Retrovirology

BioMed Central

Poster presentation

Characteristics of HIV-1 gp120 *env* sequences in mother-child pairs infected with HIV-1 subtype CRF01_AE

Tanawan Samleerat*[1,2], Martine Braibant[2], Gonzague Jourdain[3,4], Alain Moreau[2], Nicole Ngo-Giang-Huong[3,4], Pranee Leechanachai[1], Jittapol Hemvuttiphan[5], Temsiri Hinjiranandana[6], Tikamporn Changchit[7], Boonyarat Warachit[8], Veera Suraseranivong[9], Marc Lallemant[3,4] and Francis Barin[2]

Address: [1]Faculty of Associated Medical Sciences, Chiang Mai University, Chiang Mai, Thailand, [2]Université François-Rabelais, INSERM ERI 19 & CHRU de Tours, Tours, France, [3]Harvard University, IRD 054, Chiang Mai, Thailand, [4]Institut de Recherche pour le Développement, UMI 174, Chiang Mai, Thailand, [5]Phayao Hospital, Phayao, Thailand, [6]Somdej Pranangchao Sirikit Hospital, Chonburi, Thailand, [7]Phan Hospital, Chiang Rai, Thailand, [8]Hat Yai Hospital, Songkla, Thailand and [9]Bhumibol Adulyadej Hospital, Bangkok, Thailand

* Corresponding author

from Fourth Dominique Dormont International Conference. Host-Pathogen Interactions in Chronic Infections
Paris, France. 13-15 December 2007

Published: 9 April 2008

Retrovirology 2008, **5**(Suppl 1):P5 doi:10.1186/1742-4690-5-S1-P5

This abstract is available from: http://www.retrovirology.com/content/5/S1/P5

Background

Previous studies have suggested that mother-to-child transmission of HIV-1 is often characterized by acquisition of a homogeneous viral quasispecies in the infant [1-3], suggestive of a genetic bottleneck. In this study, we have analyzed the molecular characteristics of transmitted HIV-1 viruses in a homogeneous population infected by CRF01_AE variants in Thailand.

Materials and methods

Seventeen mother-child pairs were studied. The infants were not breastfed. Six infants were infected *in utero* and 11 infants were infected intrapartum. The *env* sequences covering the entire gp120 were amplified from both the proviral DNA of maternal PBMC collected at delivery and the plasma viral RNA at first positive time point of infants. The amplified products were cloned and sequenced. A total of 353 clones were available for analysis.

Results

Phylogenetic analysis indicated 2 patterns of transmission: 14 mothers transmitted a single variant and 3 mothers transmitted multiple variants to their infants. The mean genetic distance of viruses from the mothers was significantly higher than those from the infants (2.7% *vs.* 0.6%; P<0.01), but without any difference according to timing of transmission, either *in utero* or intrapartum. The length of gp120 and number of potential N-linked glycosylation sites (PNGS) were similar in both the entire gp120 and individual regions of gp120 of all mother-child pairs. However, our data indicate that 4 PNGS positions (N241, N301, N354, and N384) that appeared conserved in all infant variants but were irregularly present in the mothers might be associated to a selective advantage. In addition, we report the first case to our knowledge of transmission to an infant of a recombinant virus issued from variants from his mother.

Conclusions

Our results provide additional evidence that despite a complex viral population in the mother, only viruses of a restricted subset are transmitted to the infant, independently of the timing of transmission, *in utero* or intrapartum. We did not find that shorter gp120 or fewer PNGS were characteristics of viruses transmitted from mother to infant, as it was suggested for sexually transmitted viruses

at least for a few subtypes [4-7]. Four PNGS were selected for transmitted viruses, supporting the role of the "glycan shield" of the HIV-1 envelope in conferring a selective advantage.

References

1. Dickover RE, Garratty EM, Plaeger S, Bryson YJ: **Perinatal transmission of major, minor, and multiple maternal human immunodeficiency virus type 1 variants in utero and intrapartum.** *J Virol* 2001, **75:**2194-2203.
2. Verhofstede C, Demecheleer E, De Cabooter N, Gaillard P, Mwanyumba F, Claeys P, Chohan V, Mandaliya K, Temmerman M, Plum J: **Diversity of the human immunodeficiency virus type 1 (HIV-1) env sequence after vertical transmission in mother-child pairs infected with HIV-1 subtype A.** *J Virol* 2003, **77:**3050-3057.
3. Wolinsky SM, Wike CM, Korber BT, Hutto C, Parks WP, Rosenblum LL, Kunstman KJ, Furtado MR, Munoz JL: **Selective transmission of human immunodeficiency virus type-1 variants from mothers to infants.** *Science* 1992, **255:**1134-1137.
4. Chohan B, Lang D, Sagar M, Korber B, Lavreys L, Richardson B, Overbaugh J: **Selection for human immunodeficiency virus type 1 envelope glycosylation variants with shorter V1-V2 loop sequences occurs during transmission of certain genetic subtypes and may impact viral RNA levels.** *J Virol* 2005, **79:**6528-6531.
5. Derdeyn CA, Decker JM, Bibollet-Ruche F, Mokili JL, Muldoon M, Denham SA, Heil ML, Kasolo F, Musonda R, Hahn BH, *et al.*: **Envelope-constrained neutralization-sensitive HIV-1 after heterosexual transmission.** *Science* 2004, **303:**2019-2022.
6. Frost SD, Liu Y, Pond SL, Chappey C, Wrin T, Petropoulos CJ, Little SJ, Richman DD: **Characterization of human immunodeficiency virus type 1 (HIV-1) envelope variation and neutralizing antibody responses during transmission of HIV-1 subtype B.** *J Virol* 2005, **79:**6523-6527.
7. Li B, Decker JM, Johnson RW, Bibollet-Ruche F, Wei X, Mulenga J, Allen S, Hunter E, Hahn BH, Shaw GM, *et al.*: **Evidence for potent autologous neutralizing antibody titers and compact envelopes in early infection with subtype C human immunodeficiency virus type 1.** *J Virol* 2006, **80:**5211-5218.