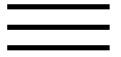


Contribution of hepatic de novo lipogenesis and reesterification of plasma non esterified fatty acids to plasma triglyceride synthesis during non-alcoholic fatty liver.

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Original article

Contribution of hepatic *de novo* lipogenesis and reesterification of plasma non esterified fatty acids to plasma triglyceride synthesis during non-alcoholic fatty liver disease

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Summary

Background

Non-alcoholic fatty liver disease (NAFLD) is frequently observed in insulin-resistant subjects and can lead to liver fibrosis and cirrhosis. The abnormalities of lipid metabolism behind this development of excess hepatic TG stores are poorly understood.

Methods

To clarify these mechanisms we measured triglyceride secretion rate and the

contributions of hepatic lipogenesis and reesterification of non-esterified fatty acids (NEFA) to this secretion in healthy subjects and in patients with clear evidence of NAFLD. All subjects were studied in the post-absorptive state. Hepatic lipogenesis was measured with deuterated water. NEFA turnover rate, triglyceride secretion rate and the contribution of NEFA reesterification to this secretion were determined with $[1-^{13}\text{C}]$ palmitate infusion.

Results

NAFLD patients had higher NEFA concentrations ($p < 0.05$) but normal NEFA turnover rates (5.23 ± 0.80 vs 5.91 ± 0.97 $\mu\text{mol}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ in control subjects, ns). Despite a trend for higher plasma triglyceride levels in patients ($p < 0.10$), triglyceride turnover rates were not increased (0.11 ± 0.01 $\mu\text{mol}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ in patients vs 0.14 ± 0.01 in controls, ns). However the contribution of hepatic lipogenesis to triglyceride secretion was largely increased in patients (14.9 ± 2.7 vs $4.6 \pm 1.1\%$ $p < 0.01$) while that of NEFA reesterification was reduced (25.1 ± 2.9 vs $52.8 \pm 6.2\%$ $p < 0.01$).

Conclusion

Enhanced lipogenesis appears as a major abnormality of hepatic fatty metabolism in subjects with NAFLD. Therapeutic measures aimed at decreasing hepatic lipogenesis would therefore be the most appropriate in order to reduce hepatic TG synthesis and content in such patients.

Résumé

Contribution de la lipogenèse hépatique *de novo* et de la réestérification des acides gras plasmatiques non estérifiés à la synthèse des triglycérides au cours de la stéatose hépatique non alcoolique

Objectif

La stéatose hépatique non-alcoolique (NAFLD) est fréquente chez les sujets insulino-résistants et peut évoluer vers la fibrose et la cirrhose. Les anomalies du métabolisme lipidique responsables de cette stéatose sont mal comprises.

Méthodes

Pour clarifier ces anomalies nous avons mesurer le débit de sécrétion de triglycérides et les contributions de la lipogenèse hépatique et de la réestérification hépatique des acides gras non estérifiés (NEFA) à cette sécrétion chez des sujets témoins et des sujets avec NAFLD. Tous les sujets ont

secretion chez des sujets sains et des sujets avec NAFLD. Tous les sujets ont été étudiés à l'état post-absorptif. La lipogenèse hépatique a été mesurée à l'aide d'eau deutériée. Le débit de renouvellement des NEFA, le débit de sécrétion des triglycérides et la contribution de la réestérification des NEFA à cette sécrétion ont été mesurés à l'aide de [1-¹³C] palmitate.

Résultats

Les sujets avec stéatose avaient des concentrations de NEFA élevées ($p < 0,05$) mais un débit de renouvellement normal ($5,23 \pm 0,80$ vs $5,91 \pm 0,97$ $\mu\text{mol.kg}^{-1}.\text{min}^{-1}$ chez les sains, ns). Malgré des concentrations de triglycérides légèrement élevées ($p < 0,10$) leur débit de sécrétion de triglycérides était normal ($0,11 \pm 0,01$ vs $0,14 \pm 0,01$ $\mu\text{mol.kg}^{-1}.\text{min}^{-1}$ chez les sains, ns). Cependant la contribution de la lipogenèse à la sécrétion de triglycérides était très supérieure chez les sujets stéatosiques ($14,9 \pm 2,7$ vs $4,6 \pm 1,1\%$ $p < 0,01$) alors que celle de la réestérification des NEFA était diminuée ($25,1 \pm 2,9$ vs $52,8 \pm 6,2\%$ $p < 0,01$).

Conclusion

Une augmentation de la lipogenèse apparaît comme une anomalie majeure du métabolisme hépatique au cours de la stéatose. Des mesures thérapeutiques visant à normaliser cette lipogenèse pourraient donc permettre de réduire la stéatose hépatique non alcoolique.



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Key-words

Stable isotopes; Liver; Steatosis; Triglyceride; Insulin-resistance

Mots-clés

Isotopes stables; Foie; Stéatose; Triglycérides; Insulino-résistance

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