UNIVERSITY OF KWAZULU-NATAL
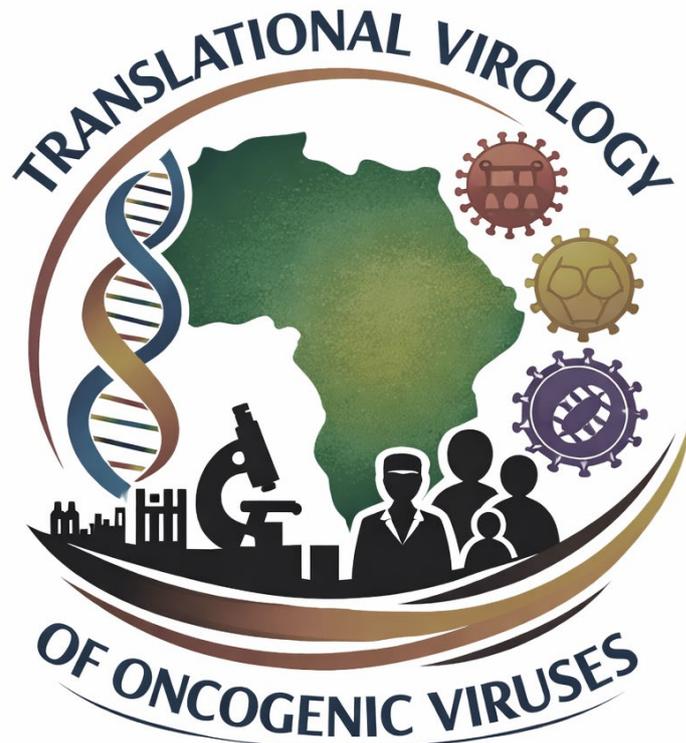
INYUVESI YAKWAZULU-NATALI

International Centre for Genetic Engineering and Biotechnology

18 – 20 MARCH 2026

TRANSLATIONAL VIROLOGY

OF ONCOGENIC VIRUSES

From Epidemiology to Intervention in Africa

Welcome Note

Dear Esteemed Delegates,

On behalf of the organising committee, we would like to welcome you to the international symposium on Translational Virology of Oncogenic Viruses, from Epidemiology to Interventions in Africa.

This scientific meeting adopts a translational approach to highlight the burden of cancers driven by viral infections and to critically examine virus-driven cancer research and interventions in Sub-Saharan Africa (SSA). The 2022 Lancet Oncology Commission Report presented a stark narrative of Africa's escalating cancer burden, drawing attention to the region's growing public health crisis. In SSA, a disproportionately high proportion of cancers are associated with viral infections, including liver cancer (hepatitis B and C viruses), cervical cancer (human papillomavirus), and Kaposi sarcoma (human herpesvirus 8). Collectively, virus-associated malignancies accounted for 27% of all incident cancers in 2018 the highest proportion of any WHO region.

The high burden of HIV in sub-Saharan Africa further amplifies this challenge, driving the incidence of HIV-associated malignancies such as Kaposi sarcoma, cervical cancer, and non-Hodgkin lymphoma. This creates a syndemic of communicable and non-communicable diseases, in which overlapping epidemics compound vulnerability and health system strain. This occurs despite significant reductions in HIV-related mortality and the large-scale expansion of antiretroviral therapy (ART) programmes across the subcontinent. As populations of people living with HIV (PLHIV) achieve longer life expectancy, the incidence of malignancies in these vulnerable populations continues to rise, shifting the disease burden from primarily communicable diseases to non-communicable morbidity and mortality. This symposium responds to this evolving landscape by fostering interdisciplinary dialogue, strengthening translational research pathways, and promoting evidence-based interventions that bridge epidemiology, molecular virology, clinical research, and public health implementation.

We encourage you to make the most of this event by actively participating in sessions, expressing your viewpoints, and forming valuable connections with fellow attendees. Your presence here contributes to a rich tapestry of knowledge sharing and collaboration, driving our community toward new horizons.

We sincerely hope that you will enjoy the sessions, and we are confident that this symposium will provide you with not only professional growth but also lasting memories and connections.

Thank you for joining us, and we wish you a rewarding and inspiring experience.

Warm regards,

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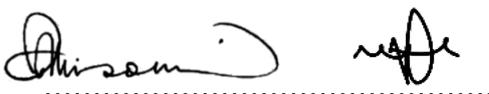
Drs N Msomi and Y Mdleleni

Scientific Meeting Organizers

Event Organisers and Scientific Committee

Dr Nokukhanya "Khanyi" Msomi

MBChB, DipHIVMan (SA), FC Path-Viro (SA), MMed, PhD

A Clinical Virologist with an interest in diagnostic virology, surveillance and the elimination of viral diseases of public health significance. Her research interest is in the control and elimination of viral hepatitis. Khanyi leads clinical and diagnostic virology laboratory services in KZN and is jointly appointed by the University of KwaZulu-Natal and the National Health Laboratory Service as the academic head of department. She is a member of the Network for Genomic Surveillance in South Africa (NGS-SA), the South African National AIDS Council (SANAC) and the Polio Eradication Committee of South Africa.

Dr Yanga Mdleleni

MSc (Cum laude), PhD

Dr Mdleleni is an academic and researcher at the school of Science and Agriculture, University of KwaZulu-Natal. She holds a PhD in Medicine (Virology) from the UKZN, Nelson Mandela School of Medicine. Her research interests include mental health and HPV pathogenesis, with a particular focus on understanding immunological and viral-host factors that drive HPV-related malignancies particularly in women living with HIV and the LGBTQI+ community. She serves on the editorial board of the Tumor Virus Research Journal and is the founder of the Nguta Foundation; a youth advocacy and empowerment organization. Yanga is a member of the international Papillomavirus society, and the South African Immunology society, as well as a former chair of the Equity and Inclusion committee at the World Economic Forum Global Shapers portfolio.

Dr Aabida Khan

Pathologist in the department of virology, National Health Laboratory Service and University of KwaZulu-Natal, based at Inkosi Albert Luthuli Central Hospital. Primarily involved in the diagnostic virology laboratory providing a consultative service, oversees the HIV drug resistance testing bench, involved in laboratory management, quality assurance, virology outreach support, teaching and training as well as being involved in research. Research interests include human papillomavirus (HPV) in head and neck cancers, HIV drug resistance, diagnostic virology, maternal and child health.

Miss Lulama Mthethwa

PhD candidate in Virology at the University of KwaZulu-Natal and a registered Medical Biological Scientist (Virology). She has research interests in hepatitis B virus (HBV) pathogenesis, viral genomics, HBV genetic variability, and biomarkers of functional cure in high-prevalence settings. She has received training in genomics, bioinformatics, next-generation sequencing (NGS), clinical virology, and molecular diagnostics through the NHLS, CERI and international institutions such as Wellcome Sanger Institute in Cambridge, UK. Her interests are to integrate molecular, bioinformatic, and clinical data to generate translational insights for public health.

Miss Olwethu Majola

PhD candidate at the University of KwaZulu-Natal. Her research interests are investigating viral host genetics of viruses such as HPV, maternal and Child health. Her current project explores vertical transmission of HPV and its links to juvenile onset recurring respiratory papillomas. Olwethu has been fortunate enough to be funded by the National Research Foundation throughout her postgraduate studies.

18 – 20 March 2026 | Durban, South Africa

Translational Virology of Oncogenic Viruses: From Epidemiology to Intervention In Sub-Saharan Africa

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PROGRAMME

18 MARCH 2026		
08:30 – 09:00	**Registration**	
09:00 – 09:20	**Welcome address**	**Dr Nokukhanya Msomi & Dr Yanga Mdleleni,** UKZN Scientific Organisers
		Professor Busi Ncama, UKZN Deputy Vice Chancellor-College of Health Science
		Prof Refiloe Masekela, Dean and Head: UKZN School of Medicine

18 MARCH 2026		
SESSION 1: EPIDEMIOLOGY OF KEY ONCOGENIC VIRUSES		
Session Chair: Dr Nokukhanya Msomi		
09:20 – 09:50	**Invited speaker** DR MAZVITA MUCHENGETI, Director National Cancer Registry, South Africa	**Epidemiology of virus-driven cancers in SSA**
09:50 – 10:20	**Invited speaker** PROF JOHN DOORBAR, Professor of Viral Pathogenesis & Fellow of Hughes Hall, Department of Pathology, University of Cambridge	**HPV Oncogenesis and insights**
10:20 – 10:50	**Tea Break**	
10:50 – 11:00	Yanga Mdleleni	The prevalence of HPV infection and incidence of HPV-related disease in Men who have sex with men
11:00 – 11:10	Kerri-Lee Francois	Correlation of high-risk HPV genotypes with cervical cytopathology in a high HIV prevalence setting
11:10 – 11:20	Nontokozo Matume	Genetic diversity of selected high-risk HPV types prevalent in Africa and not covered by current vaccines

18 MARCH 2026

Time	Speaker	Title
11:20 – 11:30	Tseganesh Yifru	Determinants of HPV associated cervical cancer screening among HIV positive women in Lesotho
11:30 – 11:40	Bontle Sibiya	Translating epidemiology into intervention, urethral high-risk HPV burden and HIV-associated immunosuppression among MSM in South Africa
11:40 – 12:05	Q&A for Session 1	
12:05 – 12:50	**Lunch Break**	

SESSION 2: HUMAN PAPILLOMA VIRUS-RELATED NEOPLASMS
Session Chair: Dr Yanga Mdleleni

Time	Speaker	Title
12:50 – 13:20	**Keynote speaker** **DR LAWRENCE BANKS,** Director: ICGEB	**Viral Oncogenesis: Mechanisms of Pathogenesis**
13:20 – 13:30	Dalia Ahmed	Association of HPV proteins E6 & E7 with prostate PSA and prostate cancer among Sudanese population
13:30 – 13:40	Excellent Maleka	Comparison of Real-Time Polymerase Chain Reaction (RT-PCR) and Immunohistochemistry (IHC) in Detecting HPV Infection in Oesophageal Squamous Cell Carcinoma (OSCC) Tissues
13:40 – 13:50	Linda Ratjama	Molecular role of HPV association with HNSC in Namibia
13:50 – 14:00	Carly Burmeister	HPV E6/E7 underpins the molecular mechanisms by which TBX3 contributes to cervical cancer
14:00 – 14:10	Michael Goldschagg	Exploring the functional significance of the interaction between the HPV capsid protein L2 and the oncogenic transcription Factors TBX2 and TBX3 in the HPV life Cycle
14:10 – 14:35	Q&A for Session 2	

SESSION 3: POSTER VIEWING AND COCKTAILS

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SESSION 4: IMPLEMENTATION RESEARCH
Session 3: Dr Andile Sibiya

Time	Speaker	Title
08:30 – 09:00	**Invited speaker** **PROF MARY KAWONGA,** Public Health Medicine Specialist and Associate Professor, Gauteng Department of Health and WITS School of Public Health.	**Implementation science of cancer prevention and control strategies**
09:00 – 09:10	Thobile Malinga	Introduction and coverage of HPV vaccination programmes in Africa: A systematic review

19 MARCH 2026

09:10 – 09:20	Nivashnee Naicker	Diagnostic accuracy of the Xpert HPV assay in women living with HIV at antiretroviral treatment initiation
09:20 – 9:30	Lemoupa Makajio	Home vs hospital-based HPV self-sampling for cervical cancer screening in the rural west region of Cameroon (cluster RCT)
09:30 – 09:40	Tchouptang Njiyip	Detection of HCV using a gold nanoparticle-based colorimetric kit: A rapid and low-cost approach
09:40 – 10:00	Q&A for Session 4	
10:00 – 10:30	**Tea Break**	

SESSION 5: VIRAL HEPATITIS
Session Chair: Dr Lawrence Banks

10:30 – 11:00	**Keynote speaker** **PROF WENDY SPEARMAN**, University of Cape Town, Head of Division of Hepatology: MBChB MMed PhD FCP(SA) FRCP(London)	**Viral Hepatitis and Cancer in Sub-Saharan Africa: Emerging Trends towards Elimination**
11:00 – 11:10	Yassir Hamadalnil	Targeting HBx-Driven HBV Replication: Inhibitory Effects of Punicalagin, SC75741 in Cell Culture Models
11:10 – 11:20	Thembelihle Tombo	Evaluation of a single assay for the surveillance of multiple viral pathogens using Next Generation Sequencing
11:20 – 11:30	Doha El-Sayed Ellakwa	Identifying of HBV DNA in liver tissues of chronic hepatitis and hepatocellular carcinoma to study the Hepatitis B virus silent infection in Egyptian patients
11:30 – 11:40	Lulama Mthethwa	Association of HBV HBx mutations with clinical outcomes in chronic HBV/HIV coinfected patients
11:40 – 12:00	Q&A for Session 5	
12:00 – 12:45	**Lunch Break**	

SESSION 6: GAMMA-HERPESVIRUSES AND RELATED
CANCERS Session Chair: Dr Aabida Khan

12:45 – 13:15	**Invited speaker** **DR OWEN NGALAMIKA**, Dermatologist and Senior Lecturer, University of Zambia	**Gamma herpes virus associated cancers is SSA: emerging trends**
13:15 – 13:45	**Invited speaker** **DR GUGU JALI**, Haematology pathologist and Clinical Haematologist	**Virus-driven haematological malignancies**

19 MARCH 2026		
13:45 – 14:15	**Invited Speaker** **PROF GEORGIA SCHAFER**, ICGEB Group Leader , Honorary Professor, University of Cape Town	The infection dynamics of gamma herpesviruses in South Africa
14:15 – 14:25	Pearl Segwagwa	Molecular Characterisation of Kaposi Sarcoma in Visceral Organs at a Tertiary Laboratory in Northern Tshwane: A Study of Two Decades
14:25 – 14:35	Lucia Naddy Mhlongo	Immunohistochemical analysis of Kaposi Sarcoma Herpesvirus in Paediatric Kaposi Sarcomas: A 21-year study at a Tertiary Laboratory in Tshwane
14:35 – 14:45	Yiqiang Wang	The Role and Implication of MXRA7 in Viral Infection-mediated Inflammatory Carcinogenesis
14:45 – 15:00	Q&A for Session 6	
15:00 – 16h00	**PRIZE GIVING CEREMONY** **INDUSTRY EXHIBITIONS AND COCKTAILS**	

| PRACTICAL WORKSHOP Venue: Department of Virology, Inkosi Albert Luthuli Central Hospital Level 5 Laboratory Building		
09:00 – 09:30	**DR JENIFFER GIANDHARI**, Head of the KwaZulu Research Innovation and Sequencing Platform, University of KwaZulu-Natal	Seminar on sequencing approaches and their clinical utility for cancer diagnosis and prognostication
09:30 – 10:00	Inqaba Biotec	TBC
10h00 – 10h30	Tea Break	
11h00 – 12h30	NATIONAL HEALTH LABORATORY SERVICE Inkosi Albert Luthuli Central Hospital	DIAGNOSTIC LABORATORY VISIT
12h30	**END OF SYMPOSIUM LUNCH**	

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We look forward to hosting you!
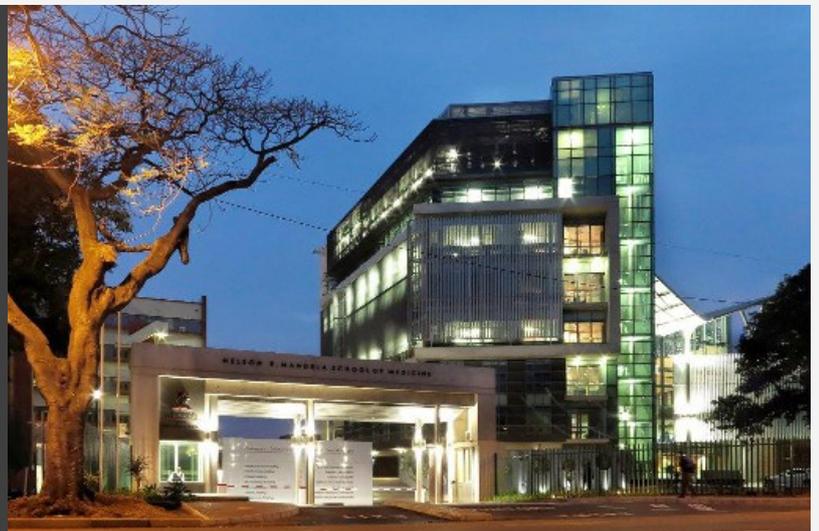
Keynote and Invited Speakers

Professor Lawrence Banks, Virologist and Director-General of the International Centre for Genetic Engineering and Biotechnology (ICGEB), Italy

Topic: Viral Oncogenesis: Mechanisms of Pathogenesis

Lawrence Banks received his Degree in Microbiology in 1981 and his PhD from Leeds University, UK in 1984. In 2019 he was elected Director-General of the International Centre for Genetic Engineering and Biotechnology (ICGEB), an international scientific research organization that promotes sustainable development through advanced research and education with Headquarters in Trieste, Italy. (www.icgeb.org). Since being appointed DG, Lawrence Banks has undertaken the enhancement of technical assistance programmes and aligning the ICGEB with the fulfilment of the SDGs. During this time, he has overseen a great expansion of ICGEB's cooperation with other international agencies including UNOSSC, TWAS, UN Tech Bank for LDCs and Bill and Melinda Gates Foundation, thereby bringing science and technology to the people that most need it. He has also been Group Leader of the Tumour Virology Laboratory at ICGEB Trieste, Italy since 1990. His current research interest focuses on Human Papillomaviruses (HPVs), the causative agents of a number of human tumours, the most important of which is cervical cancer, a major cause of cancer-related death in women worldwide.

Professor Marry Kawonga, Public Health Medicine specialist, researcher and academic, University of Witwatersrand, SA

Topic: Implementation science of cancer prevention and control strategies

Prof Kawonga is a public health physician with 25 years of experience as a public health medicine specialist practitioner, researcher and academic. I have expertise in health systems analysis; using evidence to inform the design of health policy and programmes; supporting the technical aspects of health service planning, implementation, and evaluation at provincial, district, and institutional levels; and analysing and developing national and provincial policy. She is passionate about strengthening health system capacity at individual and institutional, levels. Academic leadership includes directing undergraduate and postgraduate teaching programmes, convening public health courses; leading curriculum review and design; supervising doctoral and master's student research (supervised 27 master's and five PhDs to completion and currently supervise nine master's and three PhDs); and mentoring emerging researchers. Her research interests are in health services research and implementation science. In addition to 47 peer-reviewed articles, she has co-authored national policy, technical reports, national strategy documents and policy briefs. For 25 years She has dedicated a significant part of her research, advocacy, and policy work to cervical cancer prevention – including developing and testing strategies to put national policy into practice, co-drafting the national cervical cancer prevention policy, serving on technical teams tasked to develop implementation guidelines, serving on a WHO technical group developing a guide for essential practice, and using research evidence to develop contextually-relevant implementation guidelines for health programme managers and training guidelines for health practitioners.

Professor John Doorbar, Professor of Viral Pathogenesis at the Department of Pathology, University of Cambridge, UK

Topic: HPV Oncogenesis and insights on control

Professor Doorbar has studied Human Papillomaviruses and the life-cycle of HPV's for many years, and currently has interests in viral tropisms, latency and the different strategies used by the high and low-risk papillomavirus types. Following a recent move from the National Institute for Medical Research in London, he is now Professor of Viral Pathogenesis at the Department of Pathology, University of Cambridge, and Heads the MRC funded Papillomavirus Research group in the Division of Virology. His work encompasses the molecular biology of viral protein function as well as clinical aspects of disease progression and diagnosis using novel biomarker combinations to establish disease severity.

Professor Wendy Spearman, Head of the Division of Hepatology, Department of Medicine, Faculty of Health Sciences, University of Cape Town and Groote Schuur Hospital, Cape Town, SA

Topic: Viral Hepatitis and Cancer in Sub-Saharan Africa: Emerging Trends towards Elimination

Emeritus Professor of Medicine, Consultant Hepatologist, Division of Hepatology, Department of Medicine, Faculty of Health Sciences, University of Cape Town, South Africa. She was Head of the Division of Hepatology, Department of Medicine, Faculty of Health Sciences, University of Cape Town, and Head of Liver Transplantation, Groote Schuur Hospital, Cape Town, South Africa from 1/01/1998 - 30/06/2024. She obtained her PhD in 2008 from the University of Cape Town on "The effect of two novel C-type lectins, Ba100 and Ba25, isolated from the venom of the puff adder, Bitis arietans on T-lymphocyte proliferative responses." She is actively involved in outreach liver education programs and is the Lead of the 'Viral Hepatitis in sub-Saharan Africa" Project ECHO Clinic program. She was appointed as the lead consultant to develop the National Guidelines on the management and prevention of viral hepatitis in South Africa and as co-chair of the WHO 2024 Hepatitis B Guidelines Development Group. She is vice president of the Society on Liver Disease in Africa (SOLDA). Her research interests include viral hepatitis, MASLD, hepatocellular carcinoma, drug-induced liver injuries, liver transplantation, and novel immunosuppressants.

Dr Owen Ngalamika, physician scientist affiliated with the University of Zambia School of Medicine, and the University Teaching Hospital, Zambia.

Topic: Gamma herpes virus associated cancers is Sub-Saharan Africa: emerging trends

Dr. Owen Ngalamika (MBBS, MMed, PhD) is a physician scientist affiliated with the University of Zambia School of Medicine, and the University Teaching Hospital in Zambia. He conducts translational virology research on viral-associated cancers including Kaposi sarcoma caused by the Kaposi sarcoma-associated herpes virus (KSHV) and human papilloma virus-associated penile squamous cell carcinoma. His research focus has been on anti-viral and anti-tumor immunity both in the tumor microenvironment and in the peripheral circulation. He has investigated and published disease susceptibility for these cancers including correlates of protection, and biomarkers predicting treatment outcomes and disease recurrence.

Dr Mazvita Muchengeti, Head of Department of the South African National Cancer Registry (NCR), at the National Institute for Communicable Diseases (NICD), National Health Laboratory Service (NHLS). (MBChB, Postgraduate Diploma in HIV Management, MSc Epidemiology & Biostatistics, PhD)

Topic: Epidemiology of virus-driven cancers in Sub-Saharan Africa

Dr Muchengeti is the Head of Department of the South African National Cancer Registry (NCR), at the National Institute for Communicable Diseases (NICD), National Health Laboratory Service (NHLS). She also leads the IARC Global Initiative for Cancer Registry Development (IARC-GICR) Centre of Expertise in Johannesburg within the African Cancer Registry Network. She is a cancer epidemiologist with over 14 years of experience in cancer surveillance and epidemiological research, focusing on HIV-associated cancers, cervical cancer, and population-based cancer registration in African populations. She is a co-Principal Investigator of the South African HIV Cancer Match (SAM) Study and the South African Cervical Cancer Screening (SACCS) cohort. Dr Muchengeti is an Honorary Senior Lecturer at the University of the Witwatersrand and a Research Associate at the South African Centre for Epidemiological Modelling and Analysis (SACEMA), Stellenbosch University. She has published over 60 peer-reviewed articles and serves on several national and international advisory committees, including the South African Ministerial Advisory Committee on Cancer Prevention and Control.

Professor Georgia Schäfer, Virologist at the International Centre for Genetic Engineering and Biotechnology (ICGEB) and Honorary Professor in the Division of Medical Biochemistry at the University of Cape Town, SA.

Topic: The infection dynamics of gamma herpesviruses in South Africa

Georgia Schäfer obtained her PhD in molecular biology from Humboldt University Berlin, Germany, in 2003, followed by a post-doc at the University of Cape Town (UCT), South Africa. She is currently leading the research group "Virology-Emerging Viruses" at the International Centre for Genetic Engineering and Biotechnology (ICGEB) Cape Town, South Africa. She is also an Honorary Professor in the Division of Medical Biochemistry and an Adjunct Member of the Institute of Infectious Disease and Molecular Medicine (IDM) in the Faculty of Health Sciences at UCT. Georgia's research focusses on the infection mechanisms of Kaposi's sarcoma herpesvirus (KSHV), Epstein-Barr Virus (EBV) and Human papillomavirus (HPV)). These oncogenic viruses are particularly relevant in Sub-Saharan Africa and in the context of HIV co-infection where they cause substantial morbidity and mortality. In response to the COVID-19 outbreak, Georgia's group has set up several SARS-CoV2 diagnostic and research assays to support various national and international research activities. Georgia has published > 60 articles in peer-reviewed journals in the field and is the recipient of various national and international research grants. She has graduated >30 Hons, MSc and PhD students over the last 15 years and is strongly involved in mentoring programmes and teaching the next generation of scientists in Africa.

Dr Jeniffer Giandhari, School of Medicine and is the Head of Laboratory at the KwaZulu-Natal Research Innovation & Sequencing Platform (KRISP) at the University of KwaZulu-Natal (UKZN), SA

Topic: Seminar on sequencing approaches and their clinical utility for cancer diagnosis and prognostication

Jennifer Giandhari is a Senior Lecturer in the School of Medicine and is the Head of Laboratory at the KwaZulu-Natal Research Innovation & Sequencing Platform (KRISP) at the University of KwaZulu-Natal (UKZN). She obtained her PhD (2015) in virology at the University of Witwatersrand and the National Institute for Communicable Diseases. Her PhD dissertation contributed to HIV drug resistance research by identifying viral genetic variants and elucidating the underlying mechanism of resistance. Jennifer's research interests include advancing the understanding of viral evolution, drug resistance mechanisms, and molecular epidemiology. As the laboratory head of KRISP, she has led groundbreaking studies on pathogen surveillance, including tuberculosis, HIV, cholera, SARS-CoV2 and the Dengue virus, which have contributed to local and international public health responses. She has published extensively in high-impact scientific journals, including Science and Cell. Her research is supported by grants from the National Research Foundation (NRF), and the South African Medical Research Council (SAMRC). She is actively involved in raising funding and organizing KRISP-facilitated training programmes to provide young researchers with hands-on training in the laboratory. In 2024, she was awarded a Top 10 Published Young Researcher prize by UKZN.

Dr Gugu Jali, Clinical haematologist and Haematopathologist, Busamed and King Edward Hospital, SA

Topic: Virus-driven haematological malignancies

Dr Gugu Jali graduated with her MBChB at the University of Kwa-Zulu Natal in 2007 and qualified as a Haematopathologist at UKZN in 2015. Her MMed entitled "Prevalence and aetiology of moderate and severe thrombocytopenia in a tertiary and quaternary centre in KwaZulu-Natal", was published in 2020. She holds a Diploma in Palliative Medicine from the University of Cape Town and a Certificate in clinical haematology from the College of medicine in South Africa. Dr Jali is a consultant in the Clinical Haematology Unit at King Edward the VIII Hospital.

ABSTRACTS

T01- EPIDEMIOLOGY OF KEY ONCOGENIC VIRUSES

Correlation of High-Risk HPV Genotypes with Cervical Cytology in a High HIV- Prevalence Population in KwaZulu-Natal, South Africa

Kerri-Lee Francois[1,2], Cherise Naicker[1,2]

1.National Health Laboratory Service, Department of Virology, Inkosi Albert Luthuli Central Hospital
2. University of KwaZulu- Natal, Department of Virology, Faculty of Health Sciences.

Background: Cervical cancer remains the leading cause of cancer-related mortality among women in South Africa. Although the country has recently transitioned to high-risk human papillomavirus (HR-HPV) testing as the primary cervical cancer screening modality, data describing HR-HPV genotype distribution and its association with cervical disease in KwaZulu-Natal (KZN) remains limited. It is essential to investigate the prevalence of HPV genotypes and their distribution among women with different immune statuses and cytology results to optimise risk-based screening and triage strategies.
Methods: We conducted a retrospective analysis of HR-HPV genotyping and corresponding cytology results from liquid-based cytology specimens submitted to the National Health Laboratory Service in KwaZulu-Natal between February and April 2025, approximately one year after implementation of HPV-based screening. HR-HPV testing was performed using the Cobas® HPV and Alinity m HPV partial genotyping assays. Cytological outcomes were categorised as < High-grade squamous intraepithelial lesion (HSIL) or ≥HSIL. Data were stratified by age and HIV status and analysed using STATA version 18. Logistic regression models were used to assess associations between HR-HPV genotypes, HIV infection, age, and the presence of ≥HSIL.
Results: A total of 8,611 specimens were analysed. The mean age was 36.9 years, and 47.4% of women were HIV-infected. Cytological abnormalities ≥HSIL were identified in 15.9% of specimens. HPV 16 was the most prevalent genotype (24.2%) and was strongly associated with ≥HSIL (OR 2.95; 95% CI 2.56–3.40; p<0.001). HPV 45 was also associated with ≥HSIL, though with lower odds (OR 0.69; 95% CI 0.55–0.86; p<0.001), while HPV 18 showed no significant association. Other HR-HPV genotypes were frequently detected and associated with increased odds of ≥HSIL. HIV-infected women had higher odds of ≥HSIL compared to HIV-uninfected women (OR 1.64; 95% CI 1.44–1.86). No association was observed between HIV infection and specific HR-HPV genotype distribution. Increasing age was independently associated with disease severity, with a 3.1% increase in the odds of ≥HSIL per year, equating to an approximate 30% increase per decade among HIV-infected women.
Conclusion: HIV infection was associated with an increased risk of progression to ≥HSIL, regardless of HR-HPV genotype. The frequent detection of non-vaccine HR-HPV types and their link to high-grade cytological abnormalities highlight the need for expanded genotyping with histopathological confirmation. Local, population-based data is essential to refine HPV-based screening algorithms in South Africa. This supports genotype-specific triage, age- and HIV-associated risk stratification, as well as tailored follow-up for women living with HIV.

Genetic diversity of selected High-risk HPV types prevalent in Africa and not covered by current vaccines: A pooled sequence data analysis

Babalwa Nyide [1], Miranda Thomas [2], Lawrence Banks [2], Pamela Mkhize [1,3] and **Nontokozo D. Matume** [4,5*]

1. Discipline of Biochemistry, School of Agriculture and Science, University of KwaZulu-Natal, Pietermaritzburg, South Africa
2. Tumour Virology, International Centre for Genetic Engineering and Biotechnology (I.C.G.E.B.), Trieste, Italy
3. Centre for the AIDS Programme of Research in South Africa (CAPRISA), University of KwaZulu-Natal Medical School, Durban, 4013, South Africa
4. Discipline of Genetics, School of Agriculture and Science, University of KwaZulu-Natal, Pietermaritzburg, South Africa

5. SAMRC-UNIVEN Antimicrobial Resistance and Global Health Research Unit, HIV/AIDS & Global Health Research Programme, University of Venda, Thohoyandou, South Africa
* Correspondence: matumen@ukzn.ac.za or daphneymatume@yahoo.com

High-risk human papillomavirus (HR-HPV) types exhibit an uneven global distribution, with types 35, 51, 56, and 59 being more prevalent in Africa yet not covered by current L1-based vaccines. The genetic diversity of HR-HPV oncoproteins in Africa remains poorly characterized, despite their potential as alternative vaccine targets. This study investigates the genetic diversity of HR-HPV types 16, 18, 35, 51, 56, and 59 to inform vaccine development. A total of 14,332 sequences from the NCBI Virus database and 222 HPV reference sequences from the Papillomavirus Episteme (PaVE) database were analysed using phylogenetic analysis and variant identification. HPV16 and HPV35 exhibited close evolutionary relatedness, which may indicate shared traits relevant to vaccine design, although functional implications remain to be experimentally validated. A key finding of the study was the discovery of novel non-synonymous mutations, including E148K in HPV35 E6, S63C in HPV16 E7 and S495F in HPV18 L1, as well as known oncogenic variants such as L83V (E6) and N29S (E7) in HPV16. These findings highlight significant intra- and inter-type diversity among African HR-HPVs. This study provides new insights into the genetic diversity and evolutionary relationships of underrepresented HR-HPV types. The findings underscore the need for continued genomic surveillance and support efforts to develop region-specific vaccines that include HPV35, 51, 56, and 59 to address gaps in current vaccine coverage and help reduce the burden of HPV-related cancers in Africa.

Abstract Number: T01-3

Determinants of HPV-associated cervical cancer screening among HIV-positive women in Lesotho: Evidence from the 2023/24 DHS

Tseganesh Asefa[1]*, Hiwot Tezera Endale[2], Tiget Ayelgn Mengstie[3], Mihret Getnet[4]

[1]Clinical Trial Directorate, Armauer Hansen Research Institute, Addis Ababa, Ethiopia
[2]Department of Medical Biochemistry, School of Medicine, College of Medicine and Health Sciences, University of Gondar, Gondar, Ethiopia
[3]Department of Epidemiology and Biostatistics, Institute of Public Health, College of Medicine and Health Sciences, University of Gondar, Gondar, Ethiopia
[4]Department of Human Physiology, School of Medicine, College of Medicine and Health Sciences, University of Gondar, Gondar, Ethiopia
Email: tseganesh16@gmail.com

Introduction: Cervical cancer is an HPV-associated malignancy, and women living with HIV are at increased risk due to impaired immune clearance of oncogenic HPV infections. Despite Lesotho's high HIV prevalence (23.9%), cervical cancer screening coverage and its determinants among HIV-positive women remain insufficiently characterized. This study aimed to quantify the prevalence and determinants of cervical cancer screening among women living with HIV aged 25 years and older using the 2023/24 Lesotho DHS data.
Methods: A cross-sectional analysis was conducted using the Lesotho DHS Individual Women's Recode dataset. A weighted sample of 611.17 HIV-positive women aged ≥25 years was analyzed. Individual- and community-level determinants of cervical cancer screening uptake were assessed using multilevel mixed-effects logistic regression. Associations were reported using adjusted odds ratios with 95% confidence intervals, and a p-value < 0.05 was considered statistically significant.
Results: The prevalence of cervical cancer screening among women living with HIV was 85.39% (95% CI: 82.35–87.98%). Higher screening uptake was observed among women aged 40–44 years (AOR=3.44; 95% CI: 1.43–8.29) and those who had received a healthcare provider–performed breast examination (AOR=5.14; 95% CI: 2.55–10.36). In contrast, low parity (AOR=0.22; 95% CI: 0.06–0.78), high parity (AOR=0.26; 95% CI: 0.07–0.97), and rural residence (AOR=0.52; 95% CI: 0.28–0.95) were associated with reduced odds of screening uptake.
Conclusion and recommendations: Although cervical cancer screening coverage among women living with HIV in Lesotho is relatively high, significant disparities persist across age, parity, and place of residence. These findings highlight the need for integrated HIV and HPV-associated cancer prevention strategies, including combined breast and cervical cancer screening, targeted rural outreach, and tailored health education for multiparous and younger women. Translating epidemiological evidence into integrated service delivery can strengthen viral cancer prevention efforts in high HIV-burden settings.

Abstract Number: T01-4

TRANSLATING EPIDEMIOLOGY INTO INTERVENTION: URETHRAL HIGH-RISK HPV BURDEN AND HIV-ASSOCIATED IMMUNOSUPPRESSION AMONG MEN WHO HAVE SEX WITH MEN IN SOUTH AFRICA

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BACKGROUND: Human papillomavirus (HPV) remains a major cause of multiple anogenital and oropharyngeal cancers. While anal HPV in men who have sex with men (MSM) is well-studied, the urethral reservoir a potential site for transmission and oncogenesis remains poorly characterized, especially in high human immunodeficiency virus (HIV) prevalence settings like sub-Saharan Africa. Bridging this epidemiological gap is critical for informing targeted interventions. Thus, this study investigated the prevalence of urethral HPV infection among MSM and associated risk with HIV infection.

METHODS: A cross-sectional study was conducted among 199 MSM recruited from Gauteng and the North-West provinces, South Africa. HIV status was determined using HIV-1/2 Alere Determine™ Ag/Ab Combo test, confirmed using Uni-Gold™ Recombigen® HIV-1/2 rapid immunoassay. Urethral swabs were collected from 199 participants, however 195 samples were retrieved and extracted for DNA using the Abbott mSample Preparation System DNA Kit on the Abbott m2000sp platform, and HPV genotyping was performed on 191 samples using the Anyplex™ II HPV28 assay. Statistical analyses were conducted using R.

RESULTS: HIV prevalence was 66.3%, and urethral HPV was detected in 20.9% of participants with 16.2% having HIV/HPV co-infection. High-risk HPV types constituted 52.5% of infections, with HPV 16, 18, and 68 each present in 12.5% of cases. Types covered by Gardasil®9 accounted for 60.0% of infections. A clear relationship was observed between HIV-associated immunosuppression and urethral HPV infection: the predicted probability of HPV infection increased with declining CD4+ T-cell counts, with prevalence progressively decreasing across higher CD4 strata.

CONCLUSION: This study reveals a significant burden of urethral high-risk HPV among South African MSM, closely linked to HIV-related immunosuppression. The urethra emerges as a potential niche for oncogenic HPV, highlighting two translational priorities: integrating urethral HPV screening into routine MSM sexual health services and expanding HPV vaccination programs for high-risk groups in high HIV-burden settings.

T02 - HUMAN PAPILLOMA VIRUS-RELATED NEOPLASMS

Abstract Number: T02-1

Association of human papilloma virus (HPV) proteins E6/E7 and prostate cancer PCa incidence among Sudanese population

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Prostate cancer is the second most commonly diagnosed cancer worldwide. This study aimed to evaluate the frequency of human papillomavirus (HPV) infection among Sudanese patients with prostate cancer. A total of sixty-five (65) histologically confirmed prostate cancer cases and thirty-five (35) benign prostatic hyperplasia (BPH) samples were analysed for HPV infection using immunohistochemistry targeting the E6/E7 oncoproteins. HPV infection was detected in 41% of prostate tumour biopsies and 20% of BPH samples. Overall, 73.54% of HPV-positive patients demonstrated strong E6/E7 protein expression in tumour tissue.
A significant proportion of HPV-infected cases (71%) were associated with advanced disease stages (III and IV) (p ≤ 0.001) and higher Gleason scores (≥ 8) (p = 0.003). This study represents the first report of HPV incidence in

prostate cancer within the Sudanese population and supports the hypothesis that HPV infection may act as a co-factor in prostate cancer progression. The findings indicate a notable prevalence of HPV infection in Sudanese prostate cancer patients, particularly in advanced-stage and high-grade tumours. These results highlight the potential role of HPV in prostate carcinogenesis and underscore the need for further large-scale and mechanistic studies to elucidate the contribution of HPV to prostate cancer development and progression.

Abstract Number: T02-2

Comparison of Real-Time Polymerase Chain Reaction (RT-PCR) and Immunohistochemistry (IHC) in Detecting HPV Infection in Oesophageal Squamous Cell Carcinoma (OSCC) Tissues

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Background: Human papillomavirus (HPV) has been implicated in several squamous cell carcinomas, including oesophageal squamous cell carcinoma (OSSC), though its etiological role remains uncertain. This study investigated the prevalence and genotype distribution of HPV in OSCC and compared the diagnostic performance of immunohistochemistry (IHC) using HPV antibody and p16INK4a expression with real-time polymerase chain reaction (RT-PCR).
Methods: A retrospective cross-sectional study was conducted using formalin-fixed, paraffin-embedded (FFPE) OC specimens diagnosed between 2016 and 2021 at the National Health Laboratory Service, Dr George Mukhari Tertiary Laboratory, Gauteng Province. Of 607 OC cases identified, 112 histologically confirmed OSCC cases with adequate tissue were included. HPV detection was performed using IHC for HPV and p16INK4a, and RT-PCR was carried out using the Seegene Allplex™ II HPV28 assay for HPV genotyping. Diagnostic performance metrics and agreement between methods were evaluated.
Results: HPV was detected in 32.0% of OSCC cases by IHC HPV and in 15.0% by RT-PCR. Among RT-PCR–positive cases, HPV16 was the predominant genotype (64.7%), followed by HPV33 (17.6%) and HPV26, HPV58, and HPV69 (each 5.9%). IHC HPV showed moderate sensitivity (56.3%) and specificity (64.9%) compared with RT-PCR, with a high negative predictive value (84.1%). p16INK4a demonstrated poor specificity (33.3%) and low positive predictive value (21.6%). Agreement between IHC and RT-PCR methods was weak, while IHC HPV and p16INK4 a showed fair agreement.
Conclusion: HPV prevalence in OSCC varies according to the detection method used. Combined IHC approaches may be useful for initial screening in resource-limited settings, but RT-PCR remains essential for accurate HPV confirmation and genotyping in OSCC.

Abstract Number: T02-3

MOLECULAR ROLE OF HUMAN PAPILLOMA VIRUS ASSOCIATION WITH HEAD AND NECK SQUAMOUS CELL CARCINOMAS IN NAMIBIA

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Head and neck squamous cell carcinoma (HNSCC) is one of the most common cancers in developing countries and the Human papillomavirus (HPV) has been established as one of the main causative agents for HNSCC along with alcohol and tobacco use. High-risk HPV E6 proteins promote carcinogenesis through their C-terminal PDZ-binding motif which binds and degrades PDZ-domain-containing proteins crucial for maintaining normal cell structure and signaling. This study was aimed at investigating the molecular role of HPV associated HNSCC in Namibia. cDNA was synthesized from the RNA samples and sent for RNAseq. Cytotoxic responses to varying concentrations of nicotine and acetaldehyde were evaluated through XTT viability assays. To determine the HPV infectivity in the various cell lines, a luciferase activity assay was performed. A comparative proteomics analysis was conducted to clarify the interactions of HPV16E6 proteins in distinct cellular contexts. The study found a male-to-female ratio of 2.23:1, with most participants aged 50–59 and 63% diagnosed with laryngeal cancer. Among participants, 29% were smokers, 39.5% consumed alcohol, and 10.5% did both. In the HPV-positive non-cancerous group, seven genes were significantly downregulated (including TUT7, SLC8A1, BCL2L1), and WDR82 was

upregulated. In HPV-positive tumors, several genes (including ZBTB7C, KCNC4, ELL2) were downregulated, while OR2A5 was upregulated, suggesting epithelial plasticity and potential as a biomarker. Cytotoxicity assays showed HTC cells were resistant to nicotine but sensitive to acetaldehyde. Nicotine exposure (≥1 mM) increased HPV16 pseudovirion infectivity in C33α, HeLa, and HaCaT cells. Results from proteomic analysis showed that GOPC is unique to tonsillar cells. This protein is associated with autophagy and identified as a prognostic marker in HNSCC. Further work is recommended to explore the mechanisms of GOPC in HNSCC carcinogenesis. In conclusion, this study highlights the importance of integrating genomic, toxicological, and proteomic approaches to understand the complex virus–host interactions in HPV-related cancers. It lays the groundwork for future translational research and targeted interventions, particularly in underrepresented regions such as Sub-Saharan Africa. These findings not only advance knowledge of HPV-mediated oncogenesis in the local context but also contribute to the global discourse on cancer prevention and the role of environmental cofactors in viral pathogenesis.

Abstract Number: T02-4

HPV E6/E7 underpins the molecular mechanisms by which TBX3 contributes to cervical cancer

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Cervical cancer remains the fourth most common cancer among women worldwide, with over 85% of cases and deaths occurring in low- and middle-income countries (LMICs). This disparity is largely attributed to limited access to affordable treatments and healthcare infrastructure, which leads to late-stage diagnosis. Indeed, the human papillomavirus (HPV) is well-known to be responsible for more than 95% of cervical cancer cases and despite advances in HPV vaccination, education, and screening, cervical cancer continues to pose a substantial health burden in LMICs. This highlights the urgent need for improved and more accessible therapeutic strategies. The HPV oncoproteins E6 and E7 play pivotal roles in cervical cancer progression by targeting
key host tumour suppressors and coopting host oncogenic factors. Identifying these tumour promoters has important therapeutic potential and we have provided compelling evidence that the T-box transcription factor 3 (TBX3) is one such factor. Indeed, we previously reported that TBX3 levels are elevated in HPV-positive cervical cancer patient tissues and cell lines, and that it promotes HPV-positive cervical cancer cell proliferation and migration through its interaction and cooperation with the HPV oncoproteins E6 and E7. The current study built on this work by exploring whether the viral E6/E7 oncoproteins enhance binding of TBX3 to protein co-factors that increase TBX3 stability and/or oncogenic function in HPV-positive cervical cancer cells. To address this, co-immunoprecipitation coupled with mass spectrometry was employed to comprehensively map TBX3 interacting partners in HPV positive cervical cancer cells. This analysis identified an association between TBX3, nucleolin (multifunctional RNA binding protein), and heat shock cognate 70 (Hsc70, molecular chaperone), both of which had stronger binding to TBX3 in the presence of E6 and E7. Subsequently, Hsc70 was shown to interact and stabilise both TBX3 and nucleolin, while TBX3 and nucleolin were shown to be mutually required for one another to promote HPV-positive cervical cancer cell proliferation and migration. Given that nucleolin is already considered a druggable target, we tested the potential of the guanosine-rich oligonucleotide nucleoli targeting aptamer, AS1411, to exert anti-cervical cancer activity through its ability to disrupt TBX3/nucleolin interaction. We show that AS1411 displaced TBX3 and nucleolin from the nucleus to the cytoplasm and suppressed HPV-positive cervical cancer cell proliferation and migration. These findings position the TBX3/nucleolin/Hsc70 axis as a key host oncogenic pathway exploited by E6 and E7 to support cervical cancer progression. It further highlights a promising host directed therapeutic approach that could be adapted to resource-constrained settings. By mapping this previously uncharacterised host network, our work expands the understanding of HPV-mediated transcriptional and post-translational reprogramming and highlights potential translational relevance for high burden regions.

Abstract Number: T02-5

Exploring the functional significance of the interaction between the HPV capsid protein L2 and the oncogenic transcription Factors TBX2 and TBX3 in the HPV life Cycle

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BACKGROUND: Cervical cancer is the leading cause of cancer-related deaths in South African females and 99% of cases result from infection with high-risk Human Papillomavirus (HPV) strains, particularly HPV-16. The HPV minor capsid protein L2 assists with establishing this infection by interacting with host cellular proteins to deliver the viral genome into the host nucleus. Identifying and characterizing these host proteins may lead to targets for prophylactic and/or therapeutic HPV treatments. Of interest to this study are preliminary data showing that L2 interacts with the homologous oncogenic transcription factors TBX2 and TBX3 (TBX2/3). While we have previously shown that TBX3 interacts with the HPV E6/E7 oncoproteins to promote cervical cancer, the impact of the interaction between TBX2/3 and L2 on HPV infection is unknown, and this is explored in this study.

AIMS and METHODS: To confirm the interaction previously reported between HPV L2 and TBX2/3, co-immunoprecipitation and immunocytochemistry were performed in the CaSki and HeLa cervical cancer cells transfected with L2. To create a tool to study the impact of TBX2/3 on HPV infection, HPV-luciferase pseudoviruses were produced following standard protocols. HaCaT cells (host for HPV infection) in which TBX2/3 were depleted by siRNAs or treated with a TBX2/3-targeting repurposed drug, niclosamide, were infected with pseudoviruses. Infectivity was measured by luciferase assays and confocal microscopy.

RESULTS: The *in vivo* interaction between L2 and TBX2/3 was validated, pointing towards a function of this interaction during HPV infection. Pseudoviruses were successfully produced and used to show that TBX2/3 promote HPV infectivity. Importantly, the TBX2/3-targeting drug, niclosamide, inhibited HPV infection.

CONCLUSION: This study identifies TBX2 and TBX3 as novel targets for prophylactic and therapeutic treatment of HPV infection. It further demonstrates that niclosamide is a promising TBX2/3 targeting drug that can be repurposed to inhibit HPV infection.

T03 - IMPLEMENTATION RESEARCH

Abstract Number: T03-1

Introduction and Coverage of Human Papillomavirus Vaccination Programmes in Africa: A Systematic Review

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Background: The introduction of the human papillomavirus (HPV) vaccine has the potential to reduce cervical cancer incidence by up to 90% globally. In Africa, where cervical cancer remains a leading cause of cancer-related morbidity and mortality among women, several countries have introduced or are planning to introduce national HPV vaccination programmes. However, substantial disparities persist in vaccine rollout, coverage, and delivery strategies across the continent.

Aims: This systematic review evaluates the introduction and implementation of HPV vaccination programmes in Africa by assessing (i) the current status of HPV vaccine introduction and (ii) vaccination coverage since programme implementation.

Methods: A comprehensive literature search was conducted using PubMed, CINAHL, and Scopus databases, supplemented with data from the World Health Organization/UNICEF Electronic Joint Reporting Form (eJRF). Studies published from 2010 onwards were included and reviewed for information on HPV vaccine introduction, delivery strategies, target age groups, and vaccination coverage. Data were synthesised using a narrative approach.

Results: Of the 54 African countries, only 24 (43%) reported data on HPV vaccination programmes, with Rwanda being the first country to introduce the vaccine in 2011. Most programme introductions occurred between 2018 and 2019. School-based delivery was the predominant strategy and was associated with higher vaccination coverage. In many African settings, early sexual debut is common, underscoring the importance of timely vaccination. Despite this, coverage rates varied considerably across countries: more than 70% of girls aged 9–14 years received at least one dose of the HPV vaccine, while approximately 60% completed the full two-dose schedule.

Conclusion: Although progress has been made, HPV vaccine coverage in Africa remains below global targets. School-based delivery strategies have demonstrated effectiveness; however, enhanced social mobilisation, integration with broader healthcare services, and strengthened national immunisation systems are required to improve uptake. Addressing barriers such as cultural perceptions, logistical constraints, and funding limitations is essential to achieving the World Health Organization's cervical cancer elimination goals.

Abstract Number: T03-2

Home- Versus Hospital-Based HPV Self-Sampling for Cervical Cancer Screening in Rural West Region of Cameroon: a Cluster Randomised Controlled Trial

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Background: Improving cervical cancer (CC) screening uptake is crucial to achieve its elimination. We aimed to compare the effectiveness of home- vs hospital-based self-sampling for Human Papilloma Virus (HPV) testing in increasing participation in complete CC screening in a low resource setting.
Methods: This was a cluster-randomized controlled study conducted in Dschang Health District, Cameroon. Twenty rural health areas (HAs) were randomized unblinded. In ten HAs assigned to the home-based arm, a Community Health Worker (CHW) and a nurse went door-to-door counseling and offering to eligible women vaginal self-sampling for HPV testing. HPV-positive women were invited for triage and treatment at Dschang Hospital (DH). In the ten HAs of the hospital-based arm, a CHW went door-to door counseling and issuing invitation cards to attend screening at DH. Included participants were women 30 to 49 years old non pregnant, with no hysterectomy and who signed an informed consent. The primary outcome was completion of full screening, comprising primary HPV testing, triage of HPV-positive women with visual assessment of the cervix, and treatment of pre-cancerous lesions within three months of inclusion. Data was analyzed per intention to treat. The study was approved by Cameroonian Ethics Committee. ClinicalTrials.gov registrationNCT06166420.
Findings: 1089 out of 1112 (98%) included participants were tested for HPV in the home-based arm against 152 out of 967 (15.7%) in the hospital-based arm (p<0.001). High-risk HPV test positivity was 17.3% in home-based arm against 24.3% in hospital-based arm (p=0.03). In the home-based arm, 77(40.9%) of HPV positive women did not proceed to triage. 1010 (90·8%) participants in the home-based arm completed full screening against 152 (15.7%) in the hospital-based arm (p<0.001). The home-based intervention had a significantly higher likelihood of completing full CC screening with a risk ratio of 5.78 (95% CI 4.87 to 6.85), p <0.001. Cellular Intra-epithelial Neoplasia of grade 2 and more (CIN)2+ lesions were present in 0.9% HPV positive women in home-based arm versus 8.4% in hospital-based (p=0.049). CC screening coverage of women 30 to 49 years was 69.6% in home-based arm against 12.9% in hospital-based arm.
Interpretation: The home-based strategy achieved broad community screening coverage close to 70% despite the challenge of loss to follow-up, whereas the hospital-based strategy yielded a higher rate of high-risk HPV and CIN2+ detection. Our results will guide various stakeholders.

Abstract Number: T03-3

Detection of Hepatitis C Virus Using a Gold Nanoparticle-Based Colorimetric Kit: A Rapid and Low-Cost Approach

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Hepatitis C virus (HCV) is a positive-sense, single-stranded RNA virus, discovered in 1989 and classified within the *Flaviviridae* family. It is characterized by a high replication rate, with an estimated production of up to 10^{12} virions per day, and a highly variable genome resulting from the low fidelity of its RNA polymerase. There are six major genotypes and over 50 subtypes, posing a significant challenge for accurate diagnosis and effective treatment. The World Health Organization (WHO) estimates around 50 million chronic carriers globally and approximately 1 million new infections each year. Conventional laboratory techniques for HCV diagnosis include serological and molecular tests (NAT, such as RT-PCR), which, despite their sensitivity, present limitations such as high cost and the need for specialized personnel. In this context, the aim of this study was to develop a simple, rapid, selective, sensitive, and low-cost colorimetric biosensor based on gold nanoparticles (AuNPs) for the detection of HCV-specific oligonucleotides in saliva samples. AuNPs were synthesized using the Turkevich-Frens method, with tetrachloroauric acid and trisodium citrate under controlled temperature conditions, yielding the characteristic red coloration. Transmission electron microscopy (TEM) characterization revealed spherical particles with an average diameter of 20 nm. Thiolated oligonucleotide probes were immobilized onto the AuNPs, and the tests performed in quintuplicate included microtubes containing AuNPs, probes, complementary targets, and negative controls. Hybridization at 66.2 °C for 20 minutes, followed by cooling and the addition of trisodium chloride, allowed for visual differentiation: a red color indicated hybridization (absorbance at 530 nm), while blue color in the controls (with peaks between 600–620 nm) resulted from nanoparticle aggregation. Preliminary results highlight the strong potential of the biosensor as a promising, accessible, and effective diagnostic tool for HCV, with clear prospects for clinical validation.

T04 - VIRAL HEPATITIS

Abstract Number: T04-1

Targeting HBx-Driven HBV Replication: Inhibitory Effects of Punicalagin, SC75741 in Cell Culture Models

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Background: The Hepatitis B Virus X protein (HBx) is a multifunctional regulatory protein essential for HBV replication, known to modulate viral gene expression from the cccDNA template and interact with host cellular pathways, thereby contributing to the pathogenesis of hepatocellular carcinoma.
Aim: To evaluate the antiviral effect of Punicalagin, SC75741 on HBV replication and transcription in vitro, focusing on its impact on HBx expression, HBV RNA, HBV DNA, and cccDNA stability using differentiated HepaRG and HepG2-NTCP cell models.
Methods: HepG2, HepG2-NTCP, and differentiated HepaRG cells were used to study HBx expression and antiviral drug response. HBx was induced in Tet-inducible cell lines using doxycycline. In parallel, HepG2-NTCP and HepaRG cells were infected with HBV. Cells were treated with Punicalagin (1–10 µM), SC75741, Ledipasvir, or Dicoumarol as a control for up to 6 days. HBx protein levels were assessed via Western blot. HBV replication markers (HBsAg, HBeAg, HBV RNA, and DNA) were measured using ELISA and RT-qPCR.
Results: Punicalagin treatment significantly inhibited HBeAg secretion and reduced total intracellular HBV DNA levels by approximately 50%. A moderate (~20%) suppression of 3.5 kb HBV RNA, which encodes pregenomic and precore transcripts, was also observed. However, no effect on cccDNA levels was detected, indicating that Punicalagin's antiviral activity is likely mediated through transcriptional or post-transcriptional mechanisms rather than direct interference with the viral minichromosome. In contrast, Ledipasvir (1 µM) showed no measurable antiviral effect in this model. Dicoumarol demonstrated limited utility due to cytotoxicity associated with higher DMSO concentrations required for solubilization.
Conclusion: Punicalagin demonstrates partial antiviral activity against HBV in vitro, in a dose dependent manner, primarily by reducing viral antigen secretion and RNA/DNA replication without affecting cccDNA stability. These findings support further exploration of Punicalagin as a potential HBx-modulatory compound with therapeutic implications for functional cure strategies.

Abstract Number: T04-2

Evaluation of a single assay for the surveillance of multiple viral pathogens using Next Generation Sequencing

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The emergence and re-emergence of viral pathogens has caused a high burden of infectious diseases. This significantly impacts low- and middle-income countries, where the cost of reagents and limited local distributors pose a challenge. This highlights the necessity to find a method that can detect these viral pathogens and find a cost-effective genomic surveillance method. This study tested a miniaturized next-generation sequencing (NGS) approach using a Viral Surveillance Panel (VSP). It was first evaluated for performance on positive controls using full, half, and quarter reagent volumes. The miniaturized reagent volume approach was then used on clinical samples to see if the from the full reaction and the reduced reagent volumes. However, genome coverage decreased slightly in clinical samples with low viral loads and Ct scores. Identification of all target pathogens was successful, and the quarter-volume protocol had a success rate of 75% on clinical samples, while maintaining the same quality with the miniaturized reagent volume approach the number of samples that could be processed with a kit was quadrupled. Our results provide evidence in support of adopting these low-volume protocols to further genomic surveillance in resource limited environments.

Abstract Number: T04-3

Identifying of HBV DNA in liver tissues of chronic hepatitis and hepatocellular carcinoma to study the Hepatitis B virus silent infection in Egyptian patients

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Hepatitis B virus (HBV) infection comprises one of the most frequent causes of adult morbidity and mortality in Egypt. The presence of HBV genomes in the absence of HBV surface antigen (HBsAg) individuals is referred to as occult HBV infection. This study aimed to detect HBV integration in HBsAg negative chronic hepatitis and hepatocellular carcinoma (HCC) cases by extraction of DNA from tissue and serum of chronic hepatitis and HCC cases to find out the silent HBV infection. Subjects and Methods: Seventy-three patients divided into three groups; group I: included fifty-two sera samples for hepatocellular carcinoma (HCC) patients with negative HBsAg and positive HBcAb; of which fourteen liver tissues samples were taken (group I). Nine chronic hepatitis patients with negative HBsAg and positive HBcAb (group II) were studied in both liver tissue and serum sample. Twelve

chronic hepatitis patients with positive HBsAg and positive HBcAb were included as a control group. DNA from those groups has been separated from tissue sections that have been mounted on glass slides. Results: The present study showed that 1/52 (1.9%) of the patients with HCC were positive for HBV DNA in the serum while all the 52 HCC patients were negative for HBV DNA in the liver tissue. Conclusion: The present showed that 1/52 (1.9%) of the patients with HCC were positive for HBV DNA in the serum. The explanation for this is the low endemicity of HBV infection in Egypt which differ HBV DNA detection process.

Abstract Number: T04-4

Association of HBx mutations with clinical outcomes in chronic HBV-HIV coinfected patients in South Africa

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Background: The hepatitis B virus X protein (HBx) plays a crucial role in HBV transcription, replication, and persistence through modulation of covalently closed circular DNA (cccDNA) activity. While mutations in HBx have been reported, their effect on protein stability and viral replication dynamics remains under-investigated, especially in African cohorts.

Materials and Methods: HBx sequences from 59 HBV/HIV co-infected patients sequenced with Oxford Nanopore technology were analysed with command pipeline on Ubuntu Linux and Geneious Prime to identify mutations. The effect of mutations on protein stability was assessed using four in silico prediction tools: DynaMut2, mCSM, SDM, and STRUM, which estimated changes in Gibbs free energy ($\Delta\Delta G$). Mutation frequencies were compared across clinical groups defined by HBeAg status, HBcAb, Anti-HBe, ALT, HBV & HIV viral load. Spearman's correlation tested the relationship between $\Delta\Delta G$ and $\log_{10}$-transformed HBV viral load. Fisher's exact and Mann-Whitney U test assessed associations between mutation presence and serological status as well as with continuous variables (ALT, HBV & HIV Viral load). Univariate and multivariate model analyses were conducted in SPSS to identify any key mutations associated with viral markers.

Results: Among identified HBx mutations, C26R (78.8%), R32G (32.7%), and A36T (30.8%) were the most prevalent. The presence of the C26R mutation was significantly associated with higher HBV viral load (median $\log_{10}$ IU/mL: 4.5 vs. 2.6, $p < 0.01$). In contrast, R32G and A36T were associated with significantly lower HBV viral loads ($p < 0.05$ for both). No significant association was observed between these mutations and serological markers. Univariate and Multivariate analysis did not reveal any associations between single mutations or combinations of mutations, with serological markers. Linear regression model revealed C26R and E80A mutations to be significantly associated with higher HBV viral load, and Q87L to be associated with higher ALT levels. Spearman's correlation showed a moderate positive but statistically significant relationship between $\Delta\Delta G$ and viral load (r=0.34, p=0.019), suggesting that protein stability may influence viral replication efficiency.

Conclusion: Specific HBx mutations show differential effects on HBV viral replication. C26R may enhance viral replication, while R32G and A36T are associated with decreased viral load, potentially indicating a functional alteration in HBx activity. Our findings indicate that specific mutations in the HBx gene, particularly those enhancing protein stability, may contribute to increased viral replication and liver inflammation in HIV/HBV co-infection. These findings highlight the relevance of HBx variability in influencing HBV pathogenesis and treatment response. Further investigation is needed to determine the mechanistic impact of these mutations on cccDNA transcriptional activity and HBx-host interactions.

T05 - GAMMA-HERPESVIRUSES AND RELATED CANCERS

Abstract Number: T05-1

Clinicopathological Features and HHV-8 Expression in Gastrointestinal Kaposi Sarcoma: Over 20 Years of Experience at a Tertiary Centre in Tshwane

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Background and Objectives: Kaposi Sarcoma involving the gastrointestinal tract (GIT KS) represents an uncommon manifestation of this HHV-8 driven vascular tumour, occurring predominantly in individuals with HIV infection. Early disease is often clinically silent, and subtle mucosal lesions go undetected, typically occurring at advanced stages. Often accompanied by nodal spread and features associated with poorer clinical outcomes. This study looks to describe the clinicopathological characteristics of GIT KS diagnosed at a tertiary institution with the intention of identifying patient groups at heightened risk and informing strategies for improved diagnostic evaluation and clinical management.

Methods: This retrospective, quantitative case series analysed all GIT KS cases diagnosed between January 1, 2004, and December 31, 2024, using data obtained from the Corporate Data Warehouse, National Health Laboratory Service's. Relevant demographic, clinical, and histopathological data was collected using a specialized tool, recorded in Microsoft Excel, and analysed using STATA version 18 software.

Results: A total of 25 GIT KS cases were included; with a predominance of males 60% (n=15). The overall mean age was 42.44+-11.91 years, (range 16-64 years). with the mean ages of 45.67+-11.31 for males and females was 37.6+-11.68 years. Majority of patients were HIV positive (84%), CD4 counts were recorded for 76% of cases, of which, 32% had counts below 200 cells/ml. Viral load were available for 56% of patients, with the lowest recorded value at <50 copies/mL. The frequently involved site was gastric at 48% (n=12). Gastrointestinal bleeding (16%) was the most common presentation Overall, 72% were on ARV and, 60% tested positive for HHV-8, 8% negative, and 32% had no test performed.

Conclusion: This study highlighted that GIT KS predominantly affects middle-aged males, most commonly the stomach often presenting with gastrointestinal bleeding. Most were HIV -positive with significantly low CD4 counts, underscoring need for clinical awareness on GIT KS in immunocompromised populations to enable early detection and timeous management.

Abstract Number: T05-2

Immunohistochemical analysis of Kaposi Sarcoma Herpesvirus in Paediatric Kaposi Sarcomas: A 21-year study at a Tertiary Laboratory in Tshwane

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Introduction: Paediatric Kaposi sarcoma (KS) is a multifocal vascular tumour linked to HHV-8 infection and occurs mostly in children living with HIV. Its clinical features vary widely, it resembles other lesions, making clinical diagnosis challenging. Immunohistochemistry, especially HHV-8 staining, is therefore essential for precise diagnosis. This study investigated the prevalence and histopathological (morphology and IHC) characteristics of paediatric KS to elucidate pathways crucial for patient management.

Methodology: The study was a retrospective, descriptive cross-sectional study that was carried out on paediatric KS cases (0–18 years) diagnosed between January 2004 and December 2024. Demographic and clinical data was retrieved from the laboratory information system. Archival formalin-fixed, paraffin-embedded blocks were used to section tissue for IHC biomarkers (HHV-8, D2-40, CD117, p53, and Ki-67) analysis. Data was captured on Microsoft Excel and analysed using STATA v18, Fisher's exact test was used to evaluate correlations between clinicopathological and demographic characteristics.

Results: During the study period, paediatric KS accounted for 2.7% of the 1,095 KS cases. Among the 21 paediatric cases analysed, females slightly predominated (52%), with a mean age of 11.1 ± 4.6 years. Most children (86%) were HIV-positive, and 43% were receiving antiretroviral therapy. Cutaneous involvement was most common (71%), lower limbs (24%) was most common site, and 76% reported multifocal disease. Histologically, the nodular stage predominated (62%), and the classical KS (90%) was most common histopathological variant. HHV-8 IHC biomarker was positive in 76.2%, while P53, D2-40, CD117, and Ki-67 showed low expression (<50%). No significant associations were found between histopathological stage and clinicopathological features (p > 0.05).

Discussion and Conclusion: Paediatric KS (PKS) is common in older children with minimal sex variations. Immunohistochemistry analysis of HHV8 remain relevant in diagnosis of KS. Overall, the study indicated that the histopathological stage and variant do not influence the immunohistochemical biomarker expression.

Abstract Number: T05-3

The Role and Implication of MXRA7 in Viral Infection-mediated Inflammatory Carcinogenesis

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Background and aim: Transcripts of the now called Matrix Remodeling Associated 7 (MXRA7) were first cloned around 2000 in a study addressing the host response to hepatitis B virus. The biological features of MXRA7 remained unattended for about 15 years, till Wang teams started working around this gene, and the biomedical activities and significance of MXRA7 were disclosed gradually in inflammatory neovascularization, chemical liver injury, cutaneous injury healing, bone marrow stromal cell differentiation, and leukemia transformation. However, MXRA7's potential role in viral infection or oncogenesis remained less studied, and this study was to investigate into this question.
Methods: Agonistic activation of TLR7 with imiquimod in human keratocytes HaCat was used to mimic viral stimulation, and expression and localization of MXRA7 proteins cells were monitored with RT-PCR and immunofluorescence respectively. Skin cancer cell line A431 was used for identifying chromosomal targets of MXRA7 proteins in cell nuclei using ChIP in couple with high throughput sequencing. Identified candidate genes were further confirmed with the promoter reporting system.
Results: Upon activation of TLR7 in HaCat cells, MXRA7 proteins translocated into nuclei. ChIP assay revealed that MXRA7 proteins targeted genes closely related with cancer biology, with EGFR as the nodule of several pathways. Experimental and virtual analysis confirmed that EGFR gene was one of the main target genes responding to MXRA7 signals.
Conclusion: Preliminary data suggested that MXRA7 gene might pose an important mediator of inflammatory carcinogenesis. The significance of this finding in management of viral oncology will also be discussed.