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► To cite this version:

V Rigalleau, E de Tinguy, A Iron, J Aubertin, H Gin. Effects of lipid infusion on postabsorptive glycemia in non insulin dependent diabetes mellitus (NIDDM). *Reproduction Nutrition Development*, 1996, 36 (4), pp.429-429. <hal-00899878>

HAL Id: hal-00899878

<https://hal.science/hal-00899878v1>

Submitted on 11 May 2020

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1.1 ± 0.2 were bound to the column and were eluted by 30 mM and 1 M HCl, respectively.

We concluded that the amount of unsaturated FA in each fraction ($\approx 50\%$ of FA bound to glycogen, $\approx 90\%$ of FA in eluted fractions) may have accounted for the inhibition of Glc6Pase since it was shown that free but not bound unsaturated FA could inhibit the enzyme in vitro in relevant concentrations.

Effects of lipid infusion on postabsorptive glycemia in non insulin dependent diabetes mellitus (NIDDM). V Rigalleau, E De Tinguy, A Iron, J Aubertin, H Gin (*Clinique médicale, Tripode, 33000 Bordeaux, France*)

As proposed by Randle, lipid–glucid interactions may play a role in the hyperglycemia of NIDDM. We previously reported a hyperglycemic effect of a lipid infusion in the postabsorptive state in NIDDM patients [Rigalleau (1994) *Metabolism* 43, 1300]. This paper describes how we studied its mechanism in 30 NIDDM patients. Fifteen received a 180 min lipid infusion ('Ivélip 20%'; 0.015 mL/kg/min) and 15 received saline (controls). Glucose, TGs, FFAs levels were measured and continuous indirect calorimetry was performed beginning 30 min before the start of the infusion and continuing to the end of the testing period. Lipid infusion significantly ($P < 0.05$) slowed the postabsorptive glycemic decline (lipid: 10.5 ± 1.1 mmol/L to 10.2 ± 1.0 at 180 min, NS; saline: 11.2 ± 0.8 to 9.8 ± 0.7 , $P < 0.01$). The response was heterogeneous, six 'responders' showed an absolute hyperglycemic response (10.8 ± 1.0 to 11.8 ± 0.8), while nine 'nonresponders' did not, in a similar manner to the saline controls (11.4 ± 1.2 to 10.5 ± 1.1). The lipidic effects of the lipid infusion differed in these two groups. In 'responders', the lipid infusion produced a

greater ($P < 0.01$) increase in FFAs ('responders': 839 ± 218 $\mu\text{mol/L}$ to $3\ 335 \pm 840$; 'nonresponders': 954 ± 109 to $1\ 731 \pm 206$). In contrast, TGs ('responders': 2.20 ± 0.19 mmol/L to 8.56 ± 1.54 ; 'nonresponders': 1.76 ± 0.18 to 6.67 ± 0.89 ; NS), total lipid oxidation ('responders': 0.74 ± 0.09 mg/kg/min to 0.90 ± 0.06 ; 'nonresponders': 0.59 ± 0.16 to 0.79 ± 0.10 ; NS) and glucose oxidation ('responders': 1.27 ± 0.28 mg/kg/min to 1.14 ± 0.20 ; 'nonresponders': 1.40 ± 0.35 to 1.20 ± 0.30 ; NS) did not evolve differently in the responder and nonresponder groups. Lipid infusion modified postabsorptive glycemia in NIDDM patients. The response was heterogeneous, and needed lipolysis of the infused triglycerides. This did not occur the same way in all subjects, as previously shown in normal subjects [Peterson (1990) *Proc Natl Acad Sci USA* 87, 909]. In contrast, an elevation of lipid oxidation did not seem necessary. The normality of total lipid oxidation in NIDDM patients, therefore [De Fronzo (1988) *Diabetes* 37, 667], did not exclude a role of lipid–glucid interactions during hyperglycemia. Interaction at the oxidative level did not seem sufficient to elevate glycemia, indicating an additional effect of lipid infusion on endogenous glucose production or nonoxidative glucose disposal.

Effects of glucagon on fructose-induced alterations of glucose metabolism in man. N Paquot¹, P Schneider², E Jéquier², L Tappy² (¹ *Division of Diabetes, CHU-Sart Tilman, 4000 Liège, Belgium*; ² *Institute of Physiology, University School of Medicine, Bugnon 7, 1005 Lausanne, Switzerland*)

Gluconeogenic substrates increase gluconeogenesis but fail to enhance the overall endogenous glucose production (EGP). The mechanisms responsible for this autoregulation remain unknown. In order to assess the effects of hyperglucagonaemia on autoregulation of EGP, eight healthy sub-