Quantitative effects of common genetic variations in the 3'UTR of the human LDL-receptor gene and their associations with plasma lipid levels in the Atherosclerosis Risk in Communities study

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In this paper, we concluded that a 3'UTR sequence of the human *LDLR* gene which causes higher expression of a reporter gene in cultured cells is associated in Caucasians with plasma lipid profiles indicative of higher cardiovascular risk. This conclusion was based on the observation that expression of a reporter gene containing type II or type III haplotype sequences was

significantly lower (by about 30%) than that obtained when the reporter gene contained the type I haplotype sequence.

During the course of testing the expression of additional reporter constructs containing type IV and V human haplotype sequences, we discovered that the previous reporter gene constructs containing type II and type III sequences were mistakenly made with a wrong vector that lacks polyA addition signal elements.

After correcting this error in the earlier type II and III constructs, we now find that there are no functional differences between the five common 3'UTR haplotypes, I through V, in either mRNA levels of the reporter gene or protein levels (measured by the fluorescence) as shown in the following table, which should replace Fig. 2b, c.

Haplotypes		Reporter gene expression	
		Fluorescence	mRNA
I	GGCGCA	100 ± 13	100 ± 6
II	AGCACG	106 ± 13	133 ± 22
III	GGCGTA	102 ± 14	139 ± 15
IV	GGGGTA	108 ± 13	121 ± 13
V	GGGGCA	109 ± 13	133 ± 17

Haplotypes are nucleotide positions 44243, 44332, 44506, 44695, 44857, and 44964 (rs14158, rs3826810, rs2738464, rs2738465, rs1433099, and rs2738466). Expression is presented as mean ± SE of three experiments taking the mean of reporter gene containing type I sequence as 100

Our typing of the SNP sites, C44857T and A44964G, and the associations we reported between the inferred haplotypes and plasma lipid levels in the Atherosclerosis Risk in Communities (ARIC) Study population are

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not affected by this mistake. We conclude that these observed associations are not due to differences in the functionality of this region of DNA but are due to other factors that are in linkage disequilibrium with the

3'UTR polymorphisms in Caucasians, but not in African Americans.

The authors greatly regret any inconvenience caused by our error.