miRNA-103 MODULATES CCR5 AND AFFECTS ESTABLISHMENT OF HIV-1 LATENCY IN CD4+ T CELLS

Nicolas Bellini1,2, Robert Lodge1, Tram N.Q. Pham1,4, Jaspreet Jain1, Cécile Tremblay2, Jean-Pierre Routy1, Alon Herschhorn1, Thomas T. Murooka2 and Éric A. Cohen1,6
1Institut de recherches cliniques de Montréal, Montreal, Canada ; 2Centre de Recherche du CHUM, Montreal, Canada ; 3McGill University Health Centre, Montreal, Canada ; 4University of Minnesota, Minneapolis, USA ; 5University of Manitoba, Winnipeg, Canada ; 6Department of Microbiology, Infectiology and Immunology, Université de Montréal, Montreal, Canada

BACKGROUND
- MiRNAs are small regulatory RNAs which affect the stability or the translation of target mRNAs
- We recently identified the p53-regulated miRNA-103 as a modulator of CCR5 expression in macrophages
- It has been shown that activated-to-memory transitioning CD4+ T cells upregulate CCR5, the HIV-1 co-receptor, and are more susceptible to latent HIV-1 infection
- Hypothesis: By contributing to the control of CCR5 expression in CD4+ T lymphocytes, miRNA-103 could play a role in the establishment of latent HIV-1 reservoirs in vivo

RESULTS
- Pharmacologic stabilization of p53 in CD4+ T cells decreases CCR5-dependent HIV-1 infection via upregulation of miRNA-103, reducing the frequency of productively and latently infected cells
- Reduced CCR5 mRNA levels in CD4+ T cells of elite and viremic controllers correlates with an upward miRNA-103 expression

CONCLUSION
- Activated-to-memory transitioning cells express high levels of CCR5 and have a cellular environment conducive to latent infection
- The p53-regulated miRNA-103 targets CCR5 mRNA in CD4+ T cells and participates in its regulation
- We observed a decrease of CCR5 mRNA in CD4+ T cells from EC/V individuals as well as an upward trend of miRNA-103 in these cells, suggesting that miRNA-103 contributes in part to the control of CCR5 expression in the CD4+ T cells

ADDITIONAL INFORMATION
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- Author contact information:
  Nicolas Bellini (PhD student) 110, avenue des Pins Ouest, H2W 1R7, Montréal (Québec) Canada
  Email: nicolas.bellini@mcgill.ca