method (on average by 22.0%; \( P < 0.0001 \) by Wilcoxon signed-rank test) and based on the 99th percentile values for each assay, 9 discordances were found between assays for values within the reference interval vs increased values.

The ADVIA TnI-Ultra method showed no interference from dilutions with plasma samples that contained high concentration of triglycerides (6.6 g/L, final dilution 1:128; \( y = -0.044 + 0.14x, \ n = 8, \ R = 0.99 \)) or hemoglobin (1.47 g/L, final dilution 1:4996; \( y = 0.04 + 0.060x, \ n = 13, \ R = 0.99 \)). No apparent positive interference was seen in 58 patients with symptomatic rheumatoid arthritis [10 men and 48 women, mean (SD) age 60.8 (10.2) years] with a mean (SD) cTnI concentration was not increased 0.017 (0.023) kIU/L), because the mean (SD) cTnI concentration 1:4996; \( y = 0.04 + 0.060x, \ n = 13, \ R = 0.99 \). The present study indicates that the ADVIA TnI-Ultra method meets the quality specifications recommended by NACB and IFCC Committee for the Standardization of Cardiac Damage (5). Grant/funding support: None declared. Financial disclosures: None declared.

References


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Midtrimester Amniotic Fluid Adiponectin in Normal Pregnancy

To the Editor:

Adiponectin is an adipose tissue-derived protein with important metabolic effects and a strong correlation with insulin sensitivity. In pregnancy there is a progressive increase of insulin resistance, whereas plasma adiponectin concentrations decrease in the 2nd half of gestation (1). In contrast, cord plasma adiponectin concentrations increase throughout gestation (2). Nothing is known about the concentration, origin, or role of amniotic fluid adiponectin, particularly in relation to amniotic insulin. Therefore we evaluated adiponectin and insulin concentrations in the midtrimester amniotic fluid of women with normal pregnancies.

Beginning January 1, 2006, we selected the first 50 pregnant women who underwent a midtrimester amniocentesis for prenatal diagnosis (15–18 weeks gestation) and were found to have a normal pregnancy, defined as an uncomplicated pregnancy with full-term delivery of an infant of adequate size for gestational age. The study was approved by the institutional review board, and all women gave written informed consent.

Amniotic fluid samples were obtained by transabdominal amniocentesis and collected in 15 mL dry tubes. All samples were free of blood contamination, as estimated by microscopic inspection. The samples were immediately centrifuged for 10 min at 3000 g and stored at −70 °C. Plasma EDTA samples were centrifuged for 15 min at 10000 g within 30 min of collection and stored at −70 °C. Plasma samples required 200-fold dilution before assay. The adiponectin concentration was measured by immunoenzymatic assay (R&D Systems). The intra- and interassay imprecision (CVs) for adiponectin at a concentration of 15.0 µg/L were 3.5% and 5.5%, respectively. The intra- and interassay imprecision values (CVs) for insulin at a concentration of 4.0 mIU/L were 3.3% and 5.6%, respectively.

Amniotic and plasma adiponectin and amniotic insulin concentrations are presented as the median and the 25th–75th percentile range; all other variables are presented as the mean (SD). The Mann–Whitney U-test was used to compare continuous variables between the 2 groups. Univariate correlations between amniotic fluid adiponectin and all the other variables were assessed using the Spearman test. The statistical analysis was performed using SPSS 13.0 (SPSS Inc.). All tests were 2-sided; a \( P \) value <0.05 was considered statistically significant.

The clinical characteristics of pregnant women are reported in Table 1. Median adiponectin amniotic fluid values were 26.8 (13.9–37.3) µg/L, but when we dichotomized for sex, there was a significant difference \( (P = 0.01) \) between female 34.8 (18.2–48.7) µg/L and male fetuses 18.2 (13.4–26.8) µg/L. Univariate analysis

<p>| Table 1: Characteristics of Pregnant Women and Median Amniotic Fluid Adiponectin and Insulin Concentrations |
|---------------------------------------------|---------------------------------------------------------------|</p>
<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Median (25th–75th Percentile)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adiponectin (µg/L)</td>
<td>26.8 (13.9–37.3)</td>
</tr>
<tr>
<td>Insulin (µg/L)</td>
<td>4.0 (3.5–5.5)</td>
</tr>
<tr>
<td>Gender</td>
<td>Female/Male</td>
</tr>
<tr>
<td>Adiponectin (µg/L)</td>
<td>34.8 (18.2–48.7)/18.2 (13.4–26.8)</td>
</tr>
</tbody>
</table>

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The clinical characteristics of pregnant women are reported in Table 1. Median adiponectin amniotic fluid values were 26.8 (13.9–37.3) µg/L, but when we dichotomized for sex, there was a significant difference \( (P = 0.01) \) between female 34.8 (18.2–48.7) µg/L and male fetuses 18.2 (13.4–26.8) µg/L. Univariate analysis
showed a positive correlation between amniotic adiponectin and insulin \((r = 0.47, P = 0.001)\) and also with gestational age at amniocentesis \((r = 0.31, P = 0.03)\); in contrast amniotic fluid adiponectin did not correlate with plasma adiponectin \((r = 0.07, P = 0.6)\), with maternal age \((r = 0.01, P = 0.9)\), with maternal body mass index (BMI) \((r = -0.07, P = 0.5)\), with gestational age at delivery \((r = -0.15, P = 0.1)\), or with birth weight \((r = 0.05, P = 0.7)\).

To our knowledge, this is the first study that deals with amniotic fluid concentrations in normal pregnancy at the time of midtrimester amniocentesis. Our results suggest that amniotic adiponectin might be of fetal origin. Maternal origin seems unlikely because there was no correlation between plasma and amniotic adiponectin or between amniotic adiponectin and maternal age or BMI. In addition, Corbetta et al. \((3)\) found no adiponectin in placental tissue. Thus, a fetal origin of amniotic adiponectin appears likely; insulin production by the human fetus from 11 weeks of gestational age has been demonstrated in an experimental study \((4)\), and we have shown a strong correlation between amniotic adiponectin and amniotic insulin. Furthermore, the statistically significant difference between female and male fetuses supports the theory that amniotic adiponectin is of fetal origin. Clinical and experimental studies have demonstrated that the sex dimorphism of adiponectin concentrations might be caused by testosterone-induced inhibition of its secretion from adipocytes \((5)\).

In conclusion, based on our study of 50 normal pregnancies at the time of midtrimester amniocentesis, we suggest that adiponectin found in amniotic fluid is of fetal origin.

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References

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**Correction**

In the article entitled “Diagnosis of α1-Antitrypsin Deficiency: An Algorithm of Genotyping, Phenotyping, and Quantitation” by Melissa R. Snyder, Jerry A. Katzmann, Malinda L. Butz, Ping Yang, D. Brian Dawson, Kevin C. Halling, W. Edward Highsmith, and Stephen N. Thibodeau (Clin Chem 2006;52:2236–42; DOI: 10.1373/clinchem.2006.072991), the name of one of the coauthors of the study was inadvertently omitted from the author list. The correct author list should read: Melissa R. Snyder, Jerry A. Katzmann, Malinda L. Butz, Carmen Wiley, Ping Yang, D. Brian Dawson, Kevin C. Halling, W. Edward Highsmith, and Stephen N. Thibodeau.

Dr. Carmen Wiley’s current affiliation is Division of Laboratory Medicine, Marshfield Clinic, Marshfield, WI 54449.

The authors regret the oversight. Dr. Wiley’s contributions to the manuscript occurred at the initiation of the study and included participation in study design and assay development.

Dr. Wiley has read and agrees with the content and conclusions in the published manuscript.

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