

## Metabolic Aspects and Mechanisms

### 9.3 Non-alcoholic Fatty Liver Disease, Adiponectin and Insulin-Resistance In Dipper And Non-Dipper Essential Hypertensive Patients

F. Fallo (1), A. Dalla Pozza (1), N. Sonino (2), G. Federspil (1), M. Ermani (3),  
S. Caroselli (4), C. Catena (4), G. Soardo (4), R. Carretta (5), D. Belgrado (5),  
B. Fabris (5), L. Sechi (4)

(1)Dipartimento di Scienze Mediche e Chirurgiche, Università di Padova,  
Padova; (2)Dipartimento Di Scienze Statistiche, Università di Padova, Padova;  
(3)Dipartimento di Neuroscienze, Università di Padova, Padova; (4)Clinica  
Medica e Cattedra di Medicina Interna, Università di Udine, Udine;  
(5)Dipartimento di Medicina Clinica e Neuropatologia, Università di Trieste,  
Trieste, Italy

**Introduction.** The pathogenesis of non-alcoholic fatty liver disease (NAFLD) is multifactorial, and the presence of insulin-resistance is recognized as the pathophysiological hallmark of this condition. Arterial hypertension is referred as an insulin-resistant state, and insulin resistance may substantially contribute to the cardiovascular risk in this disorder.

**Aim.** We examined the inter-relationship between insulin sensitivity, adiponectin levels, and NAFLD in hypertensive patients with different day/night blood pressure variation.

**Methods.** Eighty never-treated patients (48M/32 F) with essential hypertension were selected for having a nocturnal decrement of blood pressure more than 10% (dippers, n=47) or less than 10% (non-dippers, n=33) of day-time values. No subject had diabetes mellitus, obesity, hyperlipidaemia, or other risk factors for hepatic disease. The two groups were similar as to sex, age and body mass index. Abdominal fat distribution and NAFLD were assessed by ultrasonography.

**Results.** Hepatic steatosis was detected in 57.5% of all patients. Non-dippers showed a higher prevalence of NAFLD than dippers (81.8% vs 40.4%, P<0.005). Insulin and homeostasis model of assessment (HOMA) index were higher ( $86.5 \pm 33.0$  vs  $55.4 \pm 27.6$  pmol/l and  $3.1 \pm 1.2$  vs  $2.1 \pm 1.2$ , respectively, P<0.001), and adiponectin was lower ( $10.1 \pm 5.8$  vs  $17.7 \pm 7.0$  microg/l, P<0.001) in non-dippers than in dippers, while no difference was found in regional fat, liver enzymes and other metabolic parameters. At multivariate analysis, factors independently associated with non-dipping were insulin (P<0.05) and adiponectin (P<0.01), with HOMA index of borderline significance (0.1>P>0.05). Non-dipping profile was a strong predictor of NAFLD (P<0.0001), independently from age and BMI.

**Conclusions.** In the absence of major risk factors for the development of NAFLD, a high prevalence of liver steatosis was associated with insulin-resistance and low adiponectin in essential hypertensive patients with non-dipping profile.