## **ERRATUM**

## Erratum to: Identity-by-descent-based heritability analysis in the Northern Finland Birth Cohort

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The sixth column of Table 1 ("Associated SNPs") has results taken from Sabatti et al. (2009), however an error was made in the processing of the results. Also, the results derived from Supplementary Table 4 of Sabatti et al. (2009), not from Supplementary Table 1 as originally stated. The corrected Table 1 is as follows:

The online version of the original article can be found under doi:10.1007/s00439-012-1230-y.

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Table 1 Heritability estimates

Trait <sup>a</sup>	GCTA <sup>b</sup>	VC IBD <sup>c</sup>	Zuk et al.d	Twin studies <sup>e</sup>	Associated SNPs <sup>f</sup>
CRP	0.02 (0.06) <sup>g</sup>	0.08 (0.16)	0.00 (0.21)	0.56 (0.07) [W]	0.041
Glucose	0.18 (0.07)**,h	0.39 (0.16)**	0.51 (0.22)*	0.67 (0.06) [S]	0.0165
Insulin	0.07 (0.07)	0.04 (0.17)	0.03 (0.22)	0.49 (0.05) [S]	0.0056
Triglycerides	0.08 (0.07)	0.00 (0.17)	0.00 (0.22)	0.65 (0.05) [W]	0.0431
HDL	0.19 (0.07)**	0.46 (0.17)**	0.27 (0.22)	0.76 (0.06) [S]	0.0638
LDL	0.29 (0.07)***	0.54 (0.17)***	0.10 (0.22)	0.78 (0.05) [S]	0.06
BMI	0.16 (0.07)**	0.00 (0.16)	0.00 (0.21)	0.80 (0.03) [W]	0.0055
Diastolic	0.08 (0.07)	0.21 (0.16)	0.09 (0.21)	0.51 (0.06) [W]	0.00
Systolic	0.06 (0.06)	0.06 (0.16)	0.06 (0.21)	0.47 (0.06) [W]	0.00

<sup>&</sup>lt;sup>a</sup> Traits have been transformed to adjust for covariates and achieve approximate normality, as described in Subjects and Methods. Results from the twin studies have not necessarily had the same transformations/adjustments

## References

Sabatti C, Service SK, Hartikainen AL, Pouta A, Ripatti S et al (2009) Genome-wide association analysis of metabolic traits in a birth cohort from a founder population. Nat Genet 41:35–46

Souren NY, Paulussen AD, Loos RJ, Gielen M, Beunen G et al (2007) Anthropometry, carbohydrate and lipid metabolism in the East Flanders Prospective Twin Survey: heritabilities. Diabetologia 50:2107–2116 Wessel J, Moratorio G, Rao F, Mahata M, Zhang L et al (2007) C-reactive protein, an 'intermediate phenotype' for inflammation: human twin studies reveal heritability, association with blood pressure and the metabolic syndrome, and the influence of common polymorphism at catecholaminergic/beta-adrenergic pathway loci. J Hypertens 25:329–343



<sup>&</sup>lt;sup>b</sup> Estimates from the GCTA software using autosomal NFBC data

c Variance components approach using IBD-based estimates of relatedness from the autosomal NFBC data

<sup>&</sup>lt;sup>d</sup> Regression approach of Zuk et al. using autosomal NFBC data

<sup>&</sup>lt;sup>e</sup> Indicative estimates of heritability from twin studies taken from previous literature. Source is denoted by [W] (Wessel et al. 2007) or [S] (Source et al. 2007). These estimates can differ from the true narrow-sense autosomal heritability of these traits in Northern Finland due to differences in environmental variances, differences in genetic make-up, incorporation of interaction effects or shared environment into family-based estimates, and contribution of the X chromosome

f Estimates of the proportion of trait variation in the NFBC data explained by SNPs significantly associated in the NFBC study or from previous studies are taken from Supplementary Table 4 of Sabatti et al. (2009). These estimates include effects from the X chromosome

<sup>&</sup>lt;sup>g</sup> Estimates of heritability are given with standard errors in parentheses

<sup>&</sup>lt;sup>h</sup> Statistical significance of estimates from this study are indicated by \* (0.01 , \*\* <math>(0.001 , and \*\*\* <math>(p < 0.001)