Increased urinary glucocorticoid metabolites are associated with metabolic syndrome, hypoadiponectinemia, insulin resistance and ? cell dysfunction

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Metabolic syndrome (MetS) may have increased cortisol (F) production caused by 11?-hydroxysteroid dehydrogenase 1 (11?-HSD1) in liver and adipose tissue and/or by HPA axis dysregulation. F is then mainly metabolized by liver reductases into inactive tetrahydrometabolites (THMs). We measured THM levels in patients with or without MetS and evaluate the correlation between THMs and anthropometric and biochemical parameters. We recruited 221 subjects, of whom 130 had MetS by ATP III. We evaluated F, cortisone (E), adipokines, glucose, insulin and lipid profiles as well as urinary (24 h) F, E and THM levels. ? Cell function was estimated by the HOMA Calculator. We observed that patients with MetS showed higher levels of THMs, HOMA-IR and leptin and lower levels of adiponectin and HOMA-? but no differences in F and E in plasma or urine.

THM was associated with weight ($r = +0.44$, $p < 0.001$), waist circumference ($r = +0.38$, $p < 0.01$), glycemia ($r = +0.37$, $p < 0.01$), and triglycerides ($r = +0.1$