tracted solely with a buffer (5). The interference appeared in premature neonates and neonates with perinatal complications. One of the suspected causes was cross-reactivity of the antisera with 17-hydroxyprogrenolone sulfate (1.66%), there being a relatively high amount of this steroid in the sera of premature neonates secondary to their lower activity of 3β-hydroxysteroid dehydrogenase and higher activity of sulfokinase. Makela and Ellis (4) were unable to decrease assay interferences by incubating sera with sulfatase/glucuronidase preparation, and they found no correlation between the concentration of 17-OHP and the gestational or chronological age of the neonate. Their findings do not support the hypothesis about steroid sulfate interference. On the other hand, they also found that the most effective solvents in the extraction of neonates' sera were the ones that are the most effective in solubilizing steroid sulfates. When the same group of authors tried to identify the interfering substance by mass spectrometry (6), insufficient material was available for definite identification, but the results suggested that it could be a steroid sulfate.

We could not identify the nature of interfering substances in AF, although indications are that at least a part of them could be protein-bound. Proteins of AF originate at least partly from maternal serum by simple filtration through a semipermeable membrane. However, the nature of the possible binding protein is at present not known. Despite the high values obtained with the DPC kit in comparison with other investigated kits, there is not much difference between the values obtained with and without ether extraction, in contrast to the results obtained in the previous report for neonatal sera. The rather poor correlation between 17-OHP values obtained with the DPC direct and extraction methods suggests significant interindividual variations in the quantity of interfering material. In one affected pregnancy, 17-OHP values in AF and fetal plasma determined with the DPC kit after ether extraction were in the reference range.

In conclusion, because of the as yet unresolved problem of interference during 17-OHP determination in AF, we do not recommend use of the DPC kit for prenatal diagnosis of CAH.

References

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A spokesman for Diagnostic Products Corp. responds:
To the Editor:
Plavšić et al. in their letter conclude that because of unidentified interferences they would not recommend the Diagnostic Products Corp. (DPC) Coat-A-Count 17a-OH Proges-terone kit for the prenatal diagnosis of CAH. In this regard, I must first point out that the intended use of this kit, as stated in the kit insert, is for determinations in serum and plasma samples. As the manufacturer of this kit, we do not have data for determinations in amniotic fluid and therefore make no claims about such an application. Indeed, amniotic fluid may not be an acceptable specimen type for this assay because of possible matrix effects and (or) interfering cross-reacting substances. However, there also are inadequacies with the supporting data cited, which should be understood. Results are cited for an amniotic-fluid specimen obtained from a patient carrying a fetus that was subsequently confirmed to have CAH. Assays performed both directly and with reconstituted ether extracts yielded results that were within the reference ranges established with the DPC kit for unaffected pregnancies, and therefore did not predict CAH. Although matched results were not provided, the authors, based on historical data, claim that another commercially available assay (used with an extraction step) yielded clinically accurate results and, by implication, would have also identified this particular specimen as abnormal.

Clearly, conclusions about how two assays compare are unwarranted in the absence of matched results. Furthermore, the buffer matrix used to reconstitute the dried ether extracts, 50 mmol/L sodium phosphate buffer, pH 7.4, containing 1 g of bovine serum albumin per liter (information supplied by Plavšić et al.) bears no resemblance to the "human serum-based standards" contained in the DPC kit. This raises questions about possible confounding matrix effects. The generally accepted practice is to use the assay's zero calibrator or a suitable specimen (consistent with the assay's intended use) for reconstituting extracts.

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Prognostic Value of Rate of Decline of Creatine Kinase and MB-Isoenzyme Activity after Acute Myocardial Infarction

To the Editor:
The rate of decline of total creatine kinase (CK; EC 2.7.3.2) activity during the course of an acute myocardial infarction (AMI) was shown by Weingeber et al. (1) to be of prognostic value. In their study, patients in whom CK values declined by <50% of their peak value within 48 h had an increased risk of re-infarction or dying. This prognosis was not related to sex, previous AMI, site of infarction, or coronary risk factors. We wanted to confirm these findings in our populations and determine if there was additional value/specificity in monitoring the rate of decline of the more heart-specific isoenzyme, CK-MB, within the first 24 h of patient treatment.