Letter to the Editor

“Relationship between the acylation-stimulating protein gene and coronary heart disease in the Xinjiang Uygur and Han populations of China”, published in Genetics and Molecular Research 13 (2): 2638-2644 (2014)

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Dear Editor,

As a scientist who works on obesity and immunity, especially the complement system, I read the paper titled “Relationship between the acylation-stimulating protein gene and coronary heart disease in the Xinjiang Uygur and Han populations of China” published in Genetics and Molecular Research 13 (2): 2638-2644 (2014).

I was surprised when I read about the “acylation-stimulating protein (ASP) gene” and its correlation with heart disease. As the authors stated in their abstract, “ASP is identical to C3a desArg, and produced through the interaction of the precursor protein C3, Factor B, and adipsin (also known as Factor D), components of the alternative complement immune pathway, which are secreted by the adipose tissue”. This is correct. This also means that there is

noacylation-stimulating protein gene, as this protein is made post-transcriptionally through the interaction of Complement C3, Factor B and Factor D. The “ASP gene” is actually the gene of precursor protein C3, which is also the main component of the complement system of innate immunity; it is directly and strongly linked with CHD. In the paper, the polymorphism is described as only affecting ASP, which is incorrect. The concept of an ASP gene is misleading. In reality, the authors have linked a polymorphism in the C3 gene with coronary heart disease in a specific population; this polymorphism could also potentially alter ASP function.

I hope my criticism is taken positively, as I only aim to correct what I believe is a genuine mistake, in order to avoid scientific inaccuracy.