Cover photograph: The HIV long terminal repeat (LTR) harbors two highly conserved \(\kappa\)B binding sites among the majority of HIV isolates. NF-\(\kappa\)B translocates into the nucleus and initiates HIV transcription and replication by binding to \(\kappa\)B binding sites. However, as a negative regulator of NF-\(\kappa\)B signaling, NF-\(\kappa\)B-interacting long noncoding RNA (NKILA) sequesters NF-\(\kappa\)B in the cytoplasm and prevents its translocation into the nucleus by forming a stable ternary complex with NF-\(\kappa\)B/\(\kappa\)B, thereby resulting in the suppression of HIV gene expression as well as the regulation of HIV latency. (See related article in September 2020, vol. 94, no. 17, e01057-20.) (Copyright © 2020 American Society for Microbiology. All Rights Reserved.)
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