HIV integration into the host DNA is not a random event.

HIV is a persistent viral infection and even in the presence of an effective antiretroviral treatment (ART) regimen, the virus hides in a latent state. In the 1st August edition of Science, investigators at the University of Washington, Seattle, have shown using very elegant molecular tools, that HIV integration is not a random event. Rather, the HIV integration sites appeared to occur very close to genes associated with cancer or cell cycle regulation. The authors state “HIV integration into genes associated with cancer or cell cycle regulation appears to confer a survival advantage that allows these cells to persist during suppressive ART.” So it seems that when a cell is triggered to proliferate and expand, this serves as an important mechanism that allows HIV to persist and perpetuate latency. Novel treatments that prevent HIV infected cells from proliferating, along with suppressing HIV replication, could lead the way to a “curative strategy”.

Wagner, T. et al. 2014. Proliferation of cells with HIV integrated into cancer genes contributes to persistent infection. *Science*.