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## 1449 HYPERGLYCEMIA, HYPERINSULINEMIA AND INFLAMMATION IN TYPE II DIABETICS ARE ASSOCIATED WITH SPECIFIC DEVIATIONS IN HAEMOSTATIC VARIABLES

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We studied the haemostatic variables implicated in macrovascular disease and metabolic regulation in diabetes mellitus in 54 type II diabetic subjects (48% female, age  $65 \pm 10$  y) on diet only, not using fibrates, phenformin or oestrogen-preparations, selected for a study (some after a two-week wash-out) on the effects of sulphonylurea. Levels of fasting glucose ( $10.7 \pm 2.2$  (SD) mmol/L), insulin (median 88 IQR 58-73), HbA1c ( $8.3 \pm 0.9$  %) and the inflammation marker CRP (median 2.6; IQR 1.4 - 4.9 mg/L) were elevated and evaluated for association with haemostatic variables. Values of APC-resistance (APTT-based), urokinase-type plasminogen activator and Factor VIIa were different for males and females and also evaluated in both sexes separately. Fasting glucose ( $r=0.66$ ) and post-prandial glucose ( $r=0.64$ ) correlated strongly with glycated haemoglobin. Fasting glucose only correlated statistically significantly ( $p=0.035$ ) with von Willebrand factor ( $r=0.29$ ). In females only, higher glucose was associated with lower APC-resistance. Fasting insulin correlated specifically with the variables of the t-PA-PAI-1 system, showing a positive correlation with both PAI-1 antigen ( $r=0.31$ ) and t-PA antigen ( $r=0.46$ ). In addition a correlation was noted with circulating thrombomodulin ( $r=0.35$ ). The inflammation marker CRP (only analysed when  $< 10$  mg/L) correlated with the white blood cell count ( $r=0.44$ ) and further with fibrinogen ( $r=0.55$ ) and factor VIIa ( $r=0.39$ ). The prostaglandin metabolites evaluated in urine showed a strong correlation between the amount of 6-keto-prostaglandin F $_{1\alpha}$  plus 2,3-dinor-6-ketoPGF $_{1\alpha}$  and CRP ( $r=0.41$ ); while the conversion to 2,3-dinor-6-ketoPGF $_{1\alpha}$  was decreased in association with fasting insulin ( $r=-0.36$ ). It is concluded that haemostatic deviations are relatively specifically associated with the various metabolic deviations of diabetes mellitus II, suggesting that depending upon the precise treatment given, we can also expect specific effects from the treatment.