Reason for Limitations of Heterophilic Blocking Tube Use on Certain Beckman Coulter Access Assays

To the Editor:
A recent Technical Brief by Ellis et al. (1) showed the usefulness of heterophilic blocking tubes (HTBs) in identifying false-positive test results on several analyzers, including the Roche Elecsys and Abbott Architect. The authors of the Technical Brief went on to suggest that HTB treatment led to particular overrecoveries in several assays on the Beckman Coulter Access analyzer. It is important to know that the reason for this observed limitation is the unique design of those particular Access assays, which according to the Technical Brief (1) “contain solid-phase goat–anti-mouse [monoclonal antibody complexes].” This design (which can be identified by the Access package inserts) prohibits the use of any blocker containing murine or mouse components (e.g., HTBs). For those assays that contain an anti-mouse component, it is advisable to check for false positives by determining whether results are linear on dilution.

Reference

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Hypertriglyceridemia: Interaction between APOE and APOAV Variants

To the Editor:
Schaefer et al. (1) recently reported an association between a distinct combination of variants in the apolipoprotein E (APOE) and APOAV genes and hypertriglyceridemia. Among 170 hypertriglyceridemic (HTG) patients, all carriers of APOE22 (n = 7) had at least 1 APOAV Trp19 allele, but this combination was not found in controls with triglyceride (TG) concentrations within the reference interval.

APOE is a structural component of TG-rich lipoproteins; it serves as a ligand for lipoprotein receptors and plays an important role in the catabolism of remnant particles (2, 3). Of the 3 common apoE isoforms, apoE4 (Cys112>Arg) and apoE2 (Arg158>Cys) differ from the commonest isoform, apoE3, by a single amino acid substitution. The APOE4 allele has been shown to be associated with increased plasma cholesterol and with an increased risk of coronary heart disease. In contrast, the APOE2 allele is associated with low plasma concentrations of cholesterol and is believed to be protective against coronary heart disease [reviewed in Refs. (2, 3)].

APOAV variants (e.g., T–1131>C and Ser19>Trp) play an important role in modulating plasma TG concentrations in humans (4). An association between the APOAV Ser19>Trp polymorphism and TG concentrations has been found in many population samples, but the total impact of this variant is not the same in different ethnic groups (4–7). The Trp19 allele was found to be associated with extremely high concentrations of plasma TG (8), and Trp/Trp homozygotes have a higher risk of myocardial infarction (6). Recently, it was reported that apoAV interacts physically with lipoprotein lipase and significantly increases its activity (9). Computational analysis of the apoAV protein suggests that the change of Ser19 to Trp could lead to impaired export of apoAV from the liver (10).

Using a previously described method, we have analyzed (by PCR and restriction analysis) APOE and APOAV variants (T–1131>C, Ser19>Trp, and Val153>Met) (6, 11) in 2559 unrelated Caucasians. This population sample included 1191 males [mean (SD) age, 49.2 (10.8) years; TGs, 2.0 (1.3) mmol/L; total cholesterol, 5.8 (1.0) mmol/L; body mass index, 28.2 (4.0) kg/m²] and 1368 females [age, 48.8 (10.6) years; TGs, 1.5 (0.8) mmol/L; total cholesterol, 5.8 (1.2) mmol/L; body mass index, 27.6 (5.5) kg/m²] recruited as a representative 1% population sample in 9 Czech districts according the WHO protocol (12). Additionally, 111 HTG individuals [TGs >10 mmol/L; mean (SD), 22.4 (24.1) mmol/L; age, 51.4 (9.6) years] and 8 individuals with type III hyperlipidemia [all APOE22 genotype; TGs, 6.7 (6.7) mmol/L; age, 56.2 (13.8) years].

As described before, we have found an association between increased concentrations of plasma TG and the presence of the Trp19 allele (6). The same allele was also found to be more frequent in HTG patients (8). In contrast to Schaefer et al. (1), we found no significant interaction between the APOAV Trp19 variant, APOE2, and hypertriglyceridemia: of 111 HTG patients, 4 were carriers of the APOE22 genotype and 1 of these had the APOAV Trp19 allele. Of the