



## PROGRAMME

<u>TIME</u>	<u>TITLE</u>	<u>PRESENTING AUTHOR</u>	<u>ABSTRACT</u>
<b>Tuesday, June 9, 2009</b>			
19:00–21:00	WELCOME DINNER; Sanibel Harbour Resort: Palms Pool Deck		
<b>Wednesday, June 10, 2009</b>			
8:30	Opening remarks	Douglas Richman; VA San Diego Healthcare System and University of California San Diego, USA	
8:45	Introduction of State of the Art Presentation	Charles Boucher; Erasmus Medical Center, the Netherlands	
9:00–9:30	Eradication of HIV infection: any problem, however complicated, if looked in the right way, becomes more complicated	David Margolis; University of North Carolina at Chapel Hill, USA	1
<b>PERSISTENCE, RESERVOIRS AND ELIMINATION STRATEGIES</b>			
Chairs:	Charles Boucher David Margolis	Erasmus Medical Center, the Netherlands University of North Carolina at Chapel Hill, USA	
9:30	Roles of NFAT, cell division and IL-7 stimulation on HIV-1 reactivation from latency in primary memory T-cells	Alberto Bosque; University of Utah, USA	2
9:45	SJ23B, a jatrophone diterpene, induces HIV receptors downregulation and HIV transcription through activation of Ras-MEK pathway and classical PKCs	Jose Alcamí; Instituto de Salud Carlos III, Spain	3
10:00	Longitudinal dynamics of persistent viremia in elite controller patients versus patients on suppressive therapy	Sarah Palmer; Swedish Institute for Infectious Disease Control, Karolinska Institute, Sweden	4
10:15	HIV-specific CD4 <sup>+</sup> T-cells may contribute to viral persistence in HIV-infected elite controllers	Peter Hunt; University of California, San Francisco, USA	5
10:30	BREAK		
11:00	HIV-1 plasma virus diversity persists despite suppression with antiretroviral therapy	Mary Kearney; National Cancer Institute, USA	6
11:15	The role of chitinase-3-like-1 in bacterial translocation during HIV-1 infection	Anthony Smith; University of Minnesota, USA	7
11:30	Markers of cellular immune activation do not correlate with levels of residual viremia in patients on long-term suppressive antiretroviral therapy	Francesca Cossarini; National Cancer Institute, National Institutes of Health, USA	8
11:45	No evolution of HIV-1 total DNA and 2-LTR circles after 24 weeks of raltegravir-containing regimen: a substudy of randomized EASIER-ANRS 138 trial	Constance Delaugerre; Saint Louis Hospital, France	9

12:00	Antiretroviral intensification does not reduce persistent HIV-1 viremia on therapy	Frank Maldarelli; NCI Drug Resistance Program, USA	10
12:15	LUNCH		
13:30–16:30	<b>POSTER VIEWING</b> Oral presentations from Wednesday Clinical Implications of Resistance HBV Drug Resistance HCV Drug Resistance Epidemiology HIV Pathogenesis, Fitness and Resistance Persistence, Reservoirs and Elimination Strategies		
16:30–17:00	Epidemiology of drug resistance in countries with long-term access to HAART	Sabine Yerly; Geneva University Hospitals, Switzerland	
	FREE EVENING		

### Thursday, June 11, 2009

8:30	Introduction of State of the Art Presentation	Brendan Larder; RDI, UK	
8:45–9:15	State of the Art Presentation	John Moore; Weill Cornell Medical College, USA	11
	V3-dependent and -independent pathways to small molecule CCR5 inhibitor resistance	John Moore; Weill Cornell Medical College, USA	
9:20–10:15	<b>RESISTANCE TO ENTRY INHIBITORS (5-minute presentations)</b>		
Chairs:	Brendan Larder John Mellors	RDI, UK University of Pittsburgh, USA	
	Different mechanisms of HIV-1 inhibition by CCR5 agonists/antagonists (maraviroc, TAK-779 and PSC-RANTES) are linked to different drug resistance mechanisms	Eric Arts; Case Western Reserve University, USA	12
	Transitions from CCR5 to CXCR4 use in the absence of antiretroviral drug pressure proceed incrementally and may occur through a multitude of genetic pathways	Wei Huang; Monogram Biosciences, USA	13
	Estimating evolutionary pathways to CXCR4 usage from cross-sectional data	Alexander Thielen; Max-Planck Institute for Informatics, Germany	14
	Screening for HIV tropism using population-based V3 genotypic analysis: a retrospective virological outcome analysis using stored plasma screening samples from MOTIVATE-1	Richard Harrigan; BC Centre for Excellence in HIV/AIDS, Canada	15
	Modification of the CCR5 binding site by mutations in gp120 influence drug susceptibility and viral infectivity in one subject with clinical resistance to vicriviroc	John A Howe; Schering-Plough Research Institute, USA	16
9:55–10:15	Panel Discussion: Resistance to Entry Inhibitors		
10:15	BREAK		
10:45–12:00	<b>RESISTANCE TO REVERSE TRANSCRIPTASE INHIBITORS: POLYMERASE DOMAIN (5-minute presentations)</b>		
	High-resolution structures of HIV-1 RT/RNA:DNA ternary complexes with tenofovir diphosphate and dATP	Kalyan Das; Center for Advanced Biotechnology and Medicine (CABM), and Rutgers University, USA	17
	The HIV-1 RT mutant Q151L shows decreased replication capacity, selective high-level resistance to GS-9148 and hypersusceptibility to tenofovir and zidovudine	Kirsten White; Gilead Sciences, Inc., USA	18

	Mechanisms associated with HIV-1 resistance to acyclovir by the V75I mutation in reverse transcriptase	Matthias Götte; McGill University, Canada	19
	Mechanisms of inhibition and resistance to translocation deficient reverse transcriptase inhibitors	Bruno Marchand; University of Missouri, USA	20
	Recombinant viruses expressing subtype B or subtype C reverse transcriptase reveal no difference in the rate of K65R resistance to tenofovir in cell culture	Charles Dobard; Centers for Disease Control and Prevention, USA	21
	HIV-1 reverse transcriptase fidelity variants alter levels of mutation during replication	Dwight Nissley; National Cancer Institute, USA	22
	Different evolution and patterns of genotypic resistance profiles in emtricitabine plus tenofovir and lamivudine plus tenofovir containing regimen	Valentina Svicher; University of Rome Tor Vergata, Italy	23
	Lamivudine resistance mutations in HBV reverse transcriptase can be selected even at extremely low levels of viral replication	Carlo F Perno; University of Rome Tor Vergata, Italy	24
11:34–11:54	Panel Discussion: Resistance to Reverse Transcriptase Inhibitors: Polymerase Domain		
<b>RESISTANCE TO INTEGRASE INHIBITORS</b>			
12:00	HIV integrase inhibitor dissociation rates correlate with efficacy <i>in vitro</i>	Jay Grobler; Merck Research Laboratories, USA	25
12:10	Drug concentration, selective advantage and selection dynamics of raltegravir-resistant mutants	Francois Clavel; Inserm U941, France	26
12:20	New integrase binding inhibitors acting in synergy with raltegravir	Laurent Thibaut; BioAlliance Pharma, France	27
12:30	LUNCH		
<b>13:30–17:00 POSTER VIEWING</b>			
	Oral presentations from Thursday		
	Mechanisms of HIV Drug Resistance		
	New Resistance Technologies and Interpretations		
	Resistance to New Antiretroviral Agents		
19:00–21:00	WORKSHOP DINNER; Sanibel Harbour Resort: Everglades Ballroom		
<b>Friday, June 12, 2009</b>			
8:30	Introduction of Plenary Presentation	Douglas Richman; VA San Diego Healthcare System and University of California San Diego, USA	
8:40–9:25	The structure of an entire HIV-1 RNA genome	Kevin Weeks; University of North Carolina at Chapel Hill, USA	28
<b>9:30–10:30 RESISTANCE TO REVERSE TRANSCRIPTASE INHIBITORS: CONNECTION/RNASE H DOMAINS (5-minute presentations)</b>			
Chairs:	Douglas Richman	VA San Diego Healthcare System and University of California San Diego, USA	
	Andrew Zolopa	Stanford University, USA	
	Subtype-specific amino acid polymorphisms in the HIV-1 reverse transcriptase connection subdomain of CRF01_AE are associated with higher 3'-azido-3'-deoxythymidine resistance	Krista Delviks-Frankenberry; National Cancer Institute, USA	29
	Mutational analysis of residue Asn348 in HIV-1 reverse transcriptase	Jessica Radzio; University of Pittsburgh, USA	30

	A novel molecular mechanism of dual resistance to nucleoside and non-nucleoside reverse transcriptase inhibitors	Vinay Pathak; National Cancer Institute, USA	31
	Virologic failure of regimens containing two NRTI plus efavirenz is not associated with the selection of mutations in the connection or RNase H domains of reverse transcriptase	Jessica Brehm; University of Pittsburgh, USA	32
	Effect of mutations in the RT connection domain on phenotypic susceptibility and virologic response to etravirine	Johan Vingerhoets; Tibotec BVBA, Belgium	33
10:12–10:30	Panel Discussion; Resistance to Reverse Transcriptase Inhibitors: Connection/RNase H Domains		
10:30	BREAK		
	<b>PROTEASE INHIBITOR RESISTANCE</b>		
11:00	Analysing drug resistance in terms of substrate recognition in HCV NS3/4A protease	Keith Romano; University of Massachusetts Medical School, USA	34
11:10	Identification of HIV-1 matrix determinants of fitness compensation in a protease inhibitor resistant virus	Chris Parry; Health Protection Agency, UK	35
	<b>GLOBAL RESISTANCE/EPIDEMIOLOGY</b>		
11:20	Computational models developed without a genotype for resource-poor countries predict response to HIV treatment with 82% accuracy	Andrew Revell; HIV Resistance Response Database Initiative, UK	36
11:30	Drug resistance patterns among HIV-infected children and adults failing Kaletra-based regimens in South Africa	Gillian Hunt; National Institute for Communicable Diseases, South Africa	37
11:40	Impact of newly HIV diagnosed individuals on the transmission of drug resistance	Sabine Yerly; Geneva University Hospitals, Switzerland	38
11:50	Successful identification of sources of transmitted drug resistant HIV-1 via a cross-sectional phylogenetic analysis	Viktor von Wyl; University Hospital Zurich, Switzerland	39
12:00 PM	LUNCH		
13:30–15:30	<b>POSTER VIEWING</b> Oral presentations from Friday Drug Resistance in Resource-Poor Countries		
15:30–16:25	<b>MINOR VARIANTS AND RESISTANCE ISSUES IN PREVENTION OF MATERNAL TO CHILD TRANSMISSION (5-minute presentations)</b>		
Chairs:	Douglas Richman	VA San Diego Healthcare System and University of California San Diego, USA	
	Andrew Zolopa	Stanford University, USA	
	Addition of extended zidovudine to extended nevirapine prophylaxis reduces resistance in infants who were HIV-infected <i>in utero</i> : the PEPI-Malawi study	Jessica Lidstrom; Johns Hopkins University School of Medicine, USA	40
	Pre-existing low-levels of the K103N HIV-1 RT mutation above a threshold is associated with virological failure in treatment-naïve patients undergoing EFV-containing antiretroviral treatment	Evguenia Svarovskaia; Gilead Sciences, Inc., USA	41
	Prevalence and clinical significance of transmitted drug resistant (TDR) HIV mutations by ultra-deep sequencing (UDS) in HIV-infected ARV-naïve subjects in CASTLE study	Max Lataillade; Bristol Myers-Squibb and Yale University School of Medicine, USA	42

	Bulk sequence-detectable resistance mutations in peripheral RNA following single-dose nevirapine are associated with poorer treatment responses but do not adequately explain treatment failure	Jeffrey Johnson; Centers for Disease Control and Prevention, USA	43
	Resistance mutations detected by oligonucleotide ligation assay of HIV-1 DNA at time of initiation of nevirapine-containing antiretroviral therapy are associated with virologic failure	Thor Wagner; University of Washington and Seattle Children's Hospital, USA	44
16:05–16:25	Panel Discussion: Minor Variants and Resistance Issues in Prevention of Maternal to Child Transmission		
16:30–17:00	Myth versus reality: preventing drug resistance in resource-limited settings	Neil Parkin; World Health Organization, Switzerland	
20:00–23:00	AWARDS DINNER; Sanibel Harbour Resort: Palms Ballroom		