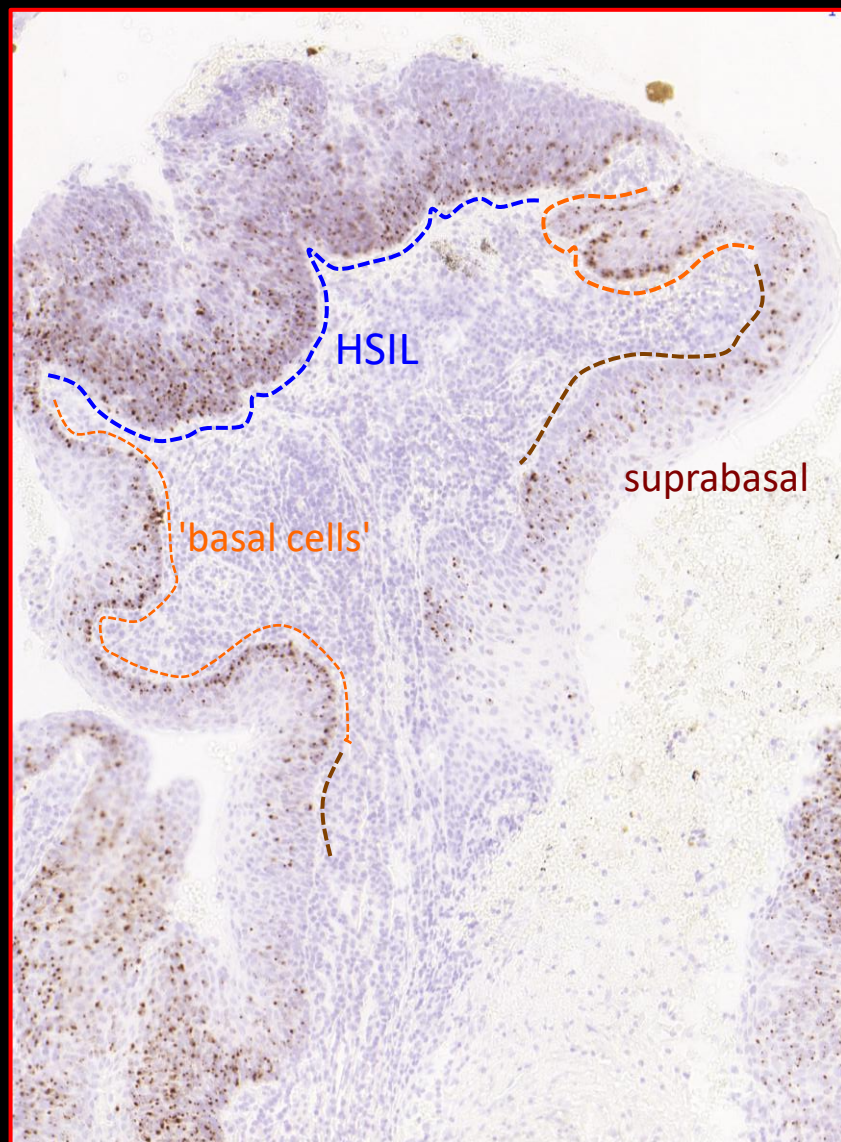
Oropharynx





Characteristics of Vulnerable Epithelial Sites

Heather Griffin, University of Cambridge & Martin J. Brenner, UCL



Characteristics of Vulnerable Epithelial Sites



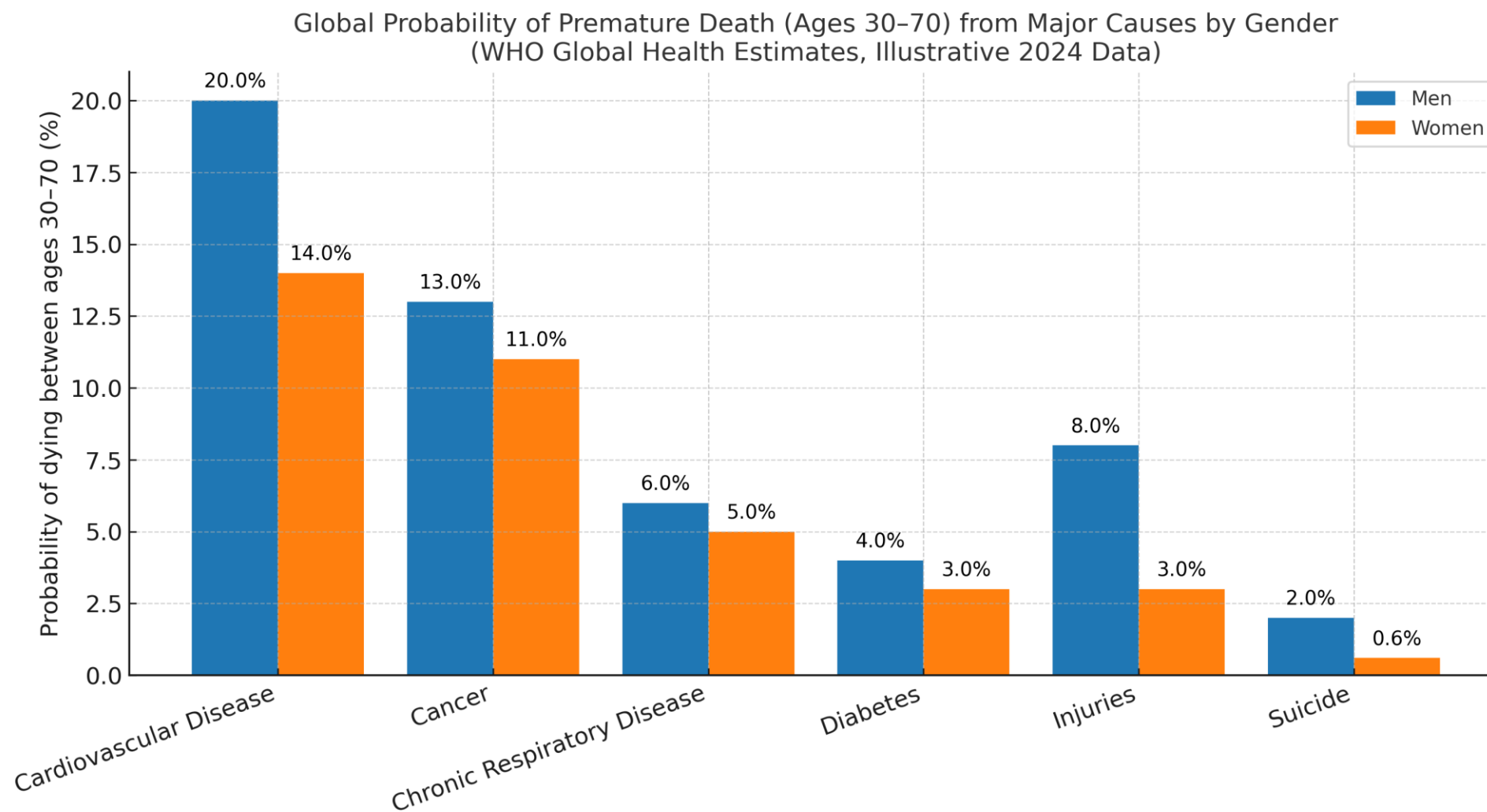


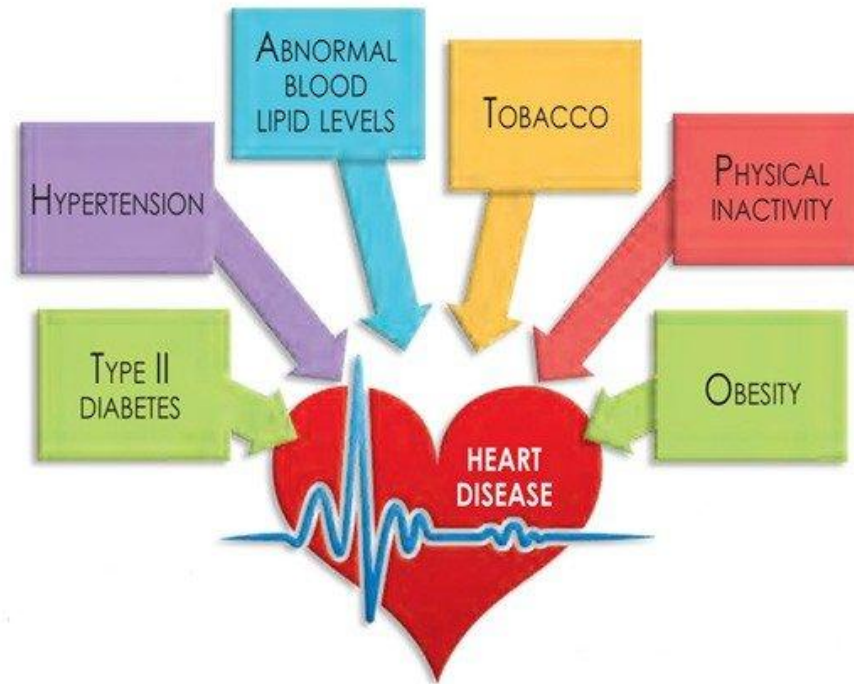
HPV AND HEART DISEASE: A MISSING LINK?



**GLOBAL ACTION ON
MEN'S HEALTH**

The global burden of NCDs





20% of CVD cases cannot be explained by conventional risk factors, e.g. tobacco, hypertension.

Could viruses, including HPV, be implicated?

VIRUSES AND HEART DISEASE

- A study of 10,000 people in the UK found the risk of major adverse cardiac events was increased among COVID-19 patients, particularly those requiring hospitalization.
- The risk among hospitalized COVID patients without known CVD was comparable to the risk of patients without COVID but with CVD, diabetes or peripheral artery disease.
- A link between influenza and CVD has been known for almost a century. The most common reported cardiovascular complications are cardiovascular death, myocardial infarction, and heart failure hospitalization.
- Influenza vaccination could reduce CVD mortality by 41%.

WHAT ABOUT HPV?

ACC.25

710
JACC April 1, 2025
Volume 85, Issue 12, Suppl A



Cardiovascular Disease Prevention

HUMAN PAPILLOMAVIRUS INFECTION AND CARDIOVASCULAR DISEASES: A META-ANALYSIS OF EFFECT SIZES FROM 249,366 PATIENTS

Poster Contributions
South Hall
Monday, March 31, 2025, Noon-1:00 p.m.

Session Title: Cardiovascular Disease Prevention 13
Abstract Category: 10. Cardiovascular Disease Prevention: Population Science
Presentation Number: 1259-34

Authors: Thaylla Vilela, Renan Yuji Ura Sudo, Stephan Akinfenwa, Bruna Silva, Celso Tiago Rodrigues, Ana Laura Silva, Marília Gobbo, Martha Staiger, Federal University of Grande Dourados, Dourados, Brazil, University of Connecticut, Farmington, CT, USA

ACC study pooled data from 7 studies:

- Almost 250,000 patients.
- Period of studies: 2011-2024.
- Age range of patients: 20-75 years.
- Follow-up period: 3-17 years.
- Countries covered: Australia, Brazil, South Korea, USA.
- Some studies adjusted for potential co-founding variables, e.g. smoking, diabetes.
- Analysis relied on observational data which prevents conclusions of causality.

Key findings:

- Overall, HPV+ patients had 40% higher likelihood of developing CVD and were 2x at risk of CAD than HPV- patients.
- When adjusted for variables such as socio-economic status, medical history, risk behaviours and family history of heart disease, patients had 33% higher risk of developing CVD.

“While we anticipated some degree of association between high-risk HPV (HR-HPV) and cardiovascular disease, the magnitude of the risk increase was striking. However, we found no significant association between HPV and hypertension, suggesting that the impact of HPV on cardiovascular health may be more specific to atherosclerosis-related diseases rather than overall blood pressure regulation. The potential mechanistic link between chronic viral infections and vascular inflammation deserves further research.”

- Lead author, Dr Stephen Akinfenwa, University of Connecticut School of Medicine.

[News from the lab](#)[HPV](#)[Research summary](#)

HPV increases risk of heart disease, finds study of 250,000 patients

New meta-analysis suggests that the HPV vaccine could not only protect us from cervical cancer, but heart problems too.

4 April 2025 • 4 min read • by [Priya Joi](#)

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CAN HPV VACCINATION PREVENT CVD?

THE AMERICAN JOURNAL
of MEDICINE
Official Journal of the Alliance for Academic Internal Medicine

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CLINICAL RESEARCH STUDY · Volume 136, Issue 3, P294-301.E2, March 2023

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The Effects of Human Papillomavirus Infection and Vaccination on Cardiovascular Diseases, NHANES 2003-2016

[Xiaopeng Liang, MD](#) ^{a,1} · [Oscar Hou In Chou, MSc](#) ^{a,1} · [Bernard M.Y. Cheung, MB, BChir, PhD](#) ^{a,b,c}  

[Affiliations & Notes](#)  [Article Info](#) 



- Investigated approx 9,350 US women aged 20-59 who were tested for vaginal HPV DNA.
- HPV infection associated with a 66% greater risk of CVD.
- HPV vaccinated women were at no greater risk of CVD.

CONCLUSIONS

- Several viruses, including COVID, influenza and HPV, have been linked to cardiovascular disease.
- HPV+ patients may have a 40% higher likelihood of developing CVD and 2x at risk of CAD than HPV- patients.
- HPV vaccination could remove this increased risk and potentially help to reduce the global burden of CVD.
- Further research is needed, including longitudinal studies of vaccinated populations.

SOURCES

- Chan NC, et al. Human papilloma virus and atherosclerotic cardiovascular disease. *European Heart Journal* 2024; 45:1083-1085. doi: 10.1093/eurheartj/ehad829.
- Dutta P, et al. Unveiling HPV's hidden link: Cardiovascular diseases and the viral intrigue. *Indian Heart Journal* 2024; 76(1):1-5. doi: 10.1016/j.ihj.2024.02.001.
- Vilela T, et al. Human Papillomavirus infection and cardiovascular diseases: a meta-analysis of effect sizes from 249,366 patients. *JACC* 2025; 85(1):Supp A. doi: 10.1016/S0735-1097%2825%2901194-5.



GLOBAL ACTION ON MEN'S HEALTH

THANK YOU!

β -HPV and skin cancer: an emerging picture

Professor Sarah Allinson

Biomedical and Life Sciences, Lancaster University

Non-melanoma skin cancer

GLOBALLY EACH YEAR:



1.3M
CASES
64,000
ANNUAL
DEATHS



2-3X
MORE
COMMON
IN MEN*

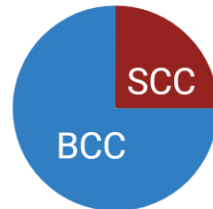
*Source: American Cancer Society



CAUSED BY
THE SUN

TWO MAIN TYPES:

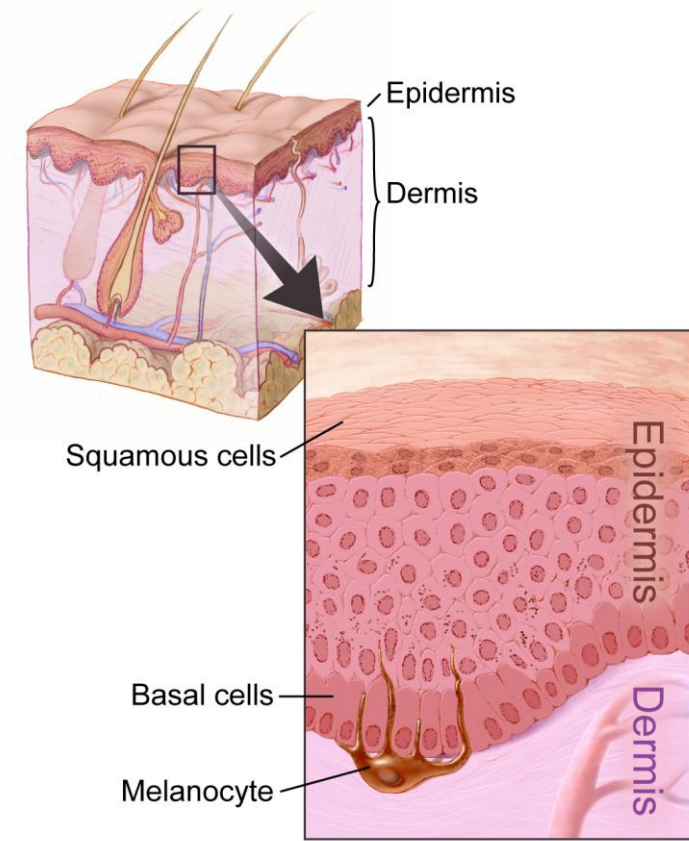
BASAL CELL CARCINOMA
SQUAMOUS CELL CARCINOMA



CASES

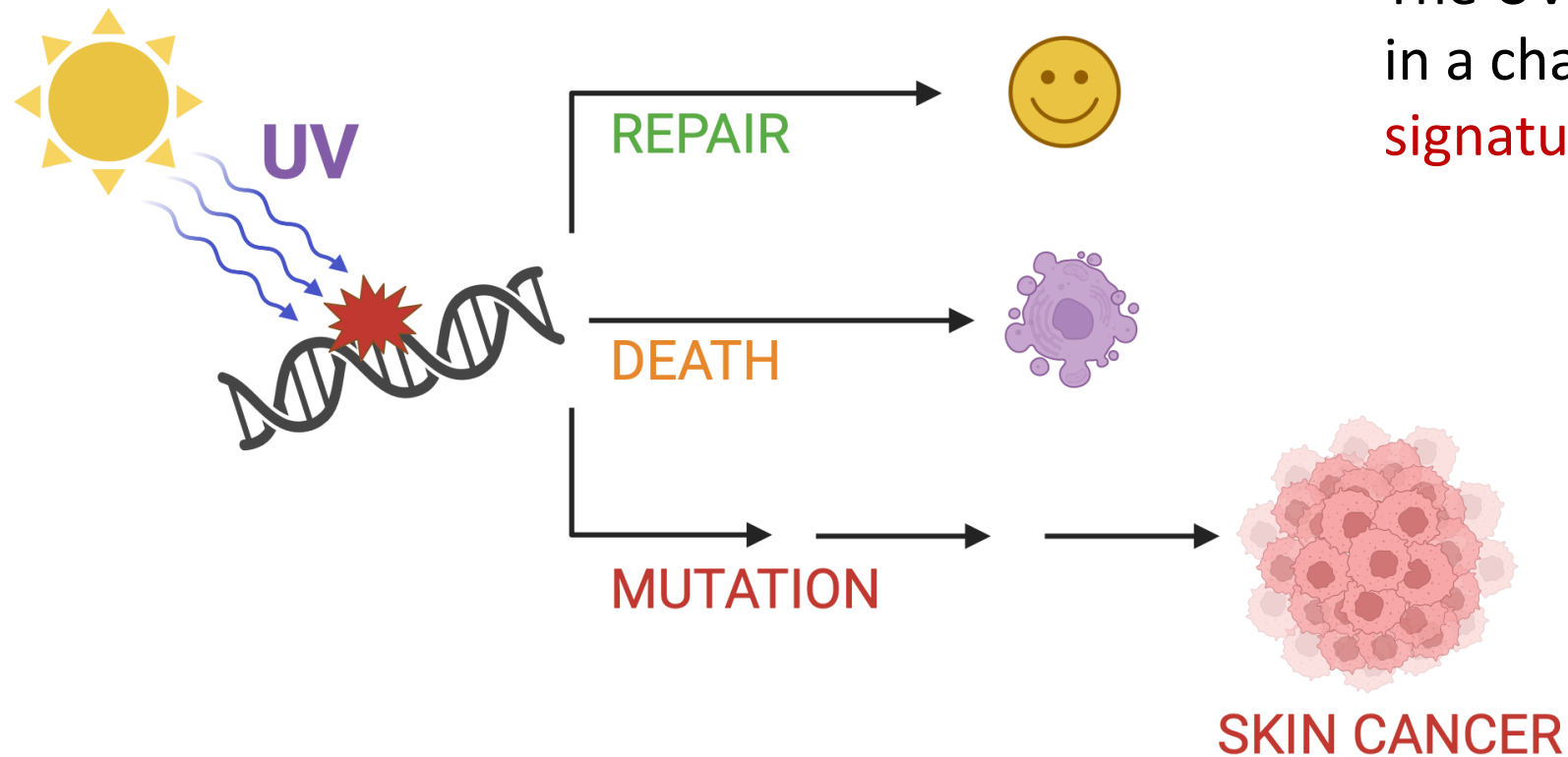


DEATHS



National Cancer Institute

UV and skin cancer



The UV damage to DNA results in a characteristic **mutation signature** seen in tumours

HPV types

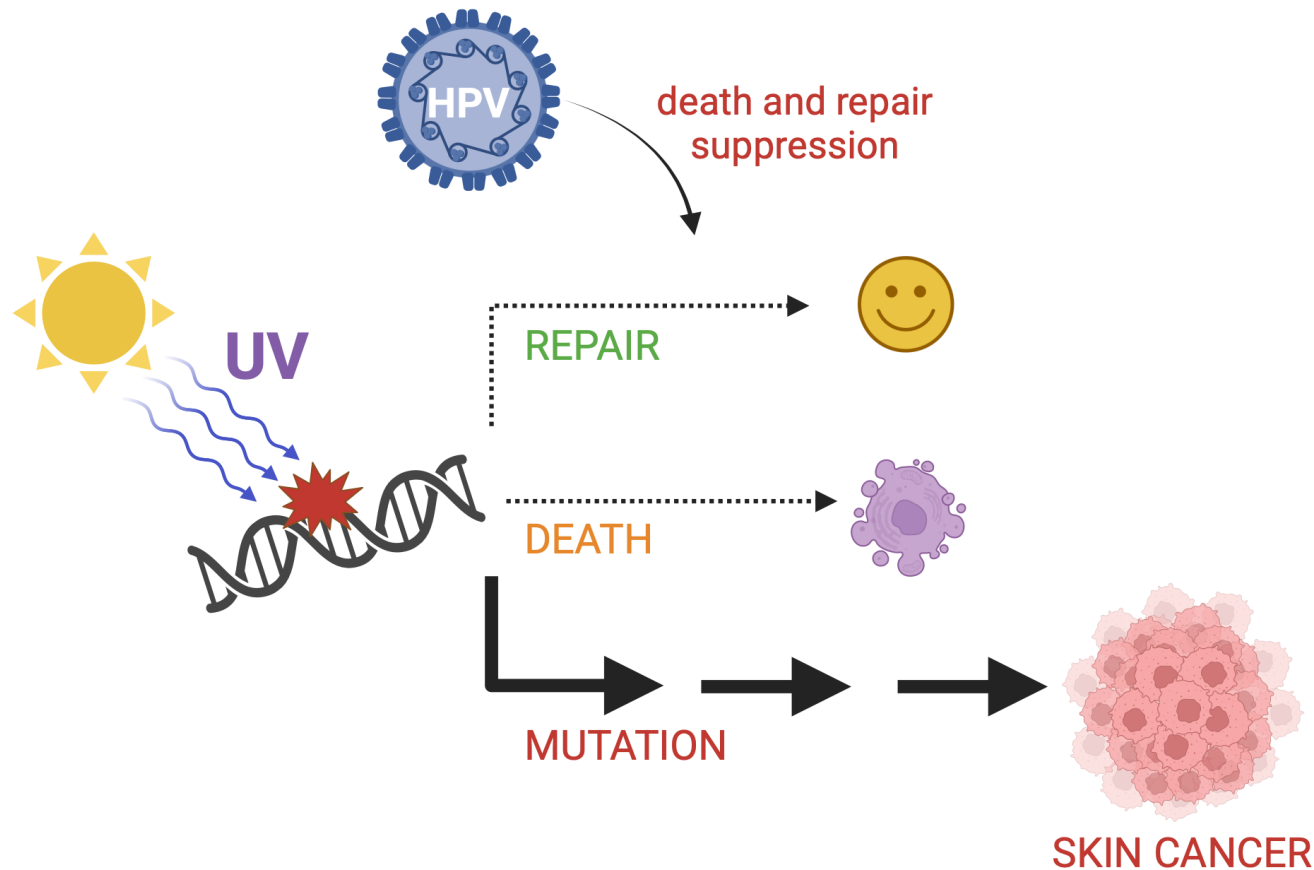
- There are **>200 different HPV subtypes** that infect humans
- Can be evolutionarily grouped into 5 types:

HPV types	Infection site	High risk subtypes?	Vaccine?
Alpha (α)	mucosal, cutaneous	Y	Y
Beta (β)	cutaneous	N	N
Gamma (γ)	cutaneous	N	N
Mu (μ)	cutaneous	N	N
Nu (ν)	cutaneous	N	N

β –HPV infection and skin cancer

- Commensal infections – often acquired at birth, **usually asymptomatic**
 - More immunogenic than high risk HPVs
- Linked to **cSCC in various conditions**:
 - Epidermodysplasia verruciformis (EV)
 - Severe combined immunodeficiency (SCID)
 - Organ transplant patients etc
- EV patients develop multiple warts
 - Can develop into cSCC in sun-exposed areas
 - 90% positive for HPV5/HPV8

So, what's going on?



“Hit and run” model

- β –HPV infection helps mutagenesis but **doesn't drive cancer growth itself**
- Skin tumours show typical UV signature
- HPV detected in AK (cancer precursor) but less in cSCC
- Mouse models show HPV not required for sustained tumour growth

Hit and run or in the driving seat?

- Recent case report* of a 34-year-old woman with multiple warts and **>40 HPV19 positive cSCC tumours** – did not respond to treatment
- Genetic profiling of woman revealed she had an inherited immunosuppressive condition (SCID)
- Investigation of tumours showed **no characteristic UV signature**
- Instead **HPV19 had become integrated into genome of cancer cells**
- A **stem cell transplant** resulted in full recovery – resolution of warts and cSCC
 - Restoration of T cell function allowed all HPV19-infected cells to be recognised and eliminated

*Ye et al (2025) *N Engl J Med* 393:469-478

Could β -HPV be cancer protective in immune competent people?

- A study reported in early 2025* suggested that **skin cells that have acquired mutations** that could lead to cancer have increased **β -HPV activity**
- This in turn **flags the infected cells to the immune system** limiting their expansion
- This could prevent cancer occurring in the first place, but a lot more work is required (several gaps in knowledge remaining)

* Son et al (2005) *Cancer Cell* 43:36-48

Future prospects

- There is an increasingly clear link between β -HPV and skin cancer **in immunocompromised patients**
- Virus usually causes cancer by “**hit and run**”, but at least one example where **cancer growth is driven by virus**, analogous to high-risk HPVs
- Less information about what happens when immune system is fully functional – more research needed!!
- β -HPV vaccines are currently in development (using a similar idea to alpha HPV vaccines)

HPV and Prostate Cancer?

Professor Daniel Kelly

European Cancer Organisation, Co-Chair, HPV/ HBV Action Network

Emeritus Professor in Healthcare Sciences, Cardiff University

kellydm@Cardiff.ac.uk

HPV & Prostate (and other) cancers

Prostate cancer is now the most common solid organ cancer in men.

Modification of risk factors is challenging (genetics, infection, testosterone level, ethnicity) **Q. Is HPV infection implicated?**

Known link with oropharyngeal cancer (now mainly a disease of men) and anogenital cancers

Cervical cancer is on a pathway to elimination in several countries

Is there evidence of a causal relationship between HPV exposure and prostate cancer?

HPV link is possible, but evidence is not definitive

HPV is found in in benign as well as cancer samples

Only one of several pathogens in the gland, so could be coincidental finding, or a causatory factor

Globally there may be a variation in HPV sub-types, leading to different conclusions in large scale studies

May be a role in promoting changes in early stages, at the level of oncogenic impact, rather than later stages

Prostate cancer is not linked to immunocompromised states, unlike cervical cancer (also smoking is not examined)

PERSPECTIVE **OPEN**



Prostate cancer and the human papilloma virus: causative association, role of vaccines, and the impact of the COVID-19 pandemic

Naomi Morka ¹✉, Joseph M. Norris^{2,3}, Mark Emberton ^{2,3} and Daniel Kelly ⁴

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Prostate cancer affects a significant proportion of men worldwide. Evidence from genetic and clinical studies suggests that there may be a causal association between prostate cancer and the human papilloma virus (HPV). As HPV is a vaccine-preventable pathogen, the possibility of a role in prostate cancer causation may reinforce the importance of effective HPV vaccination campaigns. This is of particular relevance in light of the COVID-19 pandemic, which may have considerable effects on HPV vaccine uptake and distribution.

Prostate Cancer and Prostatic Diseases (2022) 25:55–57; <https://doi.org/10.1038/s41391-021-00404-6>

Recent Systematic Reviews

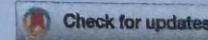
N. Morka et al.

56

Table 1. Recent systematic reviews and meta-analyses examining the association between prostate cancer and HPV.

Review author, Ref.	No. included studies	Key findings
Russo et al. [2]	30	The odds of developing prostate cancer were increased in men positive for HPV-16 (pooled OR = 1.37, $p < 0.01$), but not in HPV-18 positive men.
Lawson et al. [4]	26	High-risk HPVs were identified in 22.6% of prostate cancers, compared to 8.6% of benign or normal prostate tissues ($p = 0.001$).
Yin et al. [5]	24	In HPV-positive men, the pooled OR for developing prostate cancer was 2.27 (95% CI = 1.40–3.69).
Moghoofei et al. [6]	24	There was a positive association between HPV and prostate cancer (OR = 1.281, $p = 0.026$) and a higher incidence of HPV-16 in prostate tumours.

No. Number, HPV human papilloma virus, OR odds ratio, p probability, CI confidence interval.



OPEN Detection of high-risk Human Papillomavirus in prostate cancer from a UK based population

M. Yahya Ahmed^{1,4}, Nadia Aziz Salman^{1,4}, Sarbjinder Sandhu², M. Okan Cakir¹, Alan M. Seddon¹, Christian Kuehne³ & G. Hossein Ashrafi^{1✉}

Human papillomavirus (HPV) infection is one of the sexually transmitted diseases which have been implicated in the etiology of multiple cancers. To date, several studies have been conducted to evaluate the incidence of high-risk (HR) HPV in prostate cancer (PCa) which have generated widely conflicting data. Hence, this leaves a lack of awareness on the causal role of persistent HPV infection in the development of PCa. Although this has been investigated in a handful of countries, to the best of our knowledge, no prior studies have been conducted in the UK. In this study, polymerase chain reaction (PCR) and Sanger sequencing were implemented to analyze a total of 49 fresh prostate specimens (35 benign and 14 malignant specimens) for the presence of viral DNA of 12 HR-HPV types. Data obtained confirmed the presence of HR-HPV in 32.7% of analyzed benign and malignant prostate tissues with HPV 35 being identified as the most frequent type. Moreover, HR-HPV positivity rate was found to be higher in abnormal prostate tissues (adenocarcinoma and benign with prostatitis) compared those with normal prostate condition. Using immunohistochemistry, we have confirmed the expression of HPV E7 protein in prostate tissues positive for HPV DNA. This observation, the first reported from a UK population, suggests that the presence of HPV in prostate tissue is likely to be a related factor in the progression of certain cases of prostate cancer.

Investigated presence of HPV DNA in benign and malignant samples

Presence of HR HPV in 32.7 samples of benign & malignant samples (HPV 35).

HR HPV higher in adenocarcinoma & prostatitis

HPV DNA may be evidence of promotion of cellular change in some cases of prostate cancer.

What to conclude?

Evidence is not yet conclusive and is sometimes contradictory.

HPV may initiate or promote oncogenesis via mutations in genes that usually protect against viruses & is a current concern

HPV is found in about quarter to one third of PC samples- different HPV types seem to be associated with different risk levels (eg HPV 16)

HPV exposure needs to be explored alongside other accepted risk factors for prostate cancer

Current anti-vax rhetoric does not protect young men who may be at risk

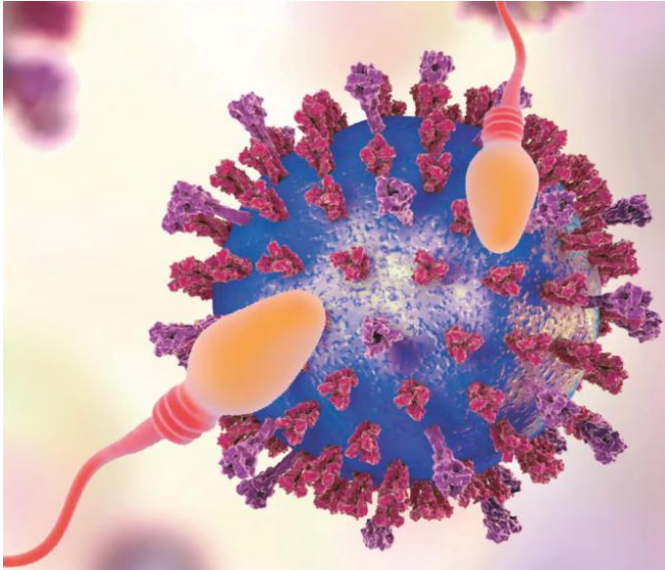
Needs further research, but supports the case for Gender Neutral Vaccination until more evidence is obtained & especially when other cancers in men are considered (especially OP)

References

Bennett C, Edwards D, Sherman SM, *et al* Which interventions improve HPV vaccination uptake and intention in children, adolescents and young adults? An umbrella review. *Sexually Transmitted Infections* 2022; 98: 599-607.

Morka N, Norris J, Emberton M, Kelly D. *Prostate Cancer & Prostatic Diseases* 2022 25; 55-57

Yaha Ahmed M *et al* Detection of high-risk Human Papilloma virus in prostate cancer from a UK based population. *Scientific Reports* 2023; 13: 7633

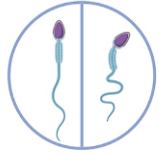


HPV in the Male Genital Tract: Key Effects on Fertility, Inflammation and Coinfections

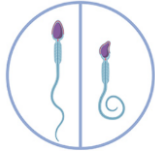
Prof. Carolina Olivera

Impact of HPV infection on male reproductive health

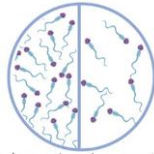
Main alterations in semen attributed to HPV



Sperm motility



Sperm morphology



Sperm concentration

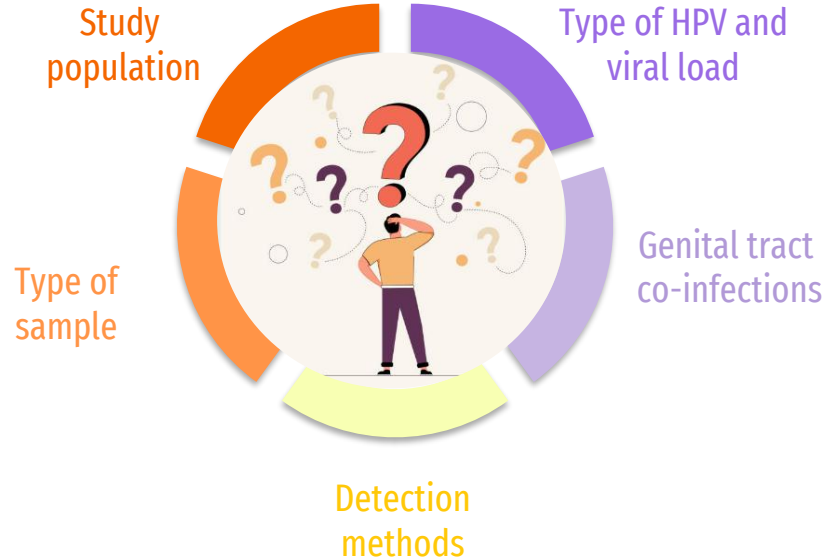


Failures in IVF



Sperm DNA fragmentation

Key factors causing differences in findings on how HPV impacts men's health

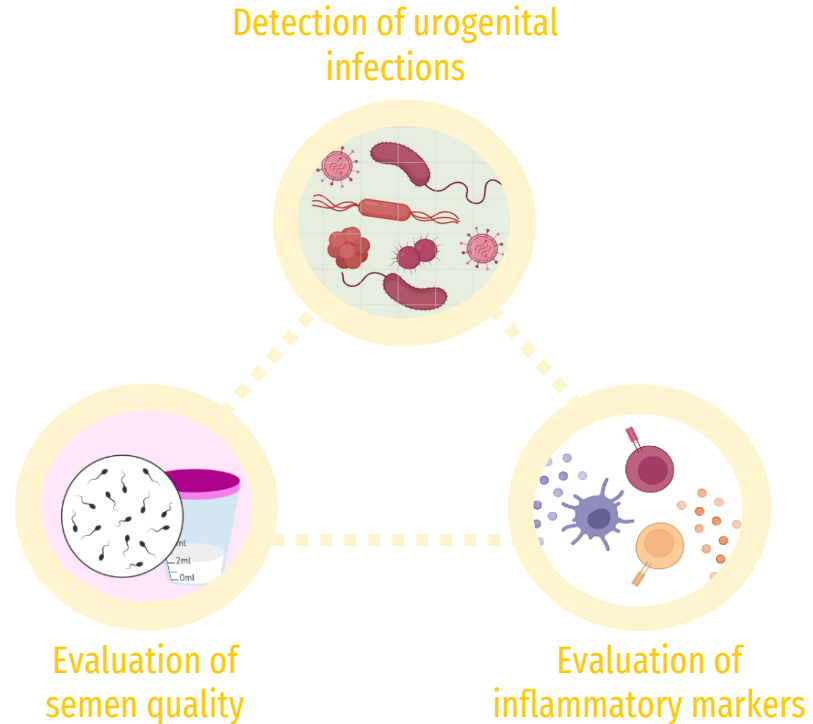


Prospective study of sexually adult young adult men attending a urology/fertility clinic

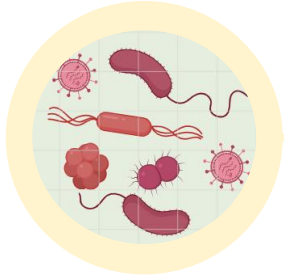


205 male patients

- From Córdoba, Argentina.
- Age range 20-61 years
- Not vaccinated for HPV
- No antibiotics at the time of analysis



Detection of urogenital infections



Spermculture

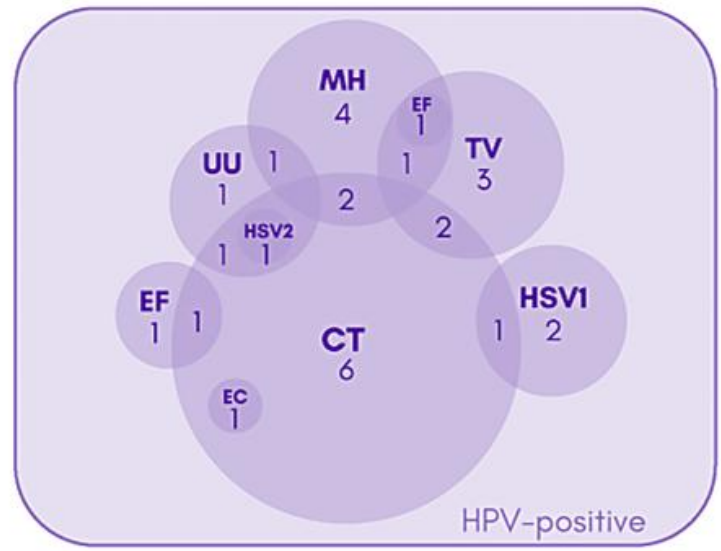
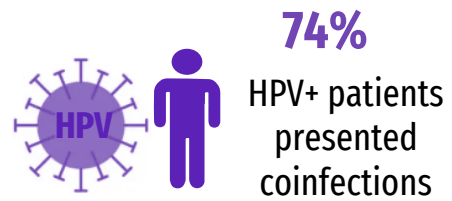
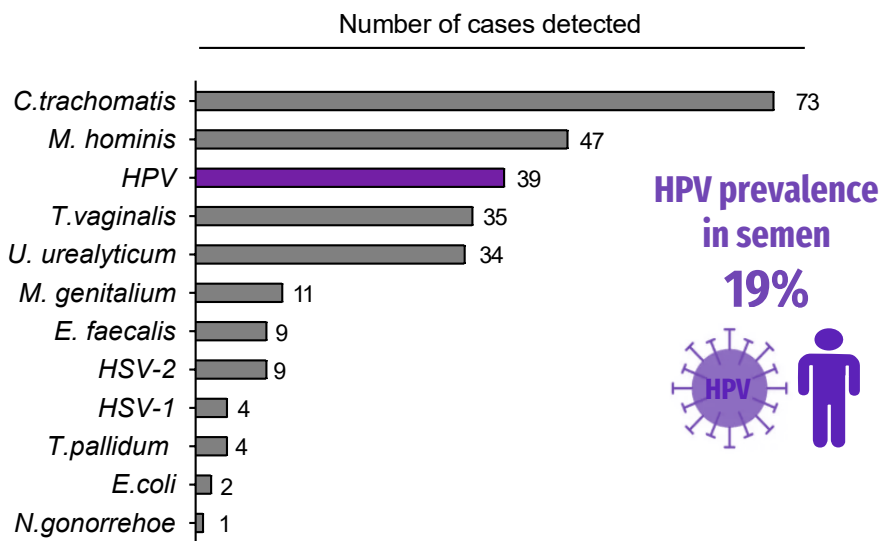
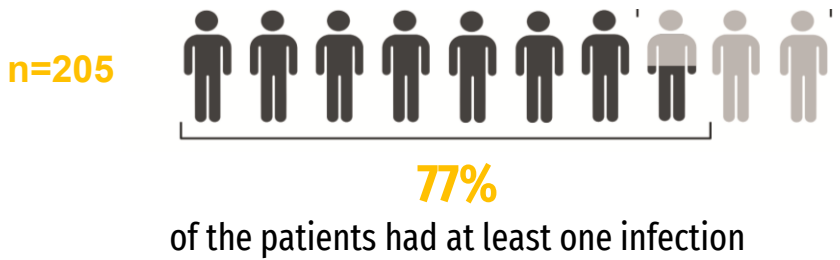
- *E. coli*
- *E. faecalis*
- *P. mirabilis*
- *Enterobacteriaceae*
- *Pseudomonas spp.*
- *Streptococcus spp.*
- *Staphylococcus spp.*
- *Corynebacteriaceae*
- *Candida spp*



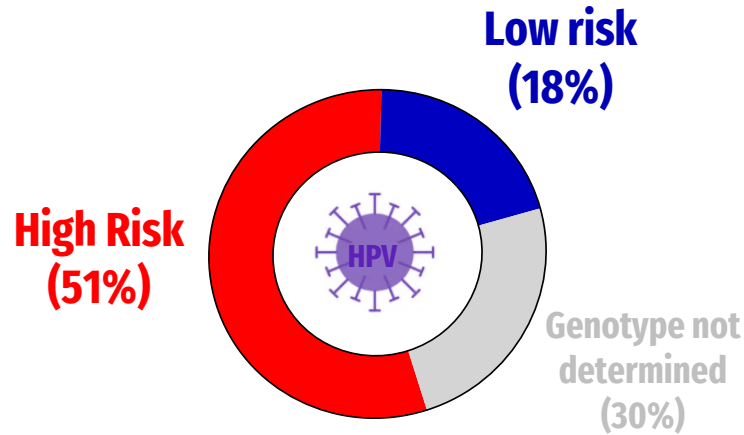
PCR

- **HPV**
- HSV-1
- HSV-2
- *M. hominis*
- *U. urealyticum*
- *C. trachomatis*
- *M. genitalium*
- *T. vaginallis*
- *N. gonorrhoeae*
- *T. pallidum*

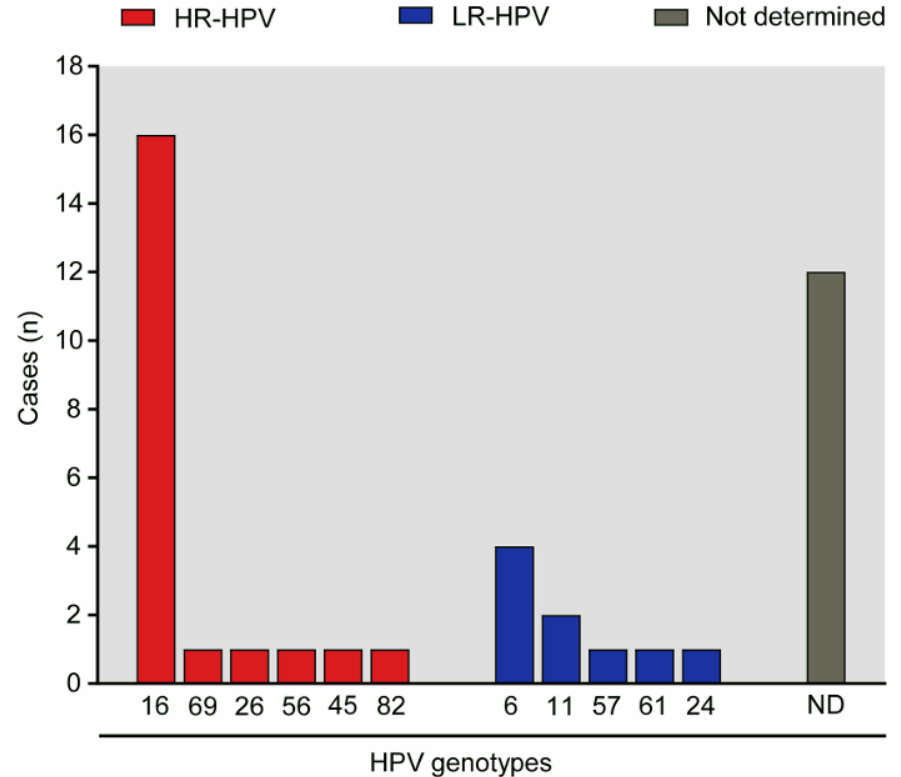
Characterization of the urogenital infection by HPV and other common uropathogens



Type-specific HPV prevalence and genotypic characterization



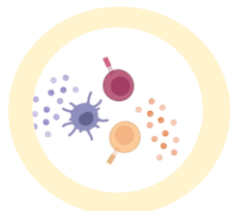
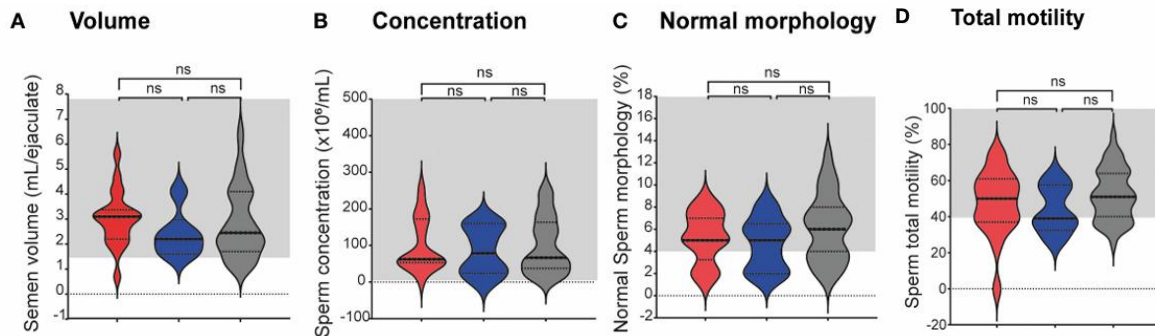
The half of the HPV+ patients had at least one **high risk genotype**. HPV-16, a high oncogenic risk genotype, was the most frequently detected.



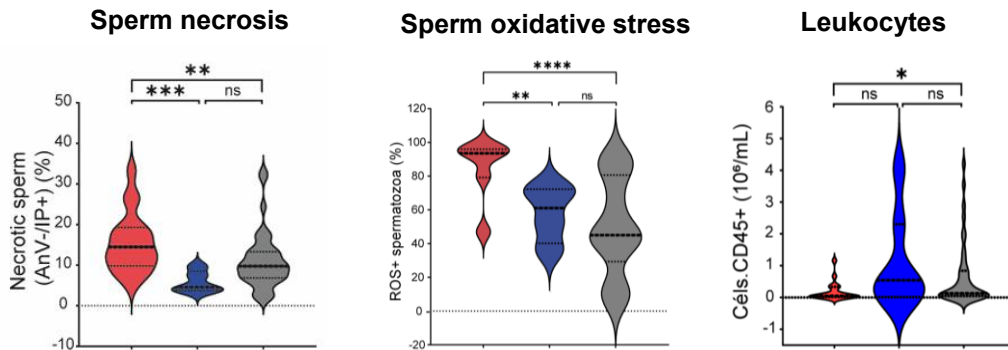
Effects of high- and low-risk HPV on semen quality



Routine
spermogram



Semen quality by
flow cytometry

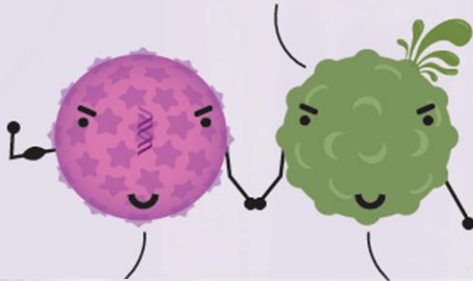


Impact of HPV and Chlamydia trachomatis (CT) coinfection on sperm quality

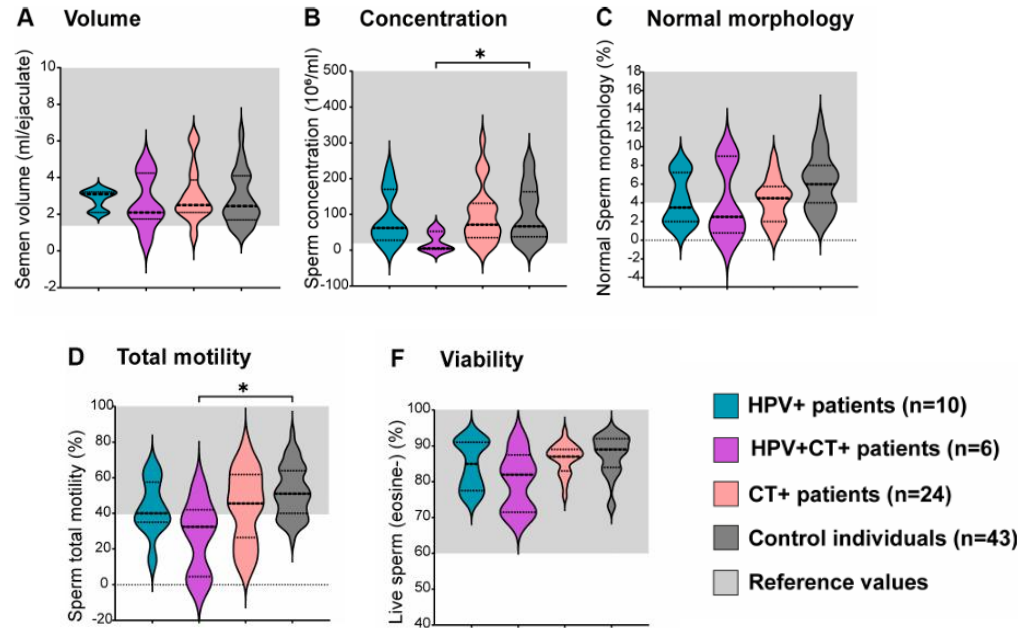
A previous study we conducted in a cohort of infertile patients found that

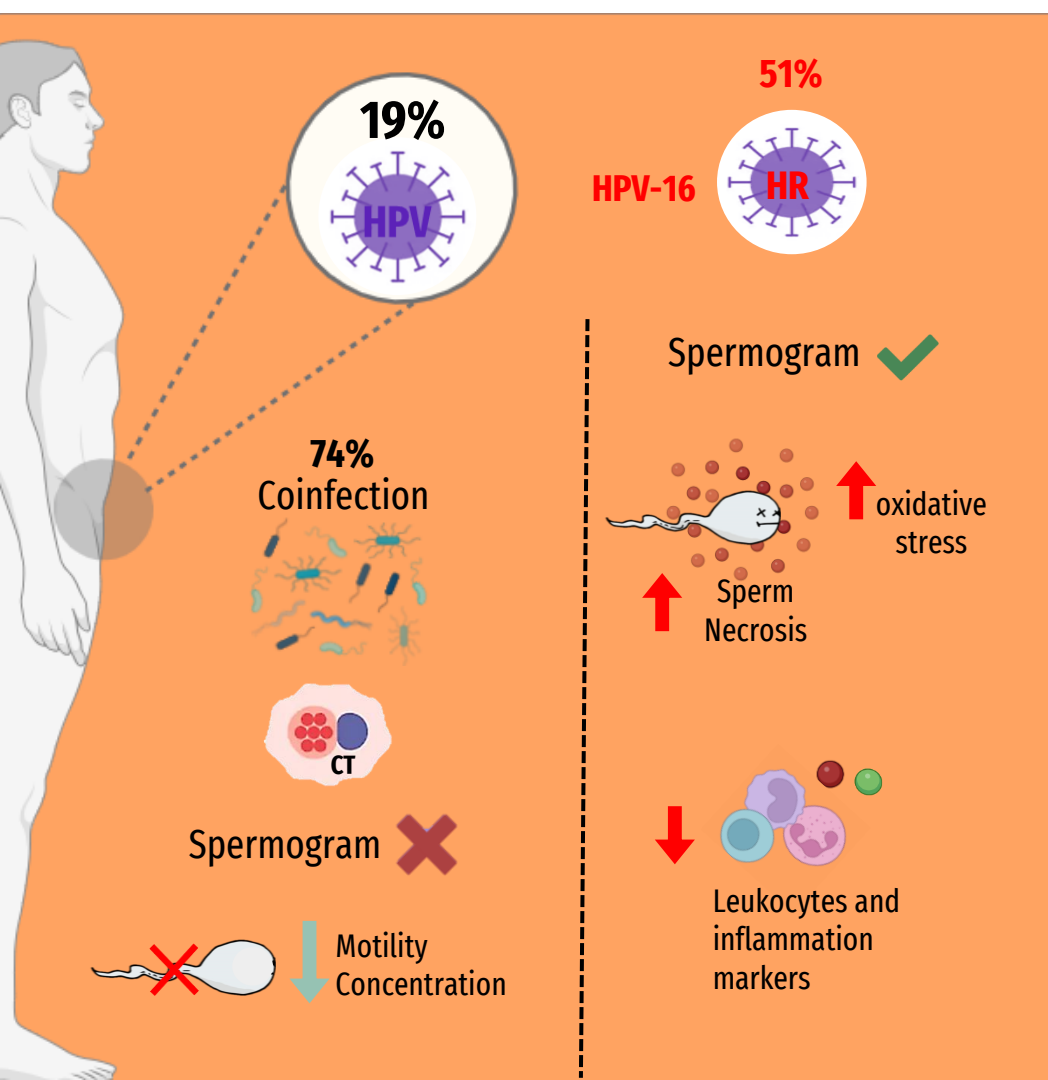
In the current cohort of patients..

HPV+ PATIENTS ARE 12 TIMES MORE LIKELY TO HAVE CHLAMYDIA THAN HPV- PATIENTS.



@darwid_illustration





→ Our study shows that **HPV infection** in men **is common**, and it often comes along with **other infections**, especially ***Chlamydia***.

→ While HPV alone doesn't always affect basic semen quality, **high-risk types like HPV-16** seem to cause more **sperm necrosis and oxidative stress**. And when **HPV and chlamydia** occur together, sperm concentration and motility drop clearly.

→ These findings remind us that **men's HPV testing and vaccination matter** — not just for preventing cancer, but also for protecting fertility and overall reproductive health.

THANKS!

Lab. Rivero/Motrich

Dra. Virginia Rivero
Dr. Rubén Motrich
Dra. Daniela Paira
Bioq. Sol Martínez
Bioq. Fernando Ferreyra
Bioq. Yair Chocobar
Dra. Janet Godoy
Bioq. Florencia Salazar
Dra. Silene Silvera

Lab. HPV y *Chlamydia* INVIV

Dra. Cecilia Cuffini
Dra. Jessica Mosmann

LAR andrology and reproduction lab

DASPU Urology departament

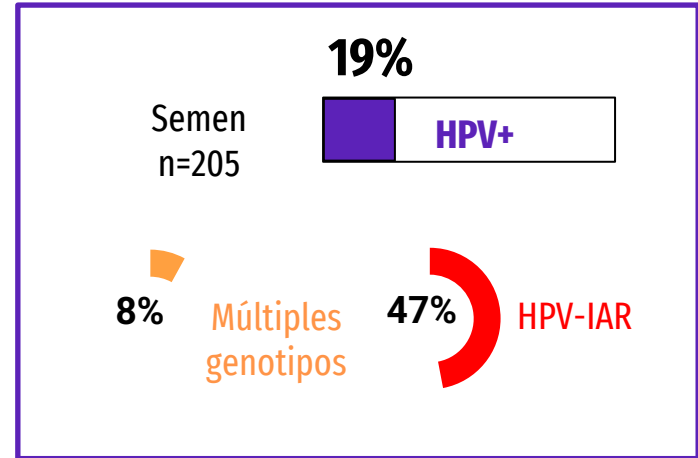
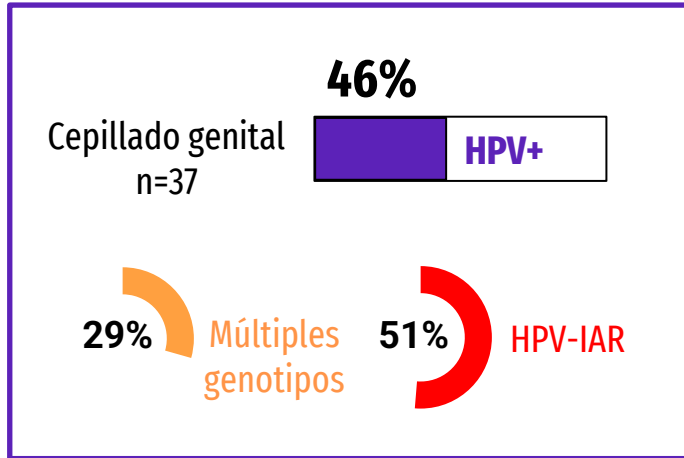
FUCDIM Urology clinic

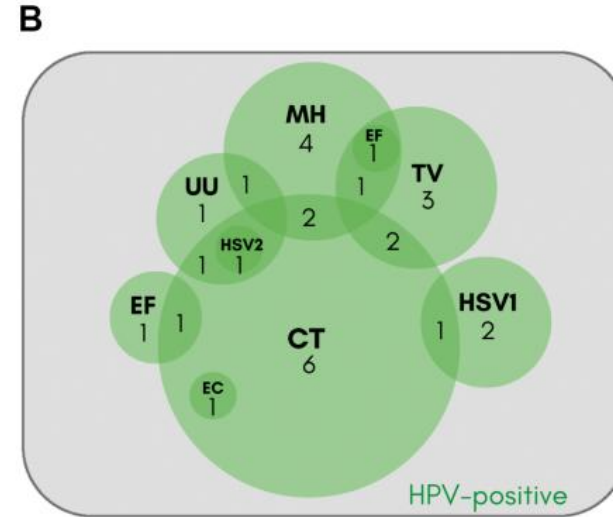
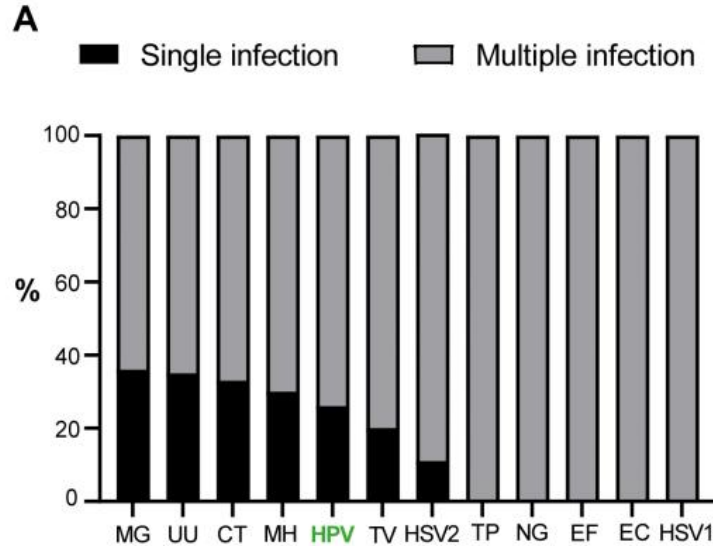
Voluntary Patients



C I B I C I

Prevalencia de HPV según el tipo de muestra evaluada





El 74% de los pacientes HPV+ presentaron coinfecciones

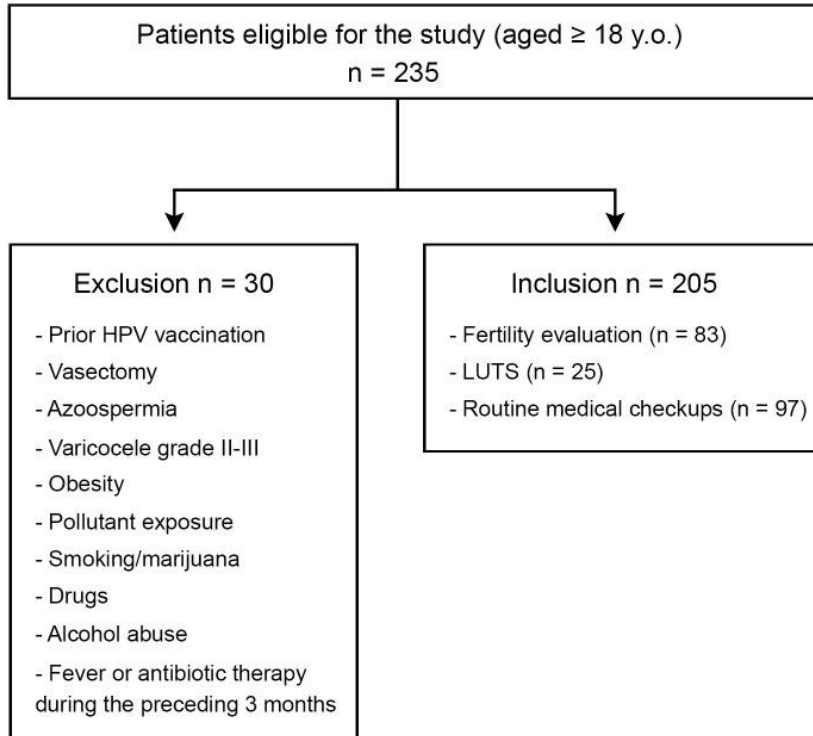
FIGURE 2

Characterization of the urogenital infection by HPV and other common uropathogens in a cohort of sexually adult young adult men attending a urology clinic. **(A)** The bar graph illustrates the coinfection rates detected according to the uropathogens analyzed. The percentage of single infection is depicted in black and the percentage of multiple infection with ≥ 2 pathogens detected is shown in gray. MG, *Mycoplasma genitalium*; UU, *Ureaplasma urealyticum*; CT, *Chlamydia trachomatis*; MH, *Mycoplasma hominis*; HPV, Human Papillomavirus; TV, *Trichomonas vaginalis*; HSV2, Herpes simplex virus type 2; TP, *Treponema pallidum*; NG, *Neisseria gonorrhoeae*; EF, *Enterococcus faecalis*; EC, *Escherichia coli*; HSV1, Herpes simplex virus type 1. **(B)** The Venn diagram illustrates the cases of HPV-associated coinfections. Coinfection cases with one other uropathogen: 6 HPV-CT, 4 HPV-MH, 3 HPV-TV, 2 HPV-HSV1, 1 HPV-EF, and 1 HPV-UU. Coinfection cases with 2 other uropathogens: 2 HPV-CT-TV, 2 HPV-CT-MH, 1 HPV-CT-HSV1, 1 HPV-CT-UU, 1 HPV-CT-EF, 1 HPV-CT-EC, 1 HPV-MH-TV, 1 HPV-MH-UU. Coinfection cases with more than 2 other uropathogens: 1 HPV-MH-EF-TV and 1 HPV-CT-UU-HSV2.

TABLE 1 Total leukocytes and different leukocyte cell subsets in semen from patients bearing high or low-risk HPV male urogenital infections.

Total leukocytes and different leukocyte cell subsets	Control individuals (n=43)	HR-HPV patients (n=20)	p-value ^a	LR-HPV patient (n=7)	p-value ^b	p-value ^c
Peroxidase-positive cells (x10 ⁵ /ml)	2.12 ± 2.84	1.01 ± 1.94	0.208	3.88 ± 6.2	0.929	0.489
CD45-positive cells (x10 ⁵ /ml)	6.75 ± 10.3	1.83 ± 2.95	0.019	11.01 ± 15.2	0.707	0.205
PMN leukocytes (x10 ³ /ml)	148.76 ± 214.6	121.07 ± 193.85	0.677	96.57 ± 182.12	0.356	0.282
Monocytes (x10 ³ /ml)	64.14 ± 114.41	37.15 ± 63.86	0.671	61.44 ± 138.46	0.207	0.207
Lymphocytes (x10 ³ /ml)	47.92 ± 98.86	10.96 ± 13.84	0.405	34.13 ± 40.78	0.615	0.183
T cells (x10 ³ /ml)	16.88 ± 46.66	3.49 ± 5.33	0.349	25.7 ± 36.38	0.526	0.340
CD4 T cells (x10 ³ /ml)	3,55 ± 9.78	1,97 ± 4.25	0.913	1.16 ± 1.75	0.803	0.538
CD8 T cells (x10 ³ /ml)	2,98 ± 4.88	1.15 ± 2,33	0.084	1.02 ± 1,67	0.487	0.682
CD4/CD8 T cells ratio	1,44 ± 1.91	2.68 ± 2.67	0.160	1.6 ± 1,98	0.620	0.449
B cells (x10 ³ /ml)	2.19 ± 4.64	3.23 ± 3.92	0.339	0.27 ± 0.28	0.531	0.268

HR-HPV patients: all HPV-positive patients presenting intermediate or high oncogenic risk genotypes; LR-HPV patients: all HPV-positive patients presenting low oncogenic risk genotypes; Control individuals: Individuals without leukocytospermia and negative for all analyzed uropathogens. Leukocyte population analyses were conducted using flow cytometry within gates defined on the total leukocyte cell population (CD45+). The subpopulations identified included polymorphonuclear leukocytes (PMN, CD11c-CD11b+), monocytes (CD14+CD11b-), and lymphocytes (CD11c-, CD11b-). T cells were characterized as CD3+ lymphocytes, with further subtyping into CD4 T cells (CD3+ CD4+ CD8-) and CD8 T cells (CD3+CD4-CD8+), along with B cells (CD3-CD20+). The results were reported as absolute leukocyte counts. p-values were calculated using the Mann-Whitney test. Comparisons were performed between HR-HPV group (^a) or LR-HPV (^b) and the control group. (^c) Indicates comparisons between HR-HPV and LR-HPV groups. Data are shown as Mean ± SD. Bold numbers indicate statistically significant differences when p < 0.05.





GLOBAL ACTION ON
MEN'S HEALTH

NOMAN
IS AN ISLAND
RACE TO END HPV
AND PREVENT 5% OF CANCERS

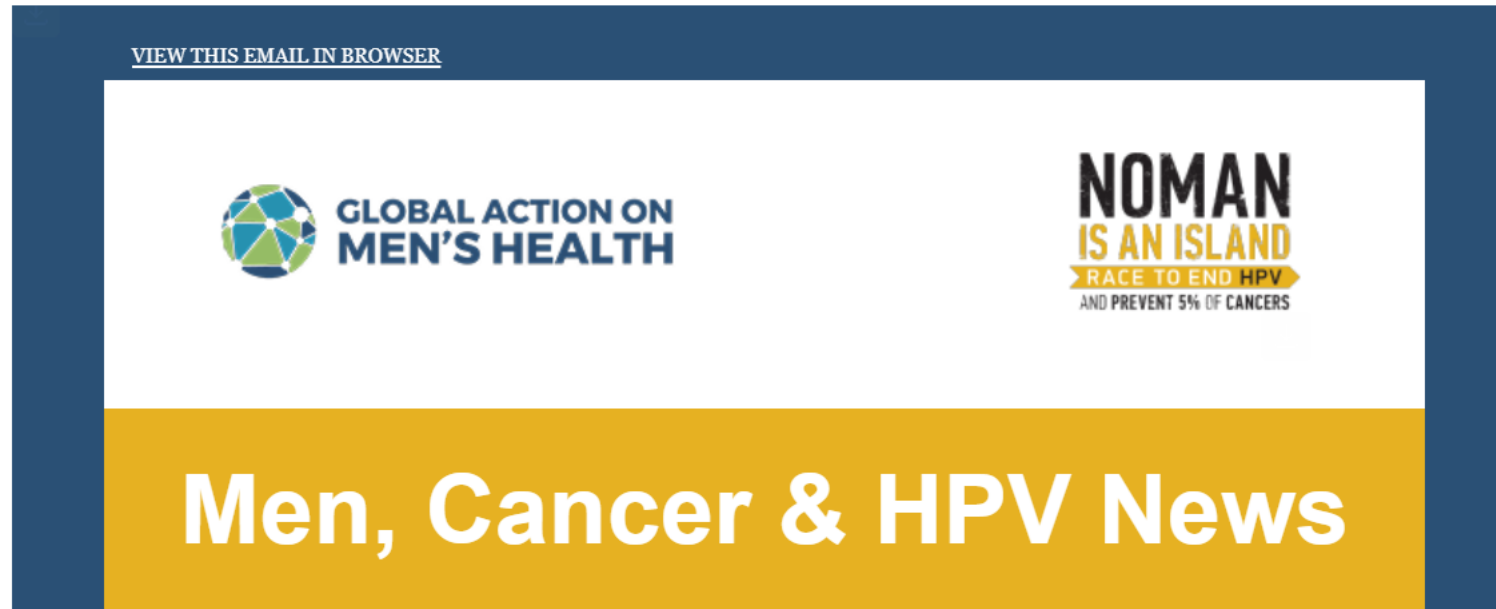
BOYS, MEN & HPV



A CALL FOR GLOBAL GENDER-NEUTRAL VACCINATION

FORTHCOMING ACTIVITIES

SIGN-UP FOR OUR QUARTERLY NEWSLETTER: Men, Cancer & HPV News



Eliminating all HPV cancers through worldwide universal vaccination by 2030

A newsletter from Global Action on Men's Health and NOMAN is an Island: Race To End HPV

Go to: <http://bit.ly/4gv3aYu>



SUPPORT OUR OTHER 2025 ACTIVITIES

- Endorse our Call to Action for global gender-neutral (universal) HPV vaccination at www.endhpvglobal.org.
- Endorse our forthcoming academic Position Statement on Men, Cancer and HPV.
- Join our men and cancer side-event at European Cancer Summit, Brussels, 19 November 2025, 17h00
- Support advocacy to change global policies and targets that restrict expansion of HPV vaccination programmes to boys.
- Watch this space for our further webinars and activities!



JOIN OUR NEXT WEBINAR IN DECEMBER

Men, Cancer & HPV Webinar: Protecting All: Advancing Universal HPV Vaccination in the MENA Region

Provisional date: Tuesday 9th December 2025,
11am BST | 12noon CEST | 4.00pm UAE

SPEAKERS TO BE CONFIRMED

Join our newsletter to receive registration details



**GLOBAL ACTION ON
MEN'S HEALTH**

NOMAN IS AN ISLAND
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AND PREVENT 5% OF CANCERS

THANKS TO THOSE WHO HAVE ENDORSED OUR CALL TO ACTION



PLEASE EVALUATE THIS WEBINAR

Go to this link or QR code to complete an anonymous rapid evaluation (only 6 questions)

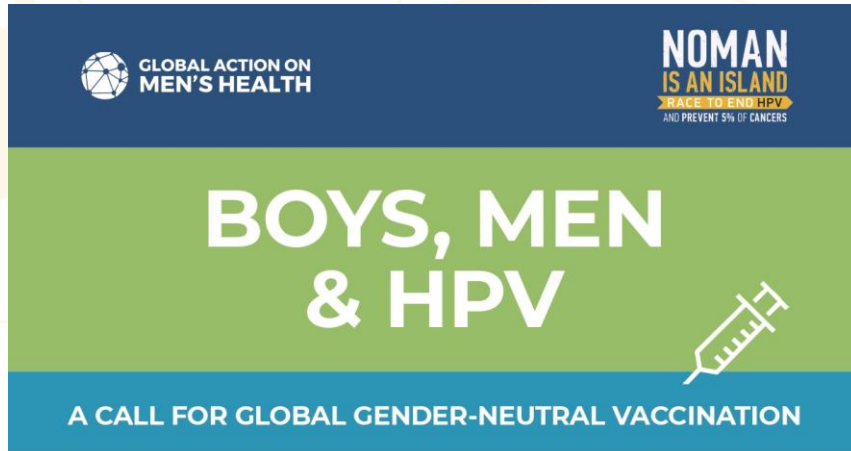
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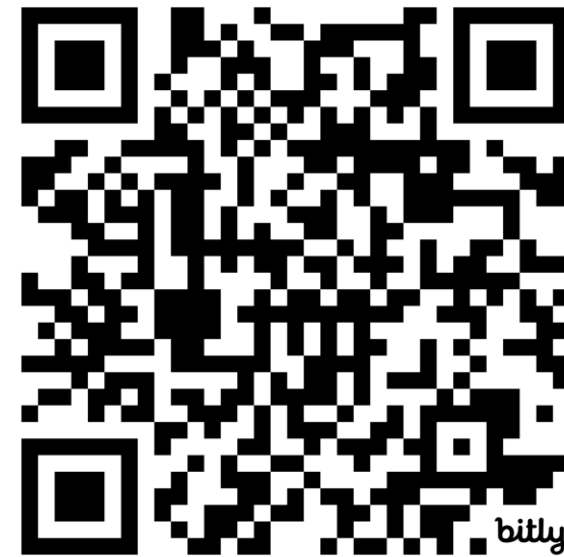
THANK YOU FOR JOINING US



Key messages

- We have the means to protect everyone – whatever their sex or gender – from high-risk human papillomavirus (HPV) infections and the cancers they cause. To achieve this, a more ambitious, ethical and equitable approach to HPV vaccination is needed at the global and national levels.
- Global Action on Men's Health, NOMAN is an Island: Race to End HPV and the supporters of this Call are seeking the worldwide adoption of gender-neutral (ie. universal) vaccination (GNV) by 2030 with a 90% uptake goal.
- We urge global public health organisations to prioritise the elimination of all the cancers caused by HPV. Every young person should be considered a primary target for vaccination by the World Health Organisation (WHO) and other key health organisations including Gavi, The Vaccines Alliance.
- From an epidemiological perspective, when both males and females are at risk of HPV, it is illogical to immunise girls alone. Only GNV can achieve the elimination of the vaccine-related high-risk HPV types and prevent the cervical and other cancers they cause.
- About 1 in 5 men has a current high-risk HPV infection. A conservative estimate of the number of new HPV cancer cases in men globally is 180,000 annually, with the actual number quite plausibly much higher.
- Despite the burden of HPV-related cancers in men, there are no established routine screening programmes for these cancers leading to delays in diagnosis and treatment.
- GNV increases the resilience of vaccination programmes, helping to protect against crises in vaccine confidence or disruption caused by pandemics, natural disasters or conflict.
- Vaccinating both boys and girls de-feminises and de-stigmatises immunisation programmes. GNV shares the responsibility for cancer prevention more equitably between the sexes.
- WHO's recent recommendation of the option of single-dose vaccination programmes, combined with increasing vaccine supply, makes GNV feasible on a global basis.

Read our report



www.endhpvglobal.org

Reference: Baker P and Winterflood D. 'Boys, Men and HPV: A Call for Global Gender-Neutral HPV vaccination. Global Action on Men's Health and NOMAN: Race to End HPV; London UK, 2024.