

Contemporary and Prospective HIV Treatment and Prevention Strategies

HIV Symposium at the Kirby Institute

Event information

Date	Thursday, 20 July 2023
Time	9am - 5pm AEST
Location	Berg Family Seminar Room, Kirby Institute, UNSW Sydney And virtual participation



Sponsored by Gilead Sciences

Location

The HIV symposium takes place at the Kirby Institute, UNSW Sydney. The Kirby Institute is located on level 6 of the Wallace Wurth building of the [UNSW Kensington campus](#) on the corner of High Street and Botany Street.

Address [Level 6, Wallace Wurth Building](#)
High Street, UNSW Sydney
Kensington NSW 2052

Telephone +61 (2) 9385 0900

Parking

All day casual visitor parking is generally available on the UNSW Kensington campus on the top levels of the multi-story carpark located via Gate 14 Barker Street and Gate 11 Botany Street Kensington (the Botany Street carpark is closest to the Wallace Wurth Building). All drivers are to register and pay for casual parking with [CellOPark App](#).

Public transport

The Wallace Wurth building is located next to the light rail stop “UNSW High Street” of the L2 line which connects the UNSW campus to both Circular Quay and Central Station.

Symposium room

The symposium takes place in the Kirby Institute seminar room on level 6 of the Wallace Wurth building. After exiting the elevator and walking around the elevators, one faces the seminar room. Please note that no food and drinks are allowed in the seminar room other than water. There is filtered water available to refill a water bottle.

Program overview

Time	Speaker	Title
8.30am	Welcome tea and coffee	
9.00am	Aunty Lola Ryan	Welcome to Country
9.10am	Anthony Kelleher	Welcome and outline of the day
Public Health Session		<i>Anthony Kelleher, Bridget Haire, Louise Causer</i>
9.20am	Meg Doherty	WHO's approach to ending AIDS and the epidemics of viral hepatitis and STIs by 2030: focus on new science and recommendations
10.00am	Skye McGregor	Australia's progress toward ending HIV: 2022 national surveillance data
10.20am	Short break	
10.25am	Pande Putu Januraga	Advancing PrEP Implementation in Indonesia: An Update on the Current Program
10.45am	Aaron Cogle	Treatment for Health and Prevention: Perspectives from the HIV Positive Community
11.05am Morning tea break – Concurrent Networking Sessions		
Basic Science Session 1		<i>Erica Vine, Kirstie Bertram</i>
11.40am	Katharine Bar	The impact of early ART initiation on autologous neutralizing antibody responses
12.00pm	Miles Davenport	Which virus rebounds after treatment interruption in SIV infection?
12.20pm	Afam Okoye	Targeting the early viral intercept: Manipulating post-ART immune host viral dynamics for HIV cure
12.40pm	Sarah Palmer	Investigating the mechanisms for post-intervention control
1.00pm Lunch – Concurrent Networking Sessions		

Basic Science Session 2 <i>Mee Ling Munier, Chansavath Phetsouphanh, Anthony Kelleher</i>		
2.00pm	Lishomwa (Lish) Ndhlovu	Journey Across Three Pandemics: Searching for vulnerabilities for intervention
2.20pm	David van Bockel	Defining the distribution of the potentially replication-competent HIV-1 reservoir during raltegravir-containing antiretroviral therapy
2.40pm	Sulggi Lee	Rapid Two-Phase Decay of Intact and Defective HIV DNA during Acute Treated HIV Infection, which is Accelerated with Earlier ART Initiation
3.00pm Short break		
3.20pm	David M. Margolis	The Effects of HIV-1 Antigen Expanded Specific T Cell Therapy (HXTC) and Vorinostat on Persistent HIV-1 in People Living with HIV on Antiretroviral Therapy
4.00pm	Anthony Kelleher	Closing comments
4.10pm Networking drinks		

Speakers and talks

Meg Doherty: WHO's approach to ending AIDS and the epidemics of viral hepatitis and STIs by 2030: focus on new science and recommendations

About the speaker:



Meg Doherty has been the Director of the Department of Global HIV, Hepatitis and Sexually Transmitted Infections Programmes at WHO since February 2020. Dr Doherty was previously the Coordinator of Treatment and Care in the Department of HIV at WHO Headquarters. She has more than 25 years of experience in HIV and infectious diseases, including leading WHO's normative and programmatic work on expanding HIV treatment to all and reducing inequalities in access to the most effective antiretrovirals for people living with HIV. Dr Doherty spent 10 years in low- and middle-income countries, advising ministries of health and international partners on implementing comprehensive HIV and infectious disease programmes.

infectious disease programmes.

Twitter: @mdoherty_hiv

Skye McGregor: Australia's progress toward ending HIV: 2022 national surveillance data

HIV notifications in Australia have declined in recent years, demonstrating the impact of pre-exposure prophylaxis, treatment as prevention, and high testing coverage. Declines since 2020 were also likely influenced by the COVID-19 pandemic. Skye will present the latest Australian HIV surveillance data, for the 2013 to 2022 period. This will provide insight into Australia's progress with elimination of HIV transmission, and where further efforts are required, to ensure equity of the Australian response.

About the speaker:



Skye McGregor is an epidemiologist and lead of the Surveillance Innovation Research Group at the Kirby Institute. She is an emerging national leader in the development, analysis, and interpretation of national sexually transmissible infections (STIs) and blood borne viruses (BBVs) surveillance datasets. Her research group leads production of the national STI and BBV annual surveillance reports for Australia, which provide a comprehensive analysis of HIV, viral hepatitis and sexually transmissible infections in Australia. Skye has more than 10 years' experience in undertaking innovative surveillance research, with a focus on treatment, prevention and health equity.

Twitter: @skye_mcgregor

Pande Putu Januraga: Advancing PrEP Implementation in Indonesia: An Update on the Current Program

The recent modeling of the HIV epidemic in Indonesia has revealed persistently high rates of HIV infection, particularly among key populations, with the MSM group being significantly affected. Drawing upon successful experiences from other settings in curbing transmission, Indonesia has adopted Pre-Exposure Prophylaxis (PrEP) as a crucial strategy. The implementation of PrEP began as a pilot initiative in high-priority districts in early 2022, and the intention is to expand the program to encompass 100 priority districts from 2024 to 2026. This presentation aims to delve into the current progress, challenges, and barriers encountered during the implementation phase and outline the strategic approaches to further advance and maximize the program's impact on the HIV epidemic in Indonesia.

About the Speaker:



Dr. Pande Putu Januraga is a professor of Public Health at Udayana University's Faculty of Medicine in Bali, Indonesia. He is the head of the Center for Public Health Innovation (CPHI), a research center devoted to developing and evaluating innovative methods for implementing public health programs. His research interests include strategies for HIV prevention and treatment of marginalized and affected communities in Bali and Indonesia. He also serves as a member of the Indonesian Ministry of Health's HIV expert panel.

Twitter: @januraga

Aaron Cogle: Treatment for Health and Prevention: Perspectives from the HIV Positive Community

People with HIV have long been the quiet heroes of Australia's HIV response. The incorporation of changing treatment technologies into their daily experience has meant healthier, longer lives and, as research has now shown, significantly restrained onward transmission in Australia. New developments in the treatment space will require yet more engagement from positive people.

Despite the importance of successful treatment to current and future prevention efforts, many in the HIV positive community report feeling made invisible in various ways. The preventative functions of ARV treatments are often emphasised to the exclusion of personal wellbeing and the complex health needs of people with HIV. Herculean, community-wide efforts to secure treatment adherence, and the work of being on treatments with HIV, are obscured behind mere indicators of treatment uptake. The potential of the U=U message remains stubbornly unrealised. And, biomedical prevention threatens to erase prevention discussions completely.

Against this backdrop, it is research that holds the potential to enhance the HIV response by understanding the experiences and contributions of PWHIV beyond mere prevention.

About the speaker:



Aaron Cogle is Executive Director of the National Association of People Living with HIV (NAPWHA). He has worked in the HIV sector for nearly 20 years and has an academic background in Policy, Corporate Law and, increasingly, how HIV intersects with the Criminal System.

Twitter: @napwha

Katharine Bar: The impact of early ART initiation on autologous neutralizing antibody responses

Initiation of antiretroviral therapy as soon as possible after HIV diagnosis limits disease impact in people living with HIV, but has unclear implications for HIV-specific immune responses. Here, we characterized the kinetics and determinants of autologous nAb development following acute and early ART initiation. Rooting our assessment to autologous transmitted/founder (TF) or early escape variant plasma virus populations sequenced at the time of early ART initiation, we mapped binding, tier 1 and autologous nAb responses in a cohort of 23 individuals diagnosed and starting ART in acute and early HIV disease. Our results suggest a threshold of viremia necessary to foster nascent autologous nAb responses. If triggered prior to ART initiation, autologous nAb responses then further evolve in breadth and potency on suppressive ART.

About the Speaker:



Dr. Katharine Bar is an Associate Professor of Medicine at the University of Pennsylvania within the Division of Infectious Diseases, and Director of the Penn Center for AIDS Research Virus and Reservoirs Core. She is a physician-scientist who studies the basic mechanisms and translational impact of virus transmission, pathogenesis and persistence in HIV, Hepatitis C, and HSV infections. Her lab's recent work is focused in three related areas: i) understanding mechanisms of HIV persistence and reactivation, ii) developing new antibody-based interventions, and iii) leveraging novel nonhuman primate model systems to test novel interventions. Clinically, she practices as an Infectious Disease specialist, with a focus on HIV medicine.

Twitter: @katharine_bar

Miles Davenport: Which virus rebounds after treatment interruption in SIV infection?

HIV provirus is readily detectable in the blood during ART, and replicating virus rapidly rebounds after treatment interruption. However, provirus detected in blood seems poorly represented in rebound virus. One explanation for this is that proviral DNA and rebounding virus are laid down at different times, and / or found in different sites. Another explanation is that the immune system 'sieves' the rebounding virus to allow only the most escaped virus to replicate. We have used bar-coded SIVmac239 model of infection and treatment interruption to study the timing of establishment of the rebounding virus and the impact of CD8 T cell pressure on rebounding virus. We find that the majority of rebounding virus is laid down around the time of ART initiation and that CD8 T cell sieving can act to suppress the rebound of non-escaped virus.

About the speaker:



Professor Miles Davenport is head of the Infection Analytics Program at the Kirby Institute, at the University of New South Wales, in Sydney Australia. He leads a team of applied mathematicians applying statistical and computational approaches to understand host-pathogen interactions in SARS-CoV-2, HIV, and malaria. He has a wide variety of clinical and experimental collaborations both within Australia and overseas and his work aims to integrate experimental data and modelling. His seminal work defining an immune correlate for protection from SARS-CoV-2 infection has been cited in multiple statements / guidelines on SARS-CoV-2 infection and vaccination.

Twitter: @IAP_Sydney

Afam Okoye: Targeting the early viral intercept: Manipulating post-ART immune host viral dynamics for HIV cure

Given that many examples of immune control of HIV and its simian counterpart SIV infection are either known, or strongly suspected to be CD8+ T cell-mediated, therapeutic strategies designed to exploit CD8+ T cell immunity hold great promise towards achieving virologic remission following antiretroviral therapy (ART) interruption. Our studies have indicated that in the setting of post-ART rebound of SIVmac239 in rhesus macaques, pre-established CD8+ T cell responses can provide for a more effective intercept of recrudescing infection than in primary infection, but that this immune interception comes well after systemic viral spread, too late for stringent control. This suggests that delays in coordinating an effective CD8+ T cell response to rebound provides the infection with the dynamic "head start" it needs to ultimately evade the host response. Here we will discuss strategies aimed at changing the dynamic balance between the host immune system and rebounding virus so as to provide an earlier and more effective interception of post-ART recrudescing infections.

About the speaker:



Afam Okoye is an Associate Professor at the Vaccine & Gene Therapy Institute, and the Division of Pathobiology and Immunology at the Oregon National Primate Research Center, Oregon Health & Science University (OHSU). He also has an affiliate appointment in the Department of Molecular Microbiology and Immunology, OHSU School of Medicine. He received a BSc in Microbiology from the University of Nigeria and MSc in Biotechnology from Nottingham Trent University before completing his doctoral studies at the University of Glasgow. He is a trained immunologist with extensive experience in the investigation of nonhuman primate models of HIV/SIV pathogenesis and immunity. His current research involves understanding the barriers to HIV eradication and developing therapeutic interventions aimed at achieving durable remission from HIV replication after ART withdrawal.

Sarah Palmer: Investigating the mechanisms for post-intervention control

Replication-competent viruses are the main barrier to HIV-1 eradication as these contribute to viral rebound if therapy is interrupted. Therefore, the genetic characterization of plasma-derived HIV-1 RNA from viremic individuals during pre-therapy or after treatment interruption may identify virus-related mechanisms behind a delay in viral rebound during an analytical treatment interruption. We applied a full-length HIV RNA assay to samples from people living with HIV during pre-therapy and after they stopped their therapy. We found that an average of 65% of plasma-derived HIV-1 RNA genomes were genetically-intact during pre-therapy. We also found that plasma-derived viral genomes from two participants were genetically-distinct during the treatment interruption timepoints where a delayed viral rebound was observed. The emergence of these distinct viral variants during an analytical treatment interruption may be associated with transient virological control. This viral control is not attributed to impaired viral replication caused by known CTL-escape or drug-resistance mutations. However, for one participant who exhibited transient virological control, we identified mutations in predicted T cell epitopes within Pol, Gag, and Nef which may affect viral replication, enhance CD8 T cell response and contribute to the delay in viral rebound. Investigating the interplay between the virus and the host immune cell response will provide insights as to how some individuals living with HIV control the virus during an analytical treatment interruption.

About the speaker:



Professor Sarah Palmer is the Co-Director of the Centre for Virus Research at The Westmead Institute for Medical Research and Professor in the Faculty of Medicine and Health, at The University of Sydney School of Medicine.

Prior to taking up this position in early 2013, she was a Senior Researcher at the Swedish Institute for Communicable Disease Control and Karolinska Institutet, in Stockholm, Sweden from 2008 to 2012. From 2000 to 2008, she headed the Virology Core Facility of the HIV Drug Resistance Program, National Cancer Institute, US National Institutes of Health, where she led efforts to develop and perfect

highly-sensitive assays such as the single-copy assay and single-cell sequencing assay which provide new insights into HIV pathogenesis and persistence during long-term suppressive therapy.

Her principal areas of research interest focus on molecular and medical virology and the application of innovative techniques and assays which provide new insights into disease pathogenesis and treatment, especially for HIV and COVID-19. Her current work focuses on understanding the genetic characteristics and dynamics of persistent HIV across a range of tissues and cells to guide and assess treatment interventions designed to reduce persistent HIV reservoirs and inform HIV eradication strategies. Dr. Palmer has her Ph.D. in Medical Sciences (Virology) from the Karolinska Institutet, and conducted her post-doctoral studies at the Center for AIDS Research, Stanford University Medical School.

Lishomwa (Lish) Ndhlovu: Journey Across Three Pandemics: Searching for vulnerabilities for intervention

Our HIV and emerging pathogens translational research program primarily centers on understanding how HIV affects the immune system in blood and tissues with a focus on discoveries that identify novel applications in balancing strategies to prevent, slow or eliminate HIV infection while optimizing quality of life outcomes. Our focus covers many disciplines including identifying neuro-immune pathways that are associated with brain injury in people living with HIV to illuminate the complex pathology surrounding these complications arising with HIV and aging. To study mechanisms that underly such complicated processes, our program has employed several novel innovations, and this has opened the door towards developing new therapeutic approaches to treat these co-morbidities. Epigenetics is the study of how behaviors and environment can cause changes in how gene are expressed and work in cells, but do not change an individual's DNA. These carefully orchestrated chemical reactions that activate and deactivate parts of the genes at strategic areas have been shown to be critical in normal brain function and this presentation will provide a synopsis of studies on how epigenetic mechanisms may be key events in HIV associated age-related complications with implications for HIV curative strategies.

About the speaker:



Lishomwa (Lish) Ndhlovu is Professor of Immunology in Medicine at Weill Cornell Medicine in the Division of Infectious Diseases. After completing medical training he received his PhD in Immunology from Tohoku University in Japan and pursued post-doctoral training at the University of California San Francisco. His research is dedicated to confronting the challenges of HIV and aging and has developed specific expertise and strategies to prevent, slow or eliminate organ specific complications.

David van Bockel: Defining the distribution of the potentially replication-competent HIV-1 reservoir during raltegravir-containing antiretroviral therapy

Anti-retroviral therapy (ART) is not curative, due to the establishment of a cellular viral reservoir that contains replication competent HIV-1 during acute/primary HIV-1 infection (PHI). Persistence of viral replication in the presence of successful ART may then become more reliant upon the proliferation, differentiation and expansion of cells already infected with HIV-1 to maintain infection and remain as a barrier to cure. Evidence of low-level viral replication on ART is supported by an increase in episomal HIV-1 2-long terminal repeat positive (2LTR+) products upon intensification on treatment with the integrase inhibitor, raltegravir (RAL); these may persist in aviremic individuals for up to three years. It is important to clarify the presence and importance of 2LTR+ genomes detected using quantification of the viral reservoir, as a potential overestimate of the replication competent reservoir. We characterise the viral reservoir using near-full length proviral sequencing (FLIPS) with a novel duplex PCR, to detect viral 2LTR+ regions from the CD4+ T-cell subsets of a cohort of PHI and chronic (CHI) patients receiving RAL over three years. Defects within genes of full-length viral products were evenly distributed (predominantly in gag, pol, env and tat, where multiple stop codons predicted antiviral activity), as was the presence of 2LTR among intact and defective genomes. Greater viral sequence homology for the PHI group (>99.6%) segregated cohorts and is consistent with successful ART. While minimal viral evolution occurred, clonal expansion of intact virus was evident from baseline and 2LTR+ virus was restricted to terminal external phylogenetic nodes, within this preliminary analysis.

About the speaker:



Dr David van Bockel is group lead of the viral sequencing group, within the Immunovirology and Pathogenesis Programme (IVPP) at the Kirby Institute. He is a basic scientist with strong translational research experience as lead laboratory investigator for multiple national and international clinical trials testing novel anti-inflammatory and immunotherapeutic small-molecule treatments for HIV co-morbidities. His current research involves understanding the impact of anti-retroviral therapies upon the viral reservoir, with the aim of improved clinical outcome.

Sulggi Lee: Rapid Two-Phase Decay of Intact and Defective HIV DNA during Acute Treated HIV Infection, which is Accelerated with Earlier ART Initiation

Lei Shi¹, Rebecca Hoh², Heather Hartig², Vivian Pae², Sannidhi Sarvadhavabhatla², Sophia Donaire², Greg Laird³, Michael Peluso², Jeffrey Martin⁴, Frederick Hecht², Christopher Pilcher², Timothy J. Henrich⁵, Jingshen Wang¹, Hiroyu Hatano⁵, Steven G. Deeks² and Sulggi A. Lee²

¹Department of Biostatistics, University of California Berkeley, Berkeley, CA 94110, USA. ²Department of Medicine, Division of HIV, Infectious Diseases & Global Medicine, University of California San Francisco, San Francisco, CA 94110, USA. ³AcceleVirDx, Baltimore, MD 21202, USA. ⁴Department of Biostatistics & Epidemiology, University of California San Francisco, CA 94158, USA. ⁵Department of Medicine, Division of Experimental Medicine, University of California San Francisco, San Francisco, CA 94110, USA.

Background: The HIV reservoir largely consists of “defective” virus. Several studies now suggest that HIV+ cells harboring “intact” (i.e., potentially replication-competent) provirus decay faster than defective provirus during long-term antiretroviral therapy (ART) suppression. However, there are limited data describing HIV reservoir decay rates during the first few months of treated acute HIV.

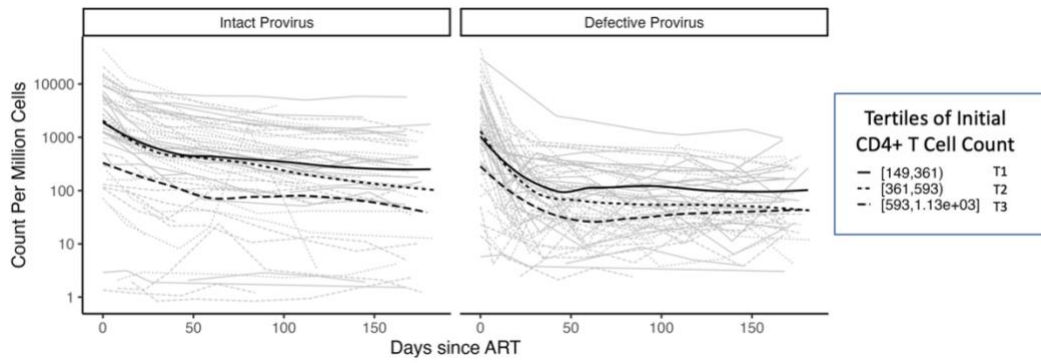
Methods: Individuals diagnosed with acute (<100 days) HIV were enrolled in the UCSF Treat Acute HIV study between 2015-2020. Participants were initiated on ART (tenofovir/emtricitabine+dolutegravir) and followed for 24+ weeks. Frequencies of intact vs. defective provirus were quantified using the intact proviral DNA assay (IPDA). Multivariate nonlinear general additive models included covariates for timing of ART initiation, initial CD4+ count, and pre-ART HIV RNA.

Results: A total of 67 (83% of screened) participants were enrolled. The proportions of Fiebig I, II, III, IV, V disease were 12%, 15%, 8%, 17%, and 48%. Median age was 30. The cohort was 98% male; 15% African-American, 30% Latino, 21% Asian, 33% Caucasian. Higher initial CD4+ T cell count and lower pre-ART plasma HIV RNA predicted more rapid decay of defective and intact HIV DNA. Plasma HIV RNA declined to <40 copies/mL after a median of 31 days of ART. Both intact and defective provirus decayed rapidly in the first 50 days (3.6% vs. 4.3%), followed by a slower decay after 50 days (1.1% vs. 1.8%). Earlier ART initiation (Fiebig I-III) was associated with steeper declines in both intact and defective HIV DNA. For each day delay in ART initiation, intact and defective HIV DNA decreased by 0.02% vs. 0.03% in the first 50 days and by 0.006% vs. 0.01% after 50 days of ART suppression.

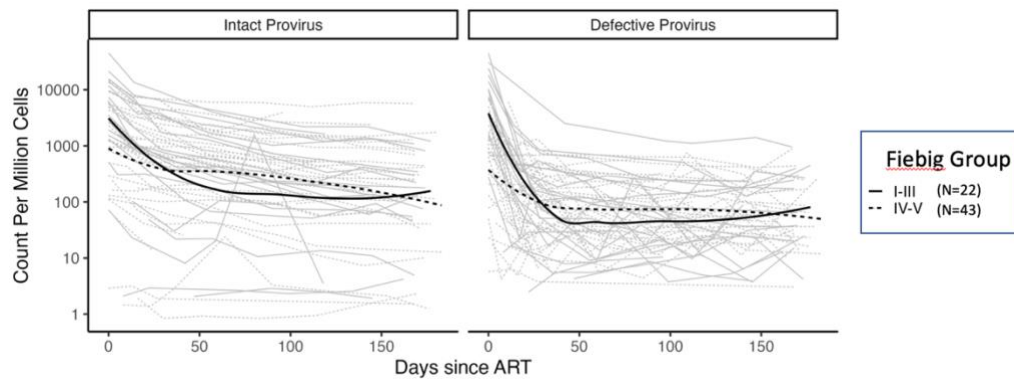
Conclusion: We observed a two-phase decay of intact and defective HIV DNA that was significantly faster for individuals with earlier ART initiation. Decay rates were accelerated with earlier ART initiation. These findings may reflect virologic (rapid decay after plasma viremia suppression) and/or immunologic (contraction of activated cells to memory cells) phenomena in individuals with very small reservoirs and relatively intact immune responses, which can be leveraged in future cure strategies.

Figure 1.

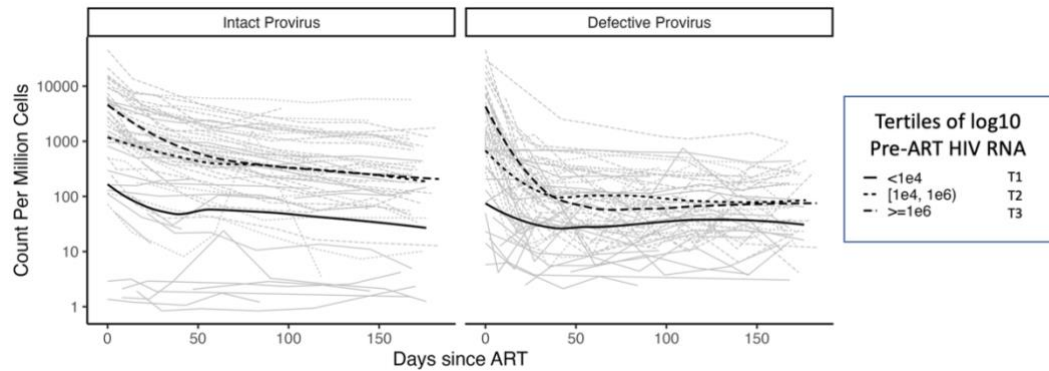
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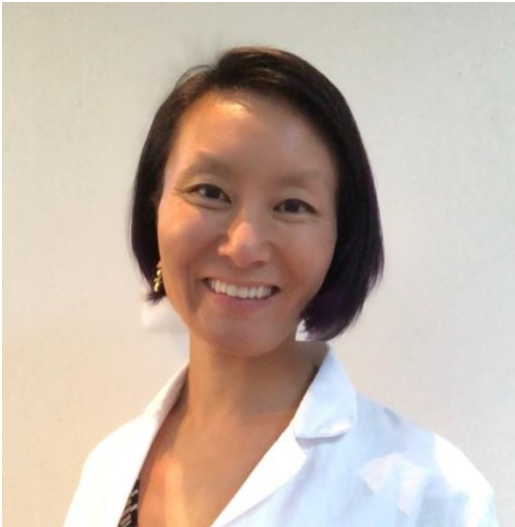
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About the speaker:



Sulggi Lee is an Associate Professor of Medicine in the Division of HIV, Infectious Diseases, and Global Medicine at the University of California San Francisco (UCSF). She earned her Bachelor's degree in Human Biology from Stanford University and her M.D./Ph.D. in Genetic Epidemiology from the University of Southern California (USC). She completed her Internal Medicine residency at Stanford Hospital and Clinics and her Infectious Diseases fellowship at UCSF. She is Principal Investigator of the UCSF Treat Acute UCSF Study, PI of the COVID-19 Host Immune Response Pathogenesis (CHIRP) study, Co-PI of the UCSF SCOPE HIV+ cohort, Co-PI of the UCSF AIDS Cancer Specimen Resource (ACSR), and Co-Director of the UCSF Center for AIDS Research (CFAR) Bioinformatics Core. She has

expertise in leading and conducting genetic epidemiologic research and translational HIV studies. Her lab investigates the role of host genetics and immunology in infectious diseases pathogenesis using high-throughput genetic sequencing and immunologic methods combined with multi-omic analytic approaches. The goal of her research is to improve clinical outcomes for human infectious diseases by leveraging unique clinical cohorts and utilizing advanced immunologic and genetic methods.

David M. Margolis: The Effects of HIV-1 Antigen Expanded Specific T Cell Therapy (HXTC) and Vorinostat on Persistent HIV-1 in People Living with HIV on Antiretroviral Therapy

Background: One approach to eradicate HIV is to interfere with mechanisms that maintain latency, and simultaneously enhance the clearance of infected cells without interrupting antiretroviral therapy (ART). The histone deacetylase (HDAC) inhibitor, vorinostat (VOR), can repeatedly induce the expression of latent HIV-1 in vivo, and allow clearance of infected cells in vitro. However, when paired with HIV vaccines or antibodies, this approach has not yielded substantial depletion of the latent reservoir in vivo. Adoptive T cell therapy has had dramatic success in the treatment of virus-related malignancies and infections following hematopoietic stem cell transplantation and has been adapted to produce ex-vivo expanded HIV-specific T cells (HXTCs), and other cell therapy products.

Methods: In this pilot study we administered VOR and HXTCs to antiretroviral (ART)-suppressed people with HIV (PWH). Six PWH received five infusions of 2×10^7 HXTCs/m² with VOR 400 mg every three days. Three PWH received five infusions of 10×10^7 HXTCs/m² with VOR. Leukapheresis was performed at baseline and after final HXTC infusion to measure the frequency of persistent HIV by Quantitative Viral Outgrowth Assay (QVOA) of resting CD4⁺ cells, cell-associated HIV RNA (rcaRNA), and intact HIV provirus assay (IPDA).

Results: Overall, PWH tolerated VOR and HXTCs, with only transient Grade 1 AEs related to study products. Biomarkers of serial VOR effect were detected in PBMCs, but evidence of enhanced antiviral activity in the total pool of circulating cells was not detected. One of 6 PWH exhibited a decrease in measures of persistent HIV after 2×10^7 HXTCs/m² infusions with VOR, and all three PWH exhibited such declines when 10×10^7 HXTCs/m² were given with VOR. However, most QVOA declines did not exceed 6-fold, a threshold required to definitively ($p > 0.05$) attribute QVOA decline to the study intervention, rather than assay variation.

Conclusions: These findings provide some support for the therapeutic strategy of HIV latency reversal and enhanced reservoir clearance, but the modest effects seen highlight the need for more effective latency reversal agents and clearance approaches that can be repeatedly employed to achieve the profound depletion of persistent HIV needed for clinical benefit.

About the Speaker:



David Margolis became interested in HIV as the pandemic emerged during his medical training. Trained at the Tufts School of Medicine and New England Medical Center, the Laboratory of Clinical Investigation at the NIH, and Program in Molecular Medicine at UMass, he has cared for people with HIV and studied the interactions between HIV and the host cell. For more than 2 decades, he and many collaborators have begun to understand the molecular basis of HIV latency, test drug and immunotherapy approaches to target persistent HIV, and develop the tools needed to cure HIV infection. A Sarah Graham Keenan Distinguished Professor of Medicine, Microbiology & Immunology, and Epidemiology at the University of North Carolina at Chapel Hill, he has led the NIH-sponsored Collaboratory of AIDS Researchers for Eradication (<http://www.delaneycare.org>) since 2011. He directs the UNC HIV Cure Center a unique environment for training, mentoring, and collaborative work across the continuum of research towards an HIV Cure.

Acknowledgements

The organising committee would like to show their respects and acknowledge the Bedegal people who are the Traditional Custodians of the Land on which this meeting takes place, and to Elders past and present.

This event is sponsored by Gilead Sciences. Gilead Sciences has not had any input to the choice of presenters or any other part of the meeting program and planning.

Organising committee

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