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CHANGES IN LIVER STEATOSIS, BODY MASS INDEX AND HEPATOCYTE APOPTOSIS AFTER SWITCH TO RALTEGRAVIR

Bertrand Lebouche, Sahar Saeed, Andreas Giannakis, Louis-Patrick Haraoui, Alexandra de Pokomandy, Joseph Cox, Jean-Pierre Routy, Marina Klein, **Giada Sebastiani**

DIVISION OF GASTROENTEROLOGY AND HEPATOLOGY
MCGILL UNIVERSITY HEALTH CENTRE, MONTREAL, QUEBEC

Centre universitaire de santé McGill

McGill University Health Centre

McGill

Background & Aim

- Non-alcoholic fatty liver disease (NAFLD) is frequent in HIV infection
- NAFLD can lead to liver cirrhosis and early mortality
- Factors associated with hepatic steatosis in HIV infection
  - Metabolic factors: Strongest, overweight-obesity.
  - Antiretroviral therapy (ART)
    - Integrase inhibitor: safe metabolic profile ? → weight gain reported
    - Single study in HIV/HCV showed reduction of steatosis with switch from efavirenz to raltegravir

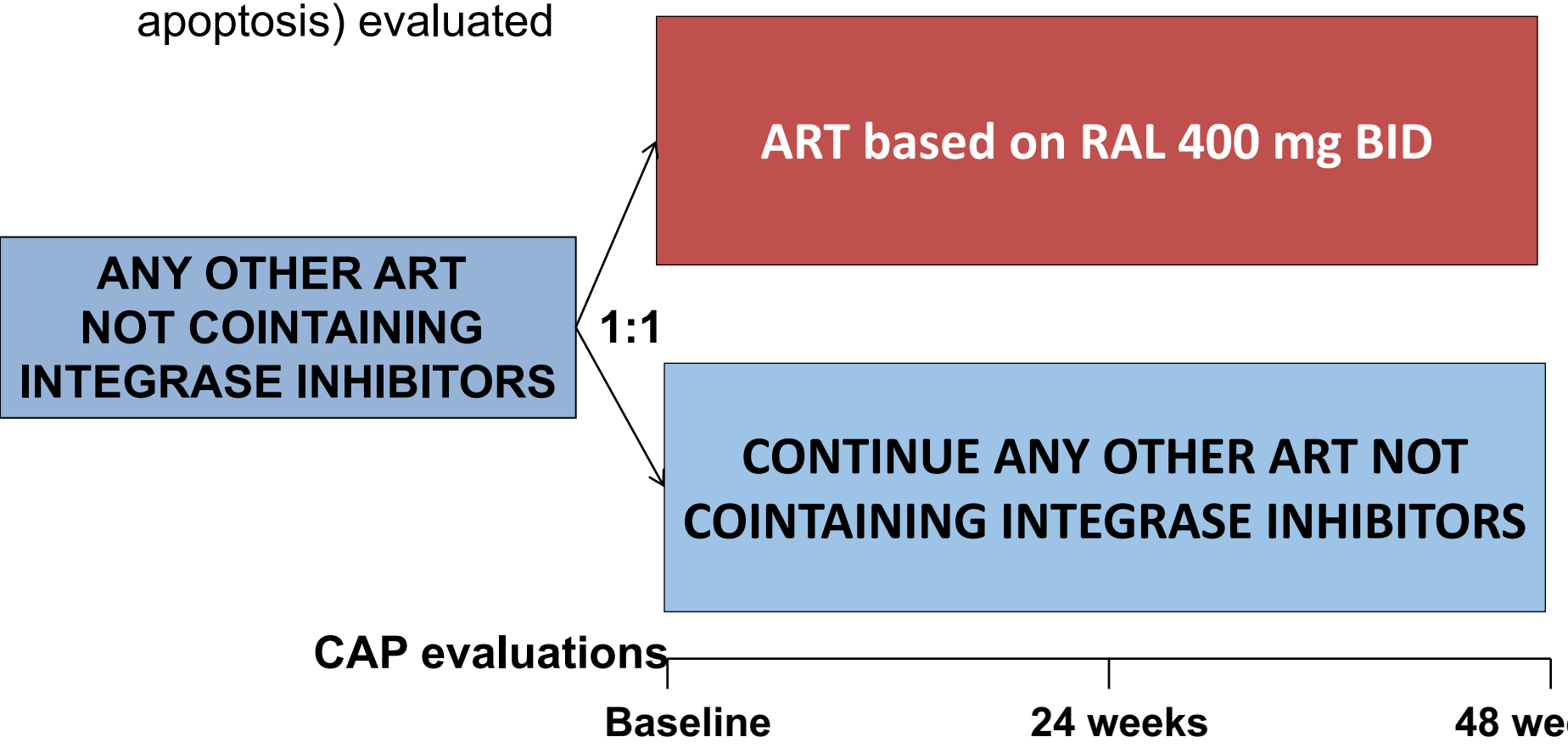
- **AIM**: to evaluate the effect of switching to raltegravir (RAL) on hepatic steatosis among HIV-infected patients with NAFLD.

Vuille-Lessard et al, AIDS 2016; Macias et al, CID 2017

Patients and Methods

Randomized, controlled, open label, phase 4 clinical trial

- CAP ≥238 dB/m, indicative of steatosis involving >10% of hepatocytes.
- HCV and HBV coinfections, as well as alcohol abuse, excluded
- Changes in CAP, BMI and cytokeratin 18 (biomarker of hepatocyte apoptosis) evaluated



Results

31 HIV mono-infected patients included (12 switched to RAL)

Change at 48 weeks compared to baseline	CAP	BMI	Cytokeratin 18
RAL	-25 (-94, 19)	-0.6 (-0.5, 0.2)	-26 (-27, -45)
Control	-26 (-40, 40)	-0.3 (0.5, 0.4)	27 (-14, 21)

At 48 weeks, 53% of patients in the RAL arm and 54% in the non-switch arm had CAP <238 dB/m, suggesting NAFLD resolution.

Conclusions

- After 48 weeks, HIV mono-infected individuals with NAFLD switching to RAL showed similar decrease in the degree of hepatic steatosis compared to the control group.

- Patients switching to RAL did not experience weight gain and had reduction in hepatocyte apoptosis compared to the control group.

Contact: giada.sebastiani@mcgill.ca