Poster presentation

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Inactivation of cell associated-HIV-1 in breast milk by treatment with the alkyl sulfate microbicide sodium dodecyl sulfate (SDS) Edouard Tuaillon^{*1,2}, Kuda Mutasa³, Pierre-Alain Rubbo¹, Laura Choteau², Florence Naudan², Karine Bollore¹, Jean-Pierre Vendrell^{1,2} and Philippe Van de Perre^{1,2}

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Background

Breast milk is recognized as a predominant mode of HIV-1 infection in infants. Cell-associated HIV-1 may be the main source of virus transmission during early phases of breast-feeding. We have previously observed that HIV-1infected cells spontaneously producing virus persist in breast-milk from women under antiretroviral therapy. Treatment of expressed milk with a microbicide such as Sodium dodecyl sulfate (SDS) is proposed as a simple and safe option to inactivate both cell free and cell associated HIV-1 when formula feeding is not practicable. However, the effect of SDS on spontaneously HIV-1-producing CD4⁺T cells in breast milk has not been fully explored.

Materials and methods

In this report human milk was spiked by HIV-1-infected cells and treated with increasing exposure time and SDS concentration. CD4+T cell apoptosis and death, cell-associated HIV-1 RNA production, and spontaneous HIV-1-Ag cell secretion were quantified after SDS treatment.

Results

Cell death increases in presence of SDS in a concentrationand time-dependent manner, 50% of T lymphocytes death after 2 minutes with 0.14% SDS and 90% after 10 minutes with 0.1% SDS (Fig. 1A). Undetectable HIV-1 RNA cell production was achieved following exposure with a minimum concentration of 0.1% SDS during 2 minutes, IC50 = 0.03% (Fig. 1B). The inhibition of HIV-1 Ag secretion was explored at a single cell level by ELISpot assay. Using this method inactivation was 100% for SDS concentrations $\geq 0.25\%$ within 2 min (Fig. 1C).

Conclusion

By comparison with results previously reported using an infectivity model based on β -galactosidase MAGI cells¹, we observed that a two fold higher SDS concentration was required to complete inactivation of HIV-1-Ag-secreting cells. This concentration remains in the reported safe limits for ingestion of SDS by children (1 g/kg/day). Regarding the possible occurrence of transmission to the infant after controlling cell-free virus in breast milk from women on antiretroviral therapy, SDS treatment of expressed breast milk may be an interesting strategy to optimized the prevention of HIV-1 pediatric transmission.



Figure I Effect of SDS exposure on T cell death (A), cell-associated HIV-I RNA production (B), and sponataeous HIV-I-Ag cell secretion (C).

References

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