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Mechanisms underlying reductions in mother-to-child transmission of human immunodeficiency virus type-1 by short-course antiretrovirals

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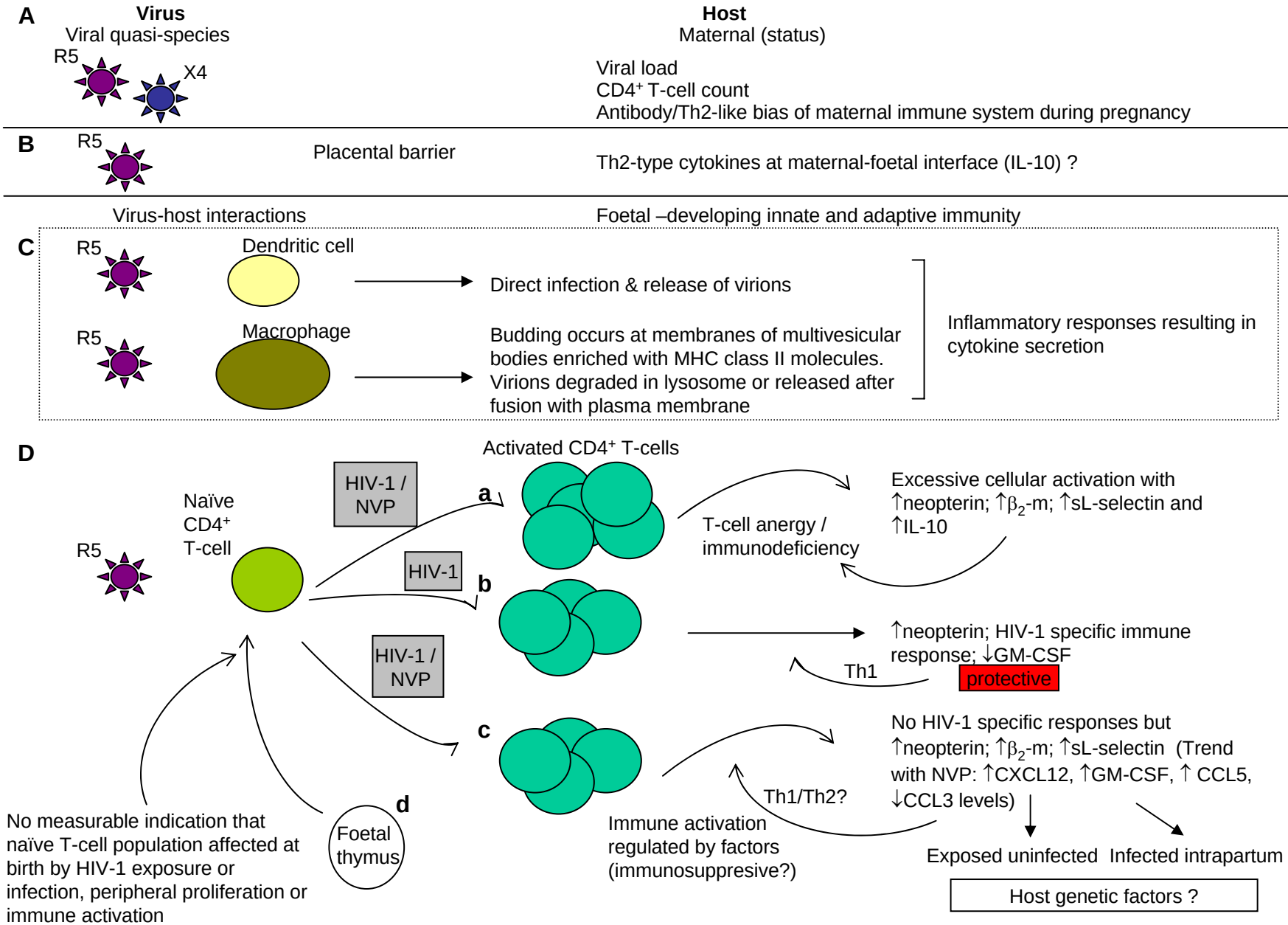
A
Virus
Viral quasi-species
R5
X4
Host
Maternal (status)
Viral load
CD4+ T-cell count
Antibody/Th2-like bias of maternal immune system during pregnancy
B
R5
Placental barrier
Th2-type cytokines at maternal-foetal interface (IL-10) ?
Virus-host interactions
Foetal –developing innate and adaptive immunity
C
R5
Dendritic cell
Direct infection & release of virions
R5
Macrophage
Budding occurs at membranes of multivesicular bodies enriched with MHC class II molecules. Virions degraded in lysosome or released after fusion with plasma membrane
Inflammatory responses resulting in cytokine secretion
D
Activated CD4+ T-cells
HIV-1 / NVP
a
Naïve CD4+ T-cell
R5
HIV-1
b
HIV-1 / NVP
c
T-cell anergy / immunodeficiency
Excessive cellular activation with ↑neopterin; ↑β2-m; ↑sL-selectin and ↑IL-10
↑neopterin; HIV-1 specific immune response; ↓GM-CSF
protective
Th1
No HIV-1 specific responses but ↑neopterin; ↑β2-m; ↑sL-selectin (Trend with NVP: ↑CXCL12, ↑GM-CSF, ↑ CCL5, ↓CCL3 levels)
Th1/Th2?
Immune activation regulated by factors (immunosuppresive?)
Exposed uninfected
Infected intrapartum
Host genetic factors ?
No measurable indication that naïve T-cell population affected at birth by HIV-1 exposure or infection, peripheral proliferation or immune activation
d
Foetal thymus