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Letter to the Editor/Erratum

Question Marks Left Over a Quantitative Assessment of *Apolipoprotein C3* Gene Polymorphisms

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Dear Editor,

On the occasion of the 2012 annual meeting of the American Society of Human Genetics in San Francisco, the scientific press concluded to a sobering "genetic influences on disease remain hidden" after discussing, among others, the progress made in the field of cardiovascular genetics [1]. In a recent attempt to resolve some of the inconsistent findings, Zhang and coworkers [2] have presented a quantitative analysis of *APOC3* variants in coronary heart disease. Unfortunately, only a fraction of the previously published data have been considered and, using the authors' inclusion criteria, over 10000 alleles are missing from the investigation [3-8]. What is more, incorrect allele counts have led to biased effects for the *Sst*I polymorphism, causing the risk-enhancing allele to become protective [9] and *vice versa* [10]. Allele counts for the T-455C variant also differ from the published data [11] and are further compromised by duplicates from overlapping samples [11,12]. With regard to both T-455C and C-482T, the vast majority of allele frequencies reported in Table 1 of the article [2] are either in error [11,13-18], missing [12], or entirely fictional [19]. Finally, failure to identify C3175G as a synonym of the *Sst*I polymorphism has led to the omission of more alleles from a publication which served to extract data on T-455C and C-482T [13].

On the whole, the article calls for numerous issues to be ironed out prior to claiming, or to refuting, significant effects of the three *APOC3* variants on coronary heart disease susceptibility.

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Conflict of interest

The author reports no conflict of interest.

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Erratum to: Letter to the Editor

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Association Between Apolipoprotein C-III Gene Polymorphisms and the Risk of Coronary Heart Disease: a Meta-analysis

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In light of Dr. Sands' letter, we have reanalyzed data from our meta-analysis of the association between apolipoprotein C-III gene polymorphisms and coronary heart disease (CHD)[1].Our original database searches did not recover several references cited by Dr. Sand[2-5], which we have now included, and we were unable to extract data useful for our analysis from two other references [6-7]. We have reexamined additional studies referred to by Dr.Sand [8-14] and recalculated our results accordingly.

The new calculations still show an association between the APOC3 SstI polymorphism and CHD under allelic contrast (P <0.0001, OR = 1.17, 95% CI = 1.08-1.27), dominant genetic (P = 0.001, OR = 1.17, 95% CI = 1.07-1.29), and recessive genetic (P = 0.01, OR = 1.36, 95% CI = 1.07-1.73) models; between the APOC3 T-455C polymorphism and CHD under allelic contrast (C vs. T, P = 0.007, OR = 1.19, 95% CI = 1.05-1.35) and dominant genetic (CT+CC vs. TT, P = 0.0004, OR = 1.26, 95% CI = 1.11-1.43), but no longer recessive genetic (CC vs. CT+TT, P = 0.12, OR = 1.25, 95% CI = 0.95-1.66) models; and no association between the APOC3 C-482T polymorphism and CHD under allelic contrast (T vs. C, P = 0.59, OR =1.02, 95%CI = 0.95-1.10), dominant genetic (TT+TC vs. CC, P = 0.17, OR = 1.08, 95% CI = 0.97-1.20)or recessive genetic (TT vs. TC+CC, P = 0.41，OR = 0.94, 95% CI = 0.82-1.09) models. Our meta-analysis suggests that the APOC3 SstI polymorphism significantly increases, the APOC3 T-455C polymorphism may increase, and the APOC3 C-482Tpolymorphism shows no association with CHD susceptibility.

We sincerely apologize for the errors in our article.

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