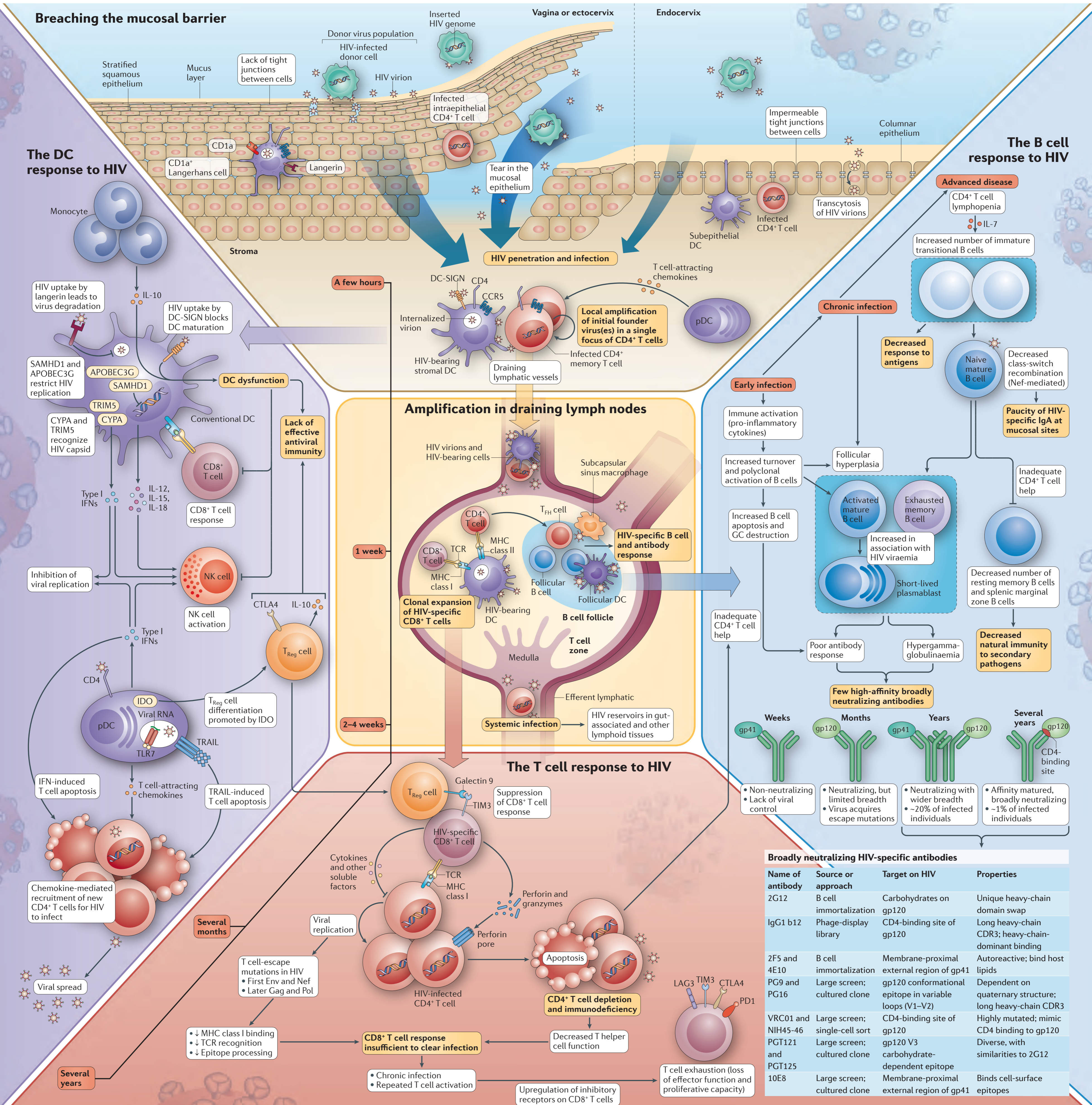


The immune response to HIV

Nina Bhardwaj, Florian Hladik and Susan Moir

Since HIV was discovered as the causative agent of AIDS almost 30 years ago, HIV infection has become a devastating pandemic, with millions of individuals becoming infected and dying from HIV-related disease every year. A global research effort over the past three decades has discovered more about HIV than perhaps any other pathogen. Immunologists continue to be intrigued by the capacity of HIV to effectively knock out an essential component of the

adaptive immune system — CD4⁺ T helper cells. This Poster summarizes how HIV establishes infection at mucosal surfaces, the ensuing immune response to the virus involving DCs, B cells and T cells, and how HIV subverts this response to establish a chronic infection. Based on a clearer understanding of HIV infection and the response to it, the field has now entered an era of renewed optimism for the development of a successful vaccine.



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Abbreviations

APOBEC3G, apolipoprotein B mRNA editing, catalytic polypeptide-like 3G; CCR5, CC-chemokine receptor 5; CDR3, complementarity-determining region 3; CTLA4, cytotoxic T lymphocyte antigen 4; CYP4, cyclophilin A; DC, dendritic cell; DC-SIGN, DC-specific ICAM3-grabbing non-integrin; GC, germinal centre; IDO, indoleamine 2,3-dioxygenase; IFN, interferon; IL, interleukin; LAG3, lymphocyte activation gene 3; NK, natural killer; PD1, programmed cell death protein 1; PDC, plasmacytoid DC; SAMHD1, SAM domain- and HD domain-containing protein 1; TCR, T cell receptor; T_H cell, T follicular helper cell; TIM3, T cell immunoglobulin domain- and mucin domain-containing protein 3; TLR7, Toll-like receptor 7; TRAIL, TNF-related apoptosis-inducing ligand; T_{Reg} cell, regulatory T cell; TRIM5, tripartite motif-containing protein 5.

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