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RETRACTION

Non-random, individual-specific methylation profiles are present at the sixth CTCF binding site in the human *H19*/*IGF2* imprinting control region.

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After publication of the article, it has been brought to our attention by Dr. Richard Scott and Prof. Nazneen Rahman at the Institute of Cancer Research (Sutton, UK) that the observed three profiles and their heritability could be fully explained by a biased amplification caused by an SNP (rs2107425) underlying the 21st of 24 bases (calculated from the 3′ terminus) of the reverse primer. Biased amplification due to a SNP so close to the 5′ end of an amplification primer is astonishing, the more as we had tried in vain to use the employed polymerase for allele-specific PCR on bisulphite treated DNA for some time. Heterogeneous methylation patterns had previously been observed by other groups in the same region [Sandovici I, Hum Mol Genet, 1569 (2003); De Castro Valente Esteves LI, Int J Mol Med 397 (2006)].

Due to an unfortunate chain of events the SNP in question was not found in our sequencing screen to identify SNPs linked to the profile. All tested samples were homozygous G/G at this nucleotide and although we changed the forward primer we did not change the reverse primer as the nucleotides complementary to the 3′ terminus are highly specific for the sixth CTCF site. We tested the PCR amplifications for the presence of preferential amplification but unfortunately again used DNAs homozygous for the perfectly matched allele. We genotyped about 20 SNPs in this region that displays a high degree of linkage disequilibrium. Regrettably, the SNP in question seems not be in LD with the neighbouring SNPs that were genotyped in all samples.

After being contacted by Dr. Richard Scott and Prof. Nazneen Rahman we genotyped ∼200 individuals and correlated the methylation profile to the SNP. An individual which is G/G for the SNP always gives the expected median methylated profile with a methylation degree of ∼40%. Heterozygous individuals can give median-, hypo- and hypermethylated methylation profiles and A/A individuals (very rare genotype) do either not work or yield a median-methylated profile.

We then used a primer complementary to the A allele for analysis of the methylation profile and indeed hypo-methylated samples become hyper-methylated and vice versa. Using a degenerate primer for the polymorphic position methylation profiles display much less variability approaching the expected values. We then used a primer avoiding the SNP and confirmed the results that a single methylation profile is found at the sixth CTCF site. The SNP also perfectly explains the observed heritability.

We must conclude that the three methylation profiles at the sixth CTCF site are due to a technical artefact. The analysis of the PCR products by pyrosequencing or any other used technology yielded the true values but based on the biased PCR amplification.

No data in the article was manipulated nor did any of the authors commit any form of misconduct. We have acted to the best of our knowledge at the time but due to an unfortunate chain of events did miss the responsible polymorphisms. We sincerely regret this error and as this region is used in clinical diagnostics, we think it necessary to clarify the situation and respectfully retract this paper.