

La date butoir pour la soumission d'un résumé est le 9 octobre 2023 à 23h59

Spécifications du format du résumé à lier au formulaire

The deadline for abstract submission is October 9, 2023 at 11:59 p.m.

Specification of the format of the summary to link to the form

Format: "Word"

Title: Calibri 12 bold. The title must fit on 2 lines

Authors: Last name, first name (Calibri 12)

Affiliation: The figures in "superscript (x^{1,2})" following the name refer to the affiliations which must contain:

- a) Name of university department, research center and/or hospital center
- b) Name of the University
- c) City, province

Body of the text: In "Calibri 12" with the following sections:

Introduction, Methodology, Results, Discussion

Acknowledgements: Name of granting agencies (IRSC, FRQ-S, RSRQ, RRSPQ, etc.)

If you have space you can add a table but **Everything must fit in a single page** with the specifications mentioned above.

See the sample summary below:

Martin Klein, Niveau Postdoct, UniversitéLaval

IL-13 increases miRNAs-155 expression and exosomal cargo in severe asthma bronchial epithelial cells leading to a delayed repair process.

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Introduction: Asthma is a chronic disease characterized by a prevailing type 2 inflammation associated with airway remodeling. miRNAs (miR) were recently shown to participate in asthma physiopathology. In asthmatic patients, miR-155 have been shown to promote Th2 response, ILC2 survival and eosinophilic inflammation. However, its role in regulating bronchial epithelial cells (BEC) remains unclear in severe asthma.

Objectives: To measure the expression of miR-155 in BEC of healthy versus severe asthma patients and how IL-13 a type 2 cytokine can modulate its expression and exosomal cargo. To evaluate the effect of miR-155 on BEC behavior and function.

Methods: BEC from healthy donor and severe asthmatics were stimulated or not with recombinant human IL-13. Exosomes were obtained by serial ultracentrifuges. BEC-derived exosomes were quantified using CD63 protein expression by Western Blot. Cellular and exosomes-derived miR-155 expression was assessed by q-PCR. miR-155 was modulated in BEC with specific miR-155 mimic or inhibitor.

Results: BEC from severe asthma express more miR-155 than cells from healthy donors and this feature is increase under IL-13 stimulation. Moreover, severe asthma BEC release more exosome with an increase cargo of miR-155 than cells from healthy donors and both were also increased under IL-13 stimulation. Interestingly, miR-155 overexpression increases IL-13R α 1 and decreases IL-13R α 2 subunit gene and protein expression, whereas miR-155 inhibition is associated with a reduce IL-13R α 1 and an increase IL-13R α 2 expression in both asthmatic and healthy donors. These IL-13R α subunit modulations are associated with a delay in BEC repairing process after mechanical injury.

Conclusion: We show that severe asthma bronchial epithelial cells express more miR-155, release more exosomes containing miR-155 than bronchial epithelial cells from healthy donors and these features increase under IL-13 stimulation. mir-155 also modulate IL-13R α subunit and BEC repairing process Thus, these data suggest a potential new IL-13/miR-155 axis in severe asthma.

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