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## Evolutionary Dynamics of HIV-1 Subtype C Accessory and Regulatory Genes in Primary Infection

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<b>Citation</b>	Rossenkhan, Raabya, Vladimir A. Novitsky, TK Sebunya, Rosemary Mubanga Musonda, BA Gashe, and Myron Elmer Essex. 2012. Evolutionary dynamics of HIV-1 subtype C accessory and regulatory genes in primary infection. <i>Retrovirology</i> 9(Suppl 2): P142.
<b>Published Version</b>	<a href="https://doi.org/10.1186/1742-4690-9-S2-P142">doi:10.1186/1742-4690-9-S2-P142</a>
<b>Accessed</b>	February 19, 2015 10:49:48 AM EST
<b>Citable Link</b>	<a href="http://nrs.harvard.edu/urn-3:HUL.InstRepos:10483963">http://nrs.harvard.edu/urn-3:HUL.InstRepos:10483963</a>
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POSTER PRESENTATION

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# Evolutionary dynamics of HIV-1 subtype C accessory and regulatory genes in primary infection

R Rossenkhan<sup>1\*</sup>, V Novitsky<sup>2</sup>, TK Sebunya<sup>3</sup>, R Musonda<sup>4</sup>, BA Gashe<sup>3</sup>, M Essex<sup>2</sup>

From AIDS Vaccine 2012

Boston, MA, USA. 9-12 September 2012

## Background

Studies addressing the dynamics of accessory and regulatory viral gene diversity and selection during early stage of HIV-1 infection are limited but crucial for progress towards vaccine research.

## Methods

Intra-patient diversity and evolution was assessed during primary HIV-1C infection, viral quasiespecies were obtained by single genome amplification (SGA) at multiple sampling time points up to one year post-seroconversion (p/s).

## Results

The mean intra-patient diversity was found to be 0.11% (95%CI; 0.02 to 0.20) for *vif*, 0.23% (95%CI; 0.08 to 0.38) for *vpr*, 0.35% (95%CI; -0.05 to 0.75) for *vpu*, 0.18% (95%CI; 0.01 to 0.35) for *tat* exon 1 and 0.30% (95%CI; 0.02 to 0.58) for *rev* exon 1 during the time period 0 to 90 days p/s. The intra-patient diversity increased gradually in all non-structural genes over the first year of HIV-1 infection, which was evident from the *vif* mean intra-patient diversity of 0.46% (95%CI; 0.28 to 0.64), *vpr* 0.44% (95%CI; 0.24 to 0.64), *vpu* 0.84% (95%CI; 0.55 to 1.13), *tat* exon 1 0.35% (95%CI; 0.14 to 0.56) and 0.42% (95%CI; 0.18 to 0.66) for *rev* exon 1 during the time period of 181 to 500 days p/s. Statistically significant increases in viral diversity were observed for *vif* ( $p=0.013$ ) and *vpu* ( $p=0.002$ ). Weak and sporadic associations between levels of viral diversity within the non-structural genes and HIV-1 RNA load during primary infection were found. Positive and negative selection

patterns over the first year post-seroconversion were assessed in each of these genes, providing insight into the selection pressures on these genes which are crucial for viral replication in-vivo.

## Conclusion

Our study highlights differential diversity and slower diversification across these HIV-1 genes. The most likely cause is different selection pressure imposed by host immune response to the encoded viral gene products that may result in different evolutionary rates.

## Author details

<sup>1</sup>HSPH/UB/BHP, Boston, MA, Botswana. <sup>2</sup>Harvard School of Public Health (HSPH)/ BHP, Boston, MA, USA. <sup>3</sup>University of Botswana (UB), Gaborone, Botswana. <sup>4</sup>Botswana Harvard AIDS Institute Partnership (BHIP), Gaborone, Botswana.

Published: 13 September 2012

doi:10.1186/1742-4690-9-S2-P142

Cite this article as: Rossenkhan *et al.*: Evolutionary dynamics of HIV-1 subtype C accessory and regulatory genes in primary infection. *Retrovirology* 2012 **9**(Suppl 2):P142.

<sup>1</sup>HSPH/UB/BHP, Boston, MA, Botswana

Full list of author information is available at the end of the article