**References**

1. Goldfine AB, Moses AC: Syndrome of extreme insulin resistance. In *Joslin's Diabetes Mellitus.* 14th ed. Kahn CR, Weir GC, King GL, Jacobson AM, Moses AC, Smith RJ, Eds. Philadelphia, Lippincott Williams & Wilkins, 2005, p. 493–504
2. Pittas AG, Joseph NA, Greenberg AS: Adipocytokines and insulin resistance. *J Clin Endocrinol Metab* 89:447–452, 2004
3. Kubota N, Terauchi Y, Kubota T, Kumagai H, Itoh S, Satoh H, Yano W, Ogata H, Tokuyama K, Takamoto I, Mineyama T, Ishikawa M, Moroi M, Sugi K, Yamauchi T, Ueki K, Tobe K, Noda T, Nagai R, Kadowaki T: Pioglitazone ameliorates insulin resistance and diabetes by both adiponectin-dependent and -independent pathways. *J Biol Chem* 281:8748–8755, 2006
4. Ullrich A, Bell JR, Chen EY, Herrera R, Petruzzelli LM, Dull TJ, Gray A, Coussens L, Liao YC, Tsubokawa M, Mason A, Seeburg PH, Grunfeld C, Rosen OM, Ramachandran J: Human insulin receptor and its relationship to the tyrosine kinase family of oncogenes. *Nature* 313:756–761, 1985
5. Kuzuya H, Matsuura N, Sakamoto M, Makino H, Sakamoto Y, Kadowaki T, Suzuki Y, Kobayashi M, Akazawa Y, Nomura M, Yoshimasa Y, Kasuga M, Goji K, Nagataki S, Oyasu H, Imura: Trial of insulin-like growth factor I therapy for patients with extreme insulin resistance syndromes. *Diabetes* 42:696–705, 1993

# A Continuous Metabolic Syndrome Risk Score

## Utility for epidemiological analyses

This study was designed to validate a continuous metabolic syndrome risk score (cMSy) using the International Diabetes Federation risk factors (1). Increasing evidence supports using a cMSy instead of a binary definition for epidemiological analyses: *1*) dichotomizing continuous outcome variables reduces statistical power (2); 2) cardiovascular risk is a progressive function of several metabolic syndrome (MSy) risk factors, eliminating the need to dichotomize these factors (3); and *3*) cardiovascular and diabetes risk increase progressively with increasing numbers of MSy risk factors, eliminating the need to dichotomize MSy (3–4).

The National Institute of Statistics randomly selected a community sample of 18- to 75-year-old Flemish adults. In total, 571 men (aged 46.7 ± 11.2 years) and 449 women (aged 45.8 ± 10.8 years) without cardiovascular disease and diabetes, tested between October 2002 and April 2004, were included.

Calculation of cMSy involved two steps. First, principal component (PC) analysis (varimax rotation) was applied to the normalized risk factors to derive PCs representing large fractions of MSy variance, revealing two PCs (eigenvalue ≥1.0). In men, PC1 and PC2 explained 33 and 28% of variance, respectively (loadings PC1 [PC2]: waist circumference 0.51 [0.55], triglycerides 0.82 [0.16], HDL cholesterol −0.85 [0.09], blood pressure 0.12 [0.72], and glucose −0.08 [0.73]). In women, PC1 and PC2 explained 33 and 27% of variance, respectively (loadings PC1 [PC2]: waist circumference 0.61 [0.54], triglycerides 0.42 [0.49], HDL cholesterol 0.12 [−0.90], blood pressure 0.83 [0.01], and glucose 0.62 [0.04]). Second, cMSy was computed by summing both individual PC scores, each weighted for the relative contribution of PC1 and PC2 in the explained variance. Resulting cMSy was 0 ± 1.42 in men and 0 ± 1.41 in women.

cMSy was higher ($P < 0.001$) in subjects with MSy as defined by the International Diabetes Federation (1) (men [12.8%]: 2.03 ± 1.00, women [8.5%]: 2.63 ± 1.28) versus subjects without (men: −0.30 ± 1.21, women: −0.24 ± 1.16). Moreover, cMSy increased progressively (Tukey's honestly significant difference comparison, $P < 0.001$) with increasing numbers of risk factors in men (0 [30.1%]: −1.21 ± 0.96; 1 [33.8%]: −0.26 ± 0.87; 2 [21.2%]: 0.67 ± 0.84; 3 [11.2%]: 1.76 ± 0.73; and ≥4 [3.7%]: 3.04 ± 0.94) and women (0 [47.7%]: −0.96 ± 0.79; 1 [28.3%]: 0.16 ± 0.82; 2 [16.3%]: 1.21 ± 0.82; 3 [5.3%]: 2.17 ± 0.81; and ≥4 [2.4%]: 4.09 ± 0.99).

cMSy is a more appropriate (2–4) and valid alternative for epidemiological analyses, although the binary definition (1) remains useful for clinical practice.

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**References**

1. The IDF consensus worldwide definition of the metabolic syndrome [article online], 2005. Available from http://www. idf.org/webdata/docs/Metabolic_syndrome_definition.pdf. Accessed 5 May 2005
2. Ragland DR: Dichotomizing continuous outcome variables: dependence of the magnitude of association and statistical power on the cutpoint. *Epidemiology* 3:434–440, 1992
3. Kahn R, Buse J, Ferrannini E, Stern M: The metabolic syndrome: time for a critical appraisal: joint statement from the American Diabetes Association and the European Association for the Study of Diabetes. *Diabetes Care* 28:2289–2304, 2005
4. Klein BEK, Klein R, Lee KE: Components of the metabolic syndrome and risk of cardiovascular disease and diabetes in Beaver Dam. *Diabetes Care* 25:1790–1794, 2002

# Reversibility of Antipsychotic Treatment–Related Diabetes in Patients With Schizophrenia

## A case series of switching to aripiprazole

At present, antipsychotic drugs are not specifically referred to in the list of substances that can induce diabetes in the most recent American Diabetes Association guidelines (1), but there is a growing body of evidence that antipsychotic drugs might have diabetogenic properties (2,3). At present, there are no clear guidelines on what the best therapeutic strategies are when diabetes is detected during treatment with antipsy-