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Obesity is a major determinant of the association of C-reactive protein levels with the number of metabolic syndrome components in recently diagnosed, drug-naive type 2 diabetes: the ADOPT study cohort

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Background and Aims: C-reactive protein (CRP), a marker of systemic inflammation, is associated with long-term cardiovascular morbidity in patients with type 2 diabetes (T2DM) and in non-diabetic patients with the Metabolic Syndrome (MS). Elevated CRP has been found in non-diabetic obese subjects. The purpose of this study was to determine the contribution of body adiposity and glucose control to CRP levels in recently diagnosed (≤ 3 years), drug-naive, T2DM patients (fasting plasma glucose ≤ 10 mmol/l).

Materials and Methods: We examined a random representative subgroup (n=903) of the US cohort in ADOPT (A Diabetes Outcome Progression Trial). The relationship between baseline variables, National Cholesterol Education Program (NCEP) Adult Treatment Panel III MS phenotype and high-sensitivity CRP (hsCRP) levels was explored.

Results: Geometric mean hsCRP significantly increased with increasing numbers of MS components based on a test for linear trend ($P < 0.0001$; Table). Similarly, BMI ($P < 0.0001$) and HbA_{1c} ($P = 0.0004$) increased with increasing numbers of MS components. Adjustment of CRP levels for body adiposity abolished the association between CRP and the number of MS components ($P = 0.237$; Table), whereas adjustment for HbA_{1c} maintained the association ($P < 0.0001$).

	# NCEP MS Components				
(N)	1 (30)	2 (94)	3 (219)	4 (332)	5 (228)
hsCRP*, mg/l [†]	1.7 (1.1, 2.5)	2.4 (1.9, 3.0)	3.6 (3.1, 4.2)	4.0 (3.6, 4.5)	4.7 (4.1, 5.4)
BMI**, kg/m ^{2†}	25.5 (23.8, 27.1)	29.3 (28.3, 30.3)	33.1 (32.5, 33.8)	34.3 (33.8, 34.9)	36.1 (35.5, 36.7)
BMI-Adjusted CRP*, mg/l [†]	3.2 (2.3, 4.7)	3.4 (2.8, 4.2)	3.8 (3.3, 4.3)	3.8 (3.4, 4.2)	3.9 (3.5, 4.5)
*Geometric Mean, **Mean, [†] (95% CI)					

Conclusion: We conclude that CRP, a marker of systemic inflammation, is strongly related to the number of MS components; however, in recently diagnosed, drug-naive T2DM patients this relationship is determined by body adiposity and not by glucose control.