
We studied the relationships between SHBG, hormonal parameters, body mass index (BMI), body composition (body fat mass and trunk fat mass) and dietary intake in a group of 33 premenopausal women aged 27.6 ± 1.1 years (mean ± SE), with a BMI of 23.4 ± 0.8 kg/(m²), without diabetes mellitus, thyroid dysfunction or pituitary disease. They had been referred for oligomenorrhea.

Body composition was evaluated using dual X-ray absorptiometry. Food diaries kept by the patients during the week preceding the collection of blood samples were analyzed with the REGAL programme (Inra, 1991). Blood samples collected on day 2-3 of a menstrual cycle after an overnight fast were used to evaluate SHBG (with an immunoelectrodiffusion assay provided by Sebia: Hydragel-SBP), insulin, testosterone (T), dehydroepiandrosterone-sulfate (DHEAS) and free thyroid hormones (FT4, FT3).

The SHBG level was negatively correlated with BMI ($r = -0.483$, $P < 0.01$), body fat mass (in %) ($r = -0.635$, $P < 0.001$), trunk fat mass ($r = -0.645$, $P < 0.001$) and percent calorie intake provided by lipids ($r = -0.387$, $P < 0.05$). No significant correlation was found between the SHBG levels and fasting insulin levels in this group of patients.

Stepwise regression using SHBG levels as variable and BMI, body fat mass, trunk fat mass and percent intake of lipids as covariates showed that the SHBG level was mainly dependent on trunk fat mass without any significant additional influence on the other x-variables.

In conclusion, even in patients referred for oligomenorrhea, SHBG levels were dependent on anthropometric and nutritional factors among whom the main one was the degree of abdominal obesity.

### OBESITY

**Dietary and metabolic differences between overweight patients with normal or elevated PAI-1 levels.** S Dumoulin ¹, I de Glisezinski ¹, F Saint-Martin ¹, S Jamrozik ¹, P Barbe ¹, P Sié ², JP Thouvenot ³, A Bennet ¹, JP Louvet ¹ (¹ Department of Endocrinology; ² Laboratory of Haemostasis; ³ Laboratory of Nutritional and General Biochemistry, hôpital Purpan, 31059 Toulouse cedex, France)

Plasma plasminogen activator inhibitor type 1 (PAI-1), an inhibitor of fibrinolysis and a risk factor for myocardial infarction and deep venous thrombosis, is elevated in obese hyperinsulinaemic patients (Juhan-Vague [1993] Thromb Haemost 70, 138-143). The aim of the study was to determine the anthropometric, metabolic and dietary characteristics of overweight patients whose PAI-1 levels remained normal.

The patients were 64 premenopausal women aged 31.3 ± 1.2 years (mean ± SE) referred for elevated body weight, whose body mass index (BMI) ranged from 24 to 35 kg/m² (29.2 ± 0.4). No patient had dia-
None of the patients had diabetes mellitus or thyroid dysfunction; none was taking any drug or had taken an oral contraceptive during the previous 3 months.

Plasma samples taken after an overnight fast were used to evaluate PAI-1 activity (Spectrolyse pL, BioPool), insulin, cholesterol and triglycerides (TG). BMI and waist-to-hip girth ratio (WHR) were calculated for each patient. Daily dietary intake was evaluated from food diaries kept during 1 week by the patients and analyzed with the REGAL programme (Inra, 1991) for calorie intake, carbohydrates, lipids (L), proteins (P), animal (AP) and vegetal proteins, saturated, monounsaturated (MFA), polyunsaturated fatty acids, saccharose and cholesterol.

Eight patients had cholesterol levels above 2.5 g/L, and seven had TG levels above 1.5 g/L. In comparison with the 41 patients with elevated PAI-1 levels, the 23 patients with normal PAI-1 levels (< 10 U/mL) had lower TG levels (0.75 ± 0.08 vs 1.10 ± 0.08 g/L, P < 0.01), lower daily intake of P (71.4 ± 3.3 vs 82.1 ± 3.3 g, P < 0.05), L (63.3 ± 4.4 vs 77.8 ± 3.8 g, P < 0.02), AP (50.0 ± 3.3 vs 60.0 ± 2.7 g, P < 0.05), MFA (19.7 ± 1.6 vs 24.4 ± 1.2 g, P < 0.05). No significant difference was found between the two groups regarding BMI (28.4 ± 0.7 vs 29.6 ± 0.5 kg/m²), WHR (0.793 ± 0.020 vs 0.846 ± 0.016), fasting insulin levels (11.9 ± 1.9 vs 15.9 ± 2.0 mIU/L), plasma cholesterol (1.92 ± 0.10 vs 2.13 ± 0.08 g/L) and the other results of the dietary intake evaluation.

In the 64 patients, PAI-1 levels were significantly correlated with fasting insulin levels (r= +0.290, P < 0.05), TG (r= +0.317, P < 0.02), intake of P (r= +0.370, P < 0.01) and AP (r= +0.389, P < 0.01), and not with the other parameters evaluated in this study; stepwise regression showed that PAI-1 levels were dependent both on TG levels and intake of AP (P < 0.05).

In conclusion, in overweight premenopausal women, PAI-1 levels were less dependent on the degree of obesity or WHR than on the dietary and metabolic characteristics of the patients. Further studies should determine if nutritional counseling aimed at reducing TG levels and taking into account the intake of AP, could lead to a normalization of PAI-1 levels in patients who remain overweight.

The nature of changes in cardiovascular vagal and sympathetic functions is different in obese and non-insulin-dependent diabetic patients. P Valensi, NT Nguyen, S Idriss, G Karam, J Pariès, P Miossec, JR Attali (Department of Endocrinology-Diabetology-Nutrition, Jean-Verdier Hospital, Paris-Nord University, Bondy, France)

We have previously shown a high prevalence of alterations in the heart rate (HR) variability in obese and diabetic patients. The aim of this study was to investigate cardiovascular vagal tone and sympathetic response in these diseases. Sixty-two non-diabetic obese and 54 non-insulin-dependent diabetic (NIDD) patients and 35 healthy controls were investigated. Heart rate variations were analysed during three standardized tests (deep-breathing, lying-to-standing, Valsalva) and the hemodynamic response was studied during a handgrip test sustained during 5 min. The standardized tests showed a parasympathetic dysfunction in 55.7% of the obese subjects and 48.9% of the NIDD. During the handgrip test the HR increase at 1 min which results from vagal withdrawal correlated negatively with age in the controls and obese subjects taken together and correlated positively with HR variations during the lying-to-standing test. In the obese subjects without parasympathetic dysfunction, it was significantly higher than in controls, suggesting vagal hypertony at rest whereas the later increase in HR and blood pressure, which results from sympathetic activation, was particularly