In Table 4 of their article it is clearly shown that intake of 16:1t was significantly positively associated with birth-weight-for-gestational-age z scores. Moreover, adjustment for race, income, parity, education, smoking status, age, prepregnancy BMI, physical activity, television viewing, and fish consumption resulted in no change in the strength of the association (indeed, in the unadjusted and adjusted models both β and 95% CIs were identical to the second decimal value). Although the statement that the association was limited to 16:1t—and to one isomeric of trans octadecadienoic acid, 18:2ct—is literally true, evaluation of the 18:1r data presented in Table 4 may allow further considerations. In the unadjusted model, 18:1r was significantly and positively associated with birth-weight-for-gestational-age z scores (β: 0.08; 95% CI: 0.01, 0.15) and significantly negatively associated to the OR for small-for-gestational-age birth (OR: 0.66; 95% CI: 0.45, 0.96). After adjustment for confounding variables, however, the significant associations disappeared. It is somewhat surprising that within the same data pool adjustment for the same confounding variables had no effect at all on the association of 16:1t to fetal growth, whereas the same adjustment caused decisive changes in the association of 18:1r to fetal growth and to the OR for small-for-gestational-age birth.

Despite the lack of correlation between 18:1r and fetal growth in the full (adjusted) model of analysis in Cohen et al’s study (1), I still think that 18:1r may have dietary relevance in the perinatal period. One important factor to be taken into consideration is that the relation of 18:1t to long-chain PUFAs (LC-PUFAs) in biological samples collected in the perinatal period. During the last decade we carried out studies investigating the relation of TFA and LC-PUFAs in blood lipids of pregnant women (2), in cord vessel wall lipids of healthy term infants (3), and in human milk lipids of women donating samples at the sixth week (4) and sixth month of lactation (5) (Table 1).

### TABLE 1

Association of 16-carbon and 18-carbon trans fatty acids with AA and DHA

<table>
<thead>
<tr>
<th>Erythrocyte membrane phosphatidylcholine lipids in pregnant women¹</th>
<th>Spanish</th>
<th>Hungarian</th>
<th>Umbilical artery</th>
<th>Umbilical vein</th>
<th>Sixth week</th>
<th>Sixth month</th>
</tr>
</thead>
<tbody>
<tr>
<td>AA</td>
<td>16:1t</td>
<td>+0.05</td>
<td>+0.11</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>18:1r</td>
<td>−0.29***</td>
<td>−0.45**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DHA</td>
<td>16:1t</td>
<td>+0.18</td>
<td>+0.11</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>18:1r</td>
<td>−0.43***</td>
<td>−0.34*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Umbilical cord artery and vein wall lipids²</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AA</td>
<td>16:1t</td>
<td>+0.17**</td>
<td>−0.12</td>
<td></td>
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</tr>
<tr>
<td></td>
<td>18:1r</td>
<td>−0.26**</td>
<td>−0.32**</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>DHA</td>
<td>16:1t</td>
<td>+0.23**</td>
<td>+0.15**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>18:1r</td>
<td>−0.21**</td>
<td>−0.15**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Human milk lipids³⁴</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AA</td>
<td>16:1t</td>
<td>+0.14***</td>
<td>+0.08</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>18:1r</td>
<td>−0.60***</td>
<td>−0.43***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DHA</td>
<td>16:1t</td>
<td>+0.07</td>
<td>+0.07</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>18:1r</td>
<td>−0.51***</td>
<td>−0.04</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

¹ Data are Spearman correlation coefficients. *······Indicates significant correlations: *P < 0.05, **P < 0.01, ***P < 0.001. AA, arachidonic acid.
² Spanish (n = 120) and Hungarian (n = 43) women were investigated at the 20th week of gestation (2).
³ Healthy, full-term Dutch infants (n = 308) were included in the study (3).
⁴ German women (n = 769) were investigated at the sixth week of lactation (4).
⁵ German women (n = 462) were investigated at the sixth month of lactation (5).
In 120 Spanish and 43 Hungarian pregnant women who were investigated at the 20th week of gestation, erythrocyte membrane phosphatidylcholine arachidonic acid (20:4n–6; AA) and DHA (22:6n–3) significantly inversely correlated to 18:1t, but not to 16:1t (Table 1) (2). In cord vessel wall lipids in 308 Dutch infants (3), inverse correlations were seen between 18:1t and AA and DHA in both artery and vein wall lipids (Table 1). In contrast, not only was a lack of inverse correlations seen but significant positive correlations were observed between 16:1t and AA and DHA (Table 1). Furthermore, 18:1t, but not 16:1t, significantly positively correlated to Mead acid (20:3n–9), which is a generally accepted indicator of essential fatty acid deficiency (3). In German mothers donating milk samples both at the sixth week (n = 769) and sixth month (n = 462) of lactation (4, 5), the contribution of 18:1t was significantly higher at the sixth week (0.87%) than at the sixth month (0.60%). At the sixth week of lactation, both AA and DHA correlated significantly inversely to 18:1t, but not to 16:1t (Table 1). By the sixth month of lactation, ie, by the time when 18:1t significantly decreased, the negative correlation between TFA and DHA disappeared, whereas 18:1t, but not C16:1t, still correlated significantly to AA (Table 1).

LC-PUFAs may play a role in pregnancy outcomes (6) and in early human neurodevelopment (7). Although supplementation trials failed to provide conclusive evidence on the effect of enhancing the LC-PUFA supply in infancy (8), the impact of LC-PUFA status on perinatal growth and neurobehavioral development is still an important issue (9). The positive association of LC-PUFAs with fetal growth and the negative association of 18:1t with LC-PUFAs may be a possible factor in the lack of correlation between 18:1t and fetal growth in the study of Cohen et al (1).

The potentially untoward role of perinatal TFA exposure on infantile development has been recognized for a relatively long time (10). The novel data presented by Cohen et al (1) calls attention to the role of 16:1t (and one isomer of trans octadecadienoic acid), but not 18:1t, in modifying fetal growth. With a review of our data that were obtained in investigations on various biological samples from different populations of healthy pregnant and lactating women as well as of healthy newborn infants, I would like to emphasize that perinatal 18:1t exposure might also deserve attention.

The author had no conflicts of interest in connection with any part of the present letter.

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REFERENCES

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Reply to T Decsi

Dear Sir:

We thank Decsi for his interest in our recent article and for summarizing his pertinent findings. He questioned why the relation between 18:1t and fetal growth was attenuated after adjustment for confounders, whereas the association of 16:1n(–7t) with fetal growth remained the same. One possible reason is the different food sources of the trans fats. Whereas 18:1t is produced by the industrial hydrogenation of vegetable oils and is therefore predominantly from fried foods and baked goods, 16:1n(–7t) occurs naturally in ruminant sources. Therefore, pregnant women who consume high amounts of 18:1t may differ in several respects from those consuming high amounts of 16:1n(–7t), and adjustment for these factors could differentially influence the results for 18:1t and 16:1t. In a study in the Netherlands, van Eijsden et al (1) found similar results: after adjustment for confounders, the association of the predominant trans-isomer of 18:1, elaidic acid (18:1n–9t, fifth compared with third quintile) with birth weight for gestational age was attenuated from −51.9 ± 24.6 g to −14.2 ± 20.9 g (1). Nevertheless, because the number of children born small for gestational age was relatively small in our cohort (n = 67), and confounding factors could act differently in different populations, we agree with Decsi that future studies should also continue to examine the impact of trans fats, including 18:1t, on fetal development.

Decsi also states that the lack of correlation between 18:1t and fetal growth may be due to the negative association between 18:1t and long-chain PUFAs, which in some studies are positively associated with fetal growth. However, in Project Viva, higher long-chain PUFA intake in pregnancy was associated with lower fetal growth (2).

None of the authors had a conflict of interest.

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Eric B Rimm