Effect of bezafibrate on postprandial lipidemia in non-insulin-dependent diabetic (NIDDM) patients. V Durlach [1], N Attia [2], D Roche [3], JL Paul [4], F Landron [5], A Girard-Globa [2] *([1] Clinique médicale U62 CHU de Reims, 51092 Reims; [2] Groupe Lipoprotéines, faculté de médecine Xavier-Bichat, 75018 Paris; [3] Laboratoire de biochimie, hôpital Laënnec, 75015 Paris; [4] Laboratoire de biochimie, hôpital Broussais, 78014 Paris; [5] Lipha Santé, Division Oberval, 69008 Lyon, France)*

Alterations of the postprandial (pp) triglyceride (TG) response might play an important role in the development of atherosclerosis-thrombosis in non-insulin-dependent diabetic (NIDDM) patients. The aim of our study was to evaluate the effects on ten male NIDDM patients of a 5 week bezafibrate (BZ) treatment (400 mg/day) on lipid tolerance, 8 h following a standardized test meal (1 100 kcal, 80% lipids, 100 000 IU vitamin A). The patients were obese (BMI 30.3 ± 2.7 kg/m^2), under fair glycemic control (Hb A1c 7.6 ± 1.6%), had moderately elevated fasting TG 2.6 ± 1.1 mmol/L and normal LDL cholesterol (2.6 ± 1 mmol/L).

As expected, BZ reduced fasting TG ($P < 0.01$) but had no effect on total cholesterol (TC) levels. Fasting glycemia was improved from 11.9 ± 3.3 to 9.7 ± 3.4 mmol/L ($P < 0.04$). PP responses were calculated as the incremental area under the curve (AUC). Chylomicron TG were reduced by 40% ($P < 0.01$) in agreement with a retinyl palmitate lowering in this fraction. Free fatty acids (FFA) were reduced by over 50% ($P < 0.05$). Very low density lipoprotein (VLDL)-TG were not reduced; however, pp VLDL particles were larger after treatment, enriched with TG but not with cholesteryl esters (CE). These results might provide further support that BZ acts by activating lipolysis but also suggest that it might improve the uptake of FFA. The TG-rich VLDL produced pp can be lipolyzed rapidly without giving rise to LDL. The decrease in FFA is an interesting metabolic feature and may play a role in the improvement of fasting glycemia.

The hypolipidemic effect of fenofibrate cannot originate from the enhanced carnitine concentration in the rat liver. P Clouet, C Legendre, M Tsoko, F Beauseigneur, J Gresti, J Boichot, J Demarquoy, J Bézard *(Nutrition cellulaire et métabolique, EA DRED 564, université de Bourgogne, BP 138, 21001 Dijon cedex, France)*

Carnitine is essential to long-chain fatty acid degradation in mitochondria, because only carnitine derivatives can be transported across the inner membrane. The enhanced carnitine concentration (x 3.5) in the liver of fenofibrate-treated rats [Henninger et al (1987) *Biochem Pharmacol* 36, 3231] has been assumed to be a factor allowing long-chain fatty acids to be directed preferentially to β-oxidation. One way to verify this

	Control	*Fenofibrate*	*Feno/BBH inhibitor*
Lipid parameters in fed state (in g/L of serum)			
Triacylglycerols	2.06 ± 0.25^a	1.69 ± 0.17^b	1.41 ± 0.15^c
Total cholesterol	0.82 ± 0.06^a	0.49 ± 0.04^b	0.37 ± 0.02^c
Total ketone bodies in fasted state (mg/100 of blood)	18.72 ± 2.35^a	14.01 ± 1.93^b	11.89 ± 1.86^c

Results are means ± SEM ($n = 5$). The values in a row with different superscript letters (a,b,c) are significantly different at $P < 0.05$ (Anova and Fisher's test comparisons).