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# Highly active antiretroviral treatment (HAART) interruption leads to an increase in mitochondrial DNA content in HIV-infected children

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## Purpose of the study

HIV infection itself and antiretroviral treatment, especially nucleoside analogue reverse transcriptase inhibitors (NRTIs), cause mitochondrial impairment in HIV-infected patients, due to the inhibition of ã-polymerase, the only enzyme responsible for mitochondrial DNA (mtDNA) replication. We investigated whether there are changes in mtDNA content after 1 year of treatment interruption in children.

## Methods

Mitochondrial DNA (mtDNA) was assessed by Real-Time Polymerase Chain Reaction (RT-PCR) in peripheral blood mononuclear cells (PBMCs) of 13 perinatally-HIV-infected pediatric patients, who underwent planned treatment interruption (PTI). MtDNA was measured at the time of PTI and 12 months later. A sequence of a highly conserved mtND2 gene and a fragment of the nuclear-coded housekeeping 18SrRNA gene were amplified separately. Changes in mtDNA amount were expressed as the ratio of ND2 mtDNA with respect to 18SrRNAnDNA.

## Summary of results

MtDNA content significantly increased from 0.89 ± 0.173 to 1.48 ± 0.3429 (66%, $p < 0.05$) after 12-month treatment interruption.

## Conclusion

MtDNA content restoration was found in a group of perinatally HIV-infected pediatric patients after 12 months of HAART interruption. Our results suggest that mitochondrial damage is rather due to the use of nucleoside analogues than to HIV infection itself. In this setting, it is important to investigate new therapeutic treatment-sparing strategies in HIV-infected pediatric patients.

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