

## Differences in HIV-1 Subtypes on Disease Progression, Prevention and Response to Antiretrovirals

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The HIV-1 diversity causes different genotypic and phenotypic properties to viral variants, rendering them distinct characteristics regarding transmission, progression to disease, viral evolution, and resistance to antiretrovirals, population dispersion, viral fitness and epidemiological characteristics [1-4].

Some studies report an association between subtype C and heterosexual transmission [5-7]. In addition, [5,6] associate the frequencies of HIV-1 C with heterosexual women [7] revealed a statistically significant association between this viral variant and a higher cell counts T CD4+ in patients newly infected. Regarding the response to antiretroviral, 19 reported that subtype C is associated with lower accumulation of mutations to Nucleoside Reverse Transcriptase Inhibitors (NRTIs) and Protease Inhibitors (PI) than subtype B in patients in antiretroviral therapy (ART). Furthermore, there was a greater viral suppression at the beginning of ART associated with HIV-1 C. In another study [8], found, *in vitro*, that HIV-1 B has a higher propensity for resistance to zidovudine (ZDV) than HIV-1 C.

The HIV-1 B GWGR, a variant of subtype B with high prevalence in Brazil, has a slower progression to AIDS than other variants individuals of the same subtype [9-11].

It's showed that BF recombinants from Argentina have a high rate of population growth, higher than any other HIV-1 subtype. The authors linked that high frequency transmission to a high viral fitness, which favors the spread of those recombinants. The ability to greater dispersion of BF recombinants in South America was also evaluated by [12] that indicated HIV-1 BF had a higher number of secondary infections arising from a single infection, defined as R0, than their parental strains "pure" B and F [13].

HIV-1 subtype F has more PIs resistance mutations (L10V, M36I, I50V) than subtype B [14,15] showed that the resistance to PIs in subtype B was associated with L90M mutation, while HIV-1 F had a higher correlation with G48V and V82A/F [16] observed *in vitro* that the greater resistance of HIV-1 F to PIs is the fact that their protease are significantly less susceptible to inhibition by these drugs, and the accumulation of natural polymorphisms in protease of subtype F improve the catalytic activity of the enzyme, resulting in increased viral viability [16]. On the other hand, the subtype F had a lower non-Nucleoside Reverse Transcriptase Inhibitors (NNRTI) resistance rate compared to subtypes B and C [17]. Some studies relate the subtype F with heterosexual transmission [18-20].

Recently, our research group published an article about demographic, clinical and antiretroviral susceptibility characteristics for HIV-1 subtype B and non-B. It was found that non-B subtypes were associated with the female, lower educational level, lower viral load and higher cell counts T CD4+. We emphasized the importance of molecular epidemiology for more information about the progress of the epidemic and the possibility of establishment of correct preventive and therapeutic measures.

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