Nutritional implications of trans fatty acids during perinatal period, in French pregnant women

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Résumé : Certaines études ont mis en évidence le passage des acides gras trans (AGT) à travers le placenta et suggéré leur interférence avec le métabolisme des acides gras essentiels (AGE). Les AGT pourraient donc affecter le développement intra-utérin du fœtus, plus ou moins selon le niveau de consommation en AGT de la mère. Dans ce contexte, l'objectif de cette étude a donc été d'apprécier, chez une population de femmes enceintes françaises, l'éventuel impact de leur consommation en AGT, évaluée par les teneurs en AGT du tissu adipeux, sur certains paramètres du nouveau-né à terme : 1) Composition en acides gras du cordon ombilical (lipides du plasma et phospholipides pariétaux) ; 2) poids à la naissance et périmètre crânien. Les compositions en acides gras des lipides plasmatiques de la mère et du nouveau-né, ainsi que celles des phospholipides du cordon ombilical ont été déterminées par chromatographie en phase gazeuse, couplée à une chromatographie sur couche mince pour les phospholipides. Le passage transplacentaire des AGT est confirmé. Cependant, le pourcentage des acides gras trans (AGT) dans les lipides du plasma maternel (0,9 % des acides gras totaux) est significativement plus élevé (p = 0,001) que celui observé dans les lipides du plasma ombilical (0,6 %). De plus, le profil en isomères trans des lipides du plasma ombilical différent de celui du plasma maternel, notamment au niveau des isomères trans de l'acide linoléique (18:2 9c12c). Les teneurs en isomères 9c, 13t + 9t, 12t et 9t,12c sont respectivement 2 et 3 fois supérieures (p < 0,001) dans les lipides du plasma ombilical comparées à celles du plasma maternel. Au niveau du cordon ombilical, les isomères trans observés dans le plasma ont tous été retrouvés, à l'exception de l'isomère 16:1 trans, dans les phospholipides (PL) de la paroi et des vaisseaux (artères et veine). Cependant, le taux du mélange d'isomères 9c13t + 9t12t est significativement plus élevé (p < 0,001) dans les PL des artères que dans ceux de la veine. Dans les PL artériels, le pourcentage de ces isomères trans est négativement corrélé (r = - 0,703, p = 0,003) à celui de l'acide arachidonique. Toutefois, pour la population française, il n'apparaît aucune corrélation entre le poids ou le périmètre crânien du nouveau-né et les teneurs en AGT dans les lipides du tissu adipeux ou du plasma de la mère.

Mots-clés : acides gras trans, acides gras polyinsaturés à longue chaîne, nutrition fœtale, tissus du cordon ombilical.

Summary : Some studies have demonstrated the transfer of trans fatty acids (TFA) across the human placenta. It was suggested that TFA might disturb the metabolism of essential fatty acids (EFA) in fetus and consequently might affect intrauterine human growth more or less according to the TFA intake level of the mother. In this context, the objective of this study was to assess, for French pregnant women, possible impact of their TFA intake on parameters of their term infants: 1/ TFA composition of the umbilical cord (plasma lipids and parietal phospholipids), 2/ birth weight and head circumference. The TFA composition of maternal and umbilical plasma lipids, and parietal phospholipids of umbilical cord were determined by gas chromatography, associated with a thin-layer chromatography for the phospholipids. Because TFA content of adipose tissue is a reliable biochemical indicator of the usual TFA intake level, maternal adipose tissue was also analyzed. Trans fatty acid (TFA) percentage was significantly higher ($p = 0.001$) in maternal (0.9% of total fatty acids) than in umbilical plasma total lipids (0.6%) thus confirming their placental passage. Moreover, trans isomer pattern of cord plasma lipids was different from the maternal's one, especially regarding trans isomers of linoleic acid (9c,12c-18:2). Percentage values of 9c,13t + 9t,12t and 9t,12c isomers were respectively, 2 and 3 times higher ($p < 0.001$) in umbilical than in maternal plasma (Except trans 16:1 acids). All trans isomers observed in umbilical plasma lipids were detected in both parietal and vessel (vein and arteries) phospholipids of umbilical cord. Nevertheless, 9c13t + 9t12t isomer mix level was significantly higher ($p < 0.001$) in artery than in vein TPL. Moreover, in artery TPL, percentage value of 9c13t+9t12t isomer mix was inversely correlated ($r = - 0.703$, $p = 0.003$) with arachidonic acid content. Nevertheless, for this French population, there was no relation between either weight or head circumference of newborn and the TFA levels in both adipose tissue and plasma lipids of their mother.

ARTICLE

Occurrence of *trans* fatty acids (TFA) in human tissues depends on their availability in the diet, because humans do not synthesize these fatty acid isomers. It is now well known that *trans* fatty acids are provided by three different dietary sources: the ruminant fats (milk and dairy products, beef, mutton, tallow) [1], the partially hydrogenated vegetable oils (PHVO) (some margarines, shortenings, bakeries and chips) [2] and to a lesser extent, the refined vegetable oils [3].

Several animal studies, *in vivo* [4-6] and *in vitro* experiments in animal tissues [7-10] as well as human fibroblast studies [11] have demonstrated that TFAs impair the microsomal desaturation and chain elongation of essential fatty acids linoleic acid (LA) and alpha-linolenic acid (LNA) to long chain polyunsaturated fatty acids (LC-PUFA). Among all *trans* isomers, 9*t*12*t*-18:2 isomer exerts the strongest impairment of delta6 desaturase activity [4, 12-14]. In adults, inhibitory effect of TFA does not cause serious undesirable health hazards when dietary LA is sufficient [15, 16]. However, the fetus is very sensitive to factors that can interfere with the synthesis of LC-PUFA, because of an extensive need of arachidonic and docosahexaenoic acids for the development of neurological structures of the brain and retina [17-19] and for the synthesis of eicosanoids [20]. In addition, the biosynthetic activity of LC-PUFA is low in fetus [21].

As reported, the level of plasma *trans* octadecenoic acid was inversely related to levels of LC-PUFA in premature infants [22]. The plasma level of TFA and birth weight were also inversely related. In human fetal tissue collected after abortions at a gestational age varying from 5 to 15 weeks and umbilical venous walls from full-term neonates, there was an inverse relation between *trans* 18:1 isomer and the n-6 LC PUFA [23]. Weight and head circumference of full-term neonates were also inversely correlated with *trans* 18:1 acid in umbilical arterial vessel walls.

In this context, it has been suggested that exposure to high levels of TFA during pregnancy may impair the growth of the fetus.

The main objective of this study was to examine the incorporation of TFA into the plasma and red blood cell (RBC) lipids of the umbilical cord blood in relation with TFA composition of the maternal plasma, in order to observe a possible discriminating placental transport of the different *trans* isomers.

In the same way, the TFA profile of maternal plasma lipids was compared with that of the cord wall, as well as cord vein and artery phospholipids, in order to show a possible selective incorporation of the different *trans* isomers into lipids of cord tissues.

Material and methods

Subject and sample collection

The study protocol was approved by the local ethical review committee and informed consent was obtained from 97 participating pregnant women (age: 37 ± 10 years) recruited in Aquitaine between 1997 and 1999. The health state of these mothers needed a childbirth (38-42 postmenstrual weeks) by caesarean surgery.

During caesarean surgery, maternal peripheral venous blood and subcutaneous adipose tissue were removed. Cord vein blood was drawn by venipuncture from the placental portion of the umbilical cord, immediately after its clamping. Clinical data concerning the mother (age, tabbaco consumption...) and the term-born infant (height, weight, head perimeter) were registered by doctor, in a book. RBC were separated from plasma by centrifugation and umbilical cord samples were immediately rinsed with an isotonic saline solution (0.9% NaCl) and then frozen and stored at - 20°C until analyzed.

Analytical procedure

Thawed samples of plasma, adipose tissue and umbilical tissues were ground in a mixture of chloroform/methanol (2:1, by vol). Total lipids were extracted from samples according to the Folch method [24]. Solvents were evaporated to dryness under a stream of nitrogen. Lipids were taken up in an appropriate volume of chloroform/methanol (2:1, by vol) and stored in glass tubes at - 20°C under nitrogen. Plasma triglycerides (TG), cholesterol esters (CE) and total phospholipids (TPL) were separated by thin layer chromatography using hexane/diethyl ether/acetic acid (90:10:1, by vol).

Fatty acids methyl esters (FAME) of total lipids and fractionated lipids were prepared with 14% boron trifluoride in methanol according to Morrison and Smith [25] and stored in hexane, at - 20° C under nitrogen.

Analyses of total FAME were carried out on a gas chromatograph (GC) (Carlo Erba 5160, Milano, Italy) equipped with a flame-ionization detector and a split injector. A fused-silica capillary column (BPX 70, 60 m x 0.25 mm i.d., 0.25 mum film; SGE, France) was used with H_2 as a carrier gas (inlet pressure: 90 kPa). The split ratio was 1/70. The column temperature was programmed from 150 to 200°C at 1.5°C/min, then to 230°C at 2.5° C/min and held at 230°C until completion of the analysis (30 min). The injection port and the detector were maintained at 250°C. The gas chromatographic peaks were integrated using a SP 4400 integrator (Spectra Physics, San Jose, CA).

All the results are expressed as weight percentages of fatty acids using the factor (F't) described by Wolff *et al.* [26]. This factor is linked to the theoretical response factor (Ft) by: F't = Ft x (fatty acid molecular weight/FAME molecular weight).

Results and discussion

***Comparison of TFA composition of mother and fetus plasma* (**Figure 1**)**

Trans fatty acid (TFA) percentage was significantly higher (p = 0.001) in maternal (0.9% of total fatty acids) than in umbilical plasma total lipids (0.6%).

This result is different from that found in a German study describing occurrence of TFA in cord plasma [22], at percentage level similar to that in the mothers. However, in that study, TFA contents of umbilical and maternal plasma total lipids were two times higher than values observed here, which was probably due to a higher intake of TFA in German women than in French, at the time of these studies.

As shown in the *Figure 1*, *trans* isomer pattern in cord plasma lipids was different from the maternal's one. *Trans* 18:1 acid level in maternal plasma (0.64% of total fatty acids) exceeded significantly (p < 0.001) that found in umbilical plasma (0.24%), while *trans* 18:2 acid percentage was significantly higher (p < 0.001) in umbilical (0.30%) than in maternal plasma (0.16%). More precisely, 9*t*12*c*-18:2 isomer and 9*c*13*t* + 9*t*12*t*-18:2 isomer mix amounts were significantly higher (p < 0.001) in umbilical (respectively, 0.15 and 0.08% of total fatty acids) than in maternal plasma (respectively, 0.05%). Whereas no difference was observed between these two compartments with respect to 9*c*12*t*-18:2 isomer level (respectively, 0.06% of total fatty acids in maternal plasma and 0.07% in umbilical plasma).

The occurrence of TFA in cord plasma confirms the transplacental transport of the different *trans* isomers. Nevertheless, the prevalence of *trans* 18:2 isomers and more precisely of 9*t*12*c*-18:2 isomer in umbilical plasma may be explained either by a preferential transfer of this isomer across the placenta, as a consequence of sequestration process, or by an accumulation in cord plasma linked to bad metabolization of this isomer by the fetus compared with the other *trans* 18:2 isomers.

The placental enzyme behaviour towards *trans* 18:2 isomers was examined through the incorporation level of these *trans* diene isomers into the lipid classes of cord plasma.

TFA incorporation into different lipid classes of umbilical plasma (Figure 2**)**

Trans 18:2 isomers occurred in the three main umbilical plasma lipid classes, but the relative percentage values of these *trans* diene isomers were significantly lower (p < 0.001) in total phospholipids (TPL) (0.14% of total fatty acids) than in triglycerides (TG) (0.41%) or in cholesterol esters (CE) (0.41%).

Moreover, these three classes of plasma lipids stanted out by differences in their proportions of various *trans* 18:2 isomers. TPL and CE were characterized by 9*t*12*c* isomer prevalence (respectively, 47 and 48% of total *trans* 18:2 acids), followed by the mix 9*c*13*t*+9*t*12*t* and 9*c*12*t* (respectively, 29 and 24%) in TPL and by 9*c*12*t* and the mix 9*c*13*t*+9*t*12*t* (respectively, 32 and 20%) in EC. Whereas in TG, the three *trans* 18:2 isomers were present in equivalent proportions (31 to 38% of total *trans* 18:2 acids). This finding may be explained by the metabolism of cholesterol. Fatty acid pattern of CE illustrates that of sn-2 position of phosphatidylcholine (PC), since CE are generated by activity of lecithin-cholesterol-acyl-transferase (LCAT) which transfers fatty acids from PC sn-2 position to cholesterol [27]. It is known [28] that 100% of 9*t*12*c* and 77% of 9*c*12*t* isomers are incorporated into the sn-2 position of maternal PC, whereas 75% of 9*c*13*t*+9*t*12*t* isomers mix are present in sn-1 of PC. Consequently, if it is also true for the cord plasma PC, then it could explain the preferential incorporation of 9*t*12*c* and secondly 9*c*12*t* into CE.

Incorporation of the maternal TFA isomers into phospholipids of umbilical blood and red blood cells (RBC) **(**Figures 3 and 4**)**

As shown in *Figure 3*, TFA percentage was significantly lower (p = 0.003) in umbilical RBC (0.25% of total fatty acids) than in umbilical plasma TPL (0.42%). Nevertheless, TFA patterns of these two compartments were similar. *Trans* 18:1 isomers contributed to the major portion to total TFA (55% of total TFA) in both RBC and plasma TPL. However, *trans* 18:2 acid level was significantly lower (p = 0.011) in RBC (0.10% of total fatty acids) than in plasma TPL (0.14%). Moreover, *trans* 18:2 acid profiles *(Figure 4)* were different in these two compartments. Although the three *trans* 18:2 isomer proportions were equivalent (37 to 40% of total *trans* 18:2 acids) in TPL of RBC, 9*t*12*c* isomer predominated (47%) over 9*c*13*t* + 9*t*12*t* isomer mix (29%) and 9*c*12*t* isomer (24%) in plasma TPL.

Maternal TFA incorporation into phospholipids of cord and vessel walls **(**Figure 5**)**

After having investigated TFA incorporation into both plasma and RBC lipids of cord vein blood, our attention was then paid to the TFA incorporation into TPL of umbilical cord parietal tissue and umbilical vessels.

Except *trans* 16:1 acids, all *trans* isomers already seen in umbilical plasma were detected in both parietal and vessel TPL of umbilical cord. TFA content, all isomers confounded, of cord wall TPL (0.31% of total fatty acids) didn't differ significantly from that found in cord vein (0.28%) or artery TPL (0.35%). Nevertheless, proportions of *trans* 18:1 and 18:2 isomers were similar (respectively, 48 and 52% of total fatty acids) in cord wall and vein TPL, whereas in artery TPL, *trans* 18:2 isomers predominated (68% of total TFA) significantly (p = 0.001) over *trans* 18:1 isomers (32% of total TFA).

Furthermore, these three compartments of umbilical cord (parietal tissue, vein and artery) were characterized by prevalence of $9c13t + 9t12t$ isomers compared with the other *trans* 18:2 isomers. Nevertheless, the isomer mix level was significantly higher (p < 0.001) in artery TPL (0.17% of total fatty acids) than in vein TPL (0.09%), itself significantly higher (p = 0.005) than in cord wall TPL (0.06%).

Moreover, in artery TPL, percentage value of $9c13t + 9t12t$ isomer mix was inversely correlated (r = -0.703, p = 0.003) with arachidonic acid (AA) content, but not with that of linoleic acid (LA), precursor of AA. This result is in agreement with the inverse relation observed between TFA and LC-PUFA in umbilical plasma lipids of both healthy children [29] and preterm infant [22]. This inverse relation was also verified between *trans* 18:1 isomer and the n-6 LC PUFA in human fetal tissue collected after abortions (gestational age 5-15 weeks) [23].

If we admit that fatty acid pattern of umbilical artery TPL reflects that of fetal tissue TPL, then these results could demonstrate a potential impairment by $9c13t + 9t12t$ isomer mix of LA metabolism, or preferential uptake of maternal plasma AA by the placenta. Major part of LC-PUFA found in fetal tissues comes from maternal blood, because placental delta6 desaturase activity is too poor to provide sufficiently LC-PUFA for fetus development [30]. Nevertheless, concerning AA, selective transplacental transport is supposed to be less important than fetal synthesis [31], thus interference of $9c13t + 9t12t$ isomer mix with delta6 desaturase activity could be the best explanation to our results. Insofar as AA is of great physiological importance during fetal development as essential membrane component of neural tissues and as precursor for eicosanoid synthesis, it could be assumed that accumulation of these *trans* dienes might have side effects on fetus development. However, these results must be considered with a lot of caution, since it concerns the mix of different *trans* 18:2 isomers.

Some studies [22, 23] have suggested that exposure to high levels of TFA during pregnancy may affect intrauterine human growth, in particular birth weight and head circumference.

So, after examination of TFA incorporation into umbilical plasma lipids and TPL of cord and vessel walls, we have devoted us to assess, for these French pregnant women, potential impact of their TFA intake during pregnancy on either weight or head circumference of newborn.

***Relation between newborn anthropometric parameters and TFA levels in maternal and umbilical plasma* (**Figures 6 *to* 9**)**

Because TFA content of adipose tissue is a reliable biochemical indicator of the usual TFA intake level [32, 33], we studied newborn anthropometric parameters in relation to TFA level of maternal adipose tissue lipids *(Figures 6 and 7)*. Based on dietary questionnaire, we found that average TFA consumption of this pregnant women population was 3.1 ± 1.2g/d/pers., ranging from 0.9 to 6.5g/d/pers. (or 0.5 to 2.5% of total energy intake). No correlation was observed between TFA level of maternal adipose tissue and either newborn weight *(Figure 6)* or head circumference *(Figure 7)*. Correlation absence persisted even after having applied corrections which took account of confounding factors such as mother's age, BMI and possible tabacco consumption.

This first result shows that TFA intake level of these French pregnant women seems to be sufficiently low to induce no growth failure.

However, we have verified in the second time if TFA amount, transferred across the placenta from maternal plasma lipids, could be put in relation to previous antropometric parameters *(Figures 8 and 9)*. As for the adipose tissue, we noticed that TFA level of maternal plasma lipids was related to neither newborn weight *(Figure 8)* nor head circumference *(Figure 9)*, even after multifactorial corrections.

Our results do not agree with those reported by Koletzko *et al.* [22] who demonstrated negative correlations between birth weight of premature infant and percentage of either total TFA in umbilical TPL or *trans* 18:1 acid percentage in CE. Similarly, Van Houvelingen *et al.* [23] have also observed inverse relation between *trans* 18:1 acid in umbilical arterial vessel walls and weight and head circumference of full-term neonates.

This discrepancy might be explained by sensitivity to TFA in full-term lower than in preterm infant whose LC-PUFA stores are extremely more limited compared to term infant [29], and by TFA intake of mothers which was higher in Germany and Netherlands than in Aquitaine [34].

CONCLUSION

In summary, we observed differences in TFA proportions between maternal and cord plasma total lipids. These data could demonstrate either potential discriminating placental transport of different TFA isomers or metabolism selectivity of different TFA isomers by the fetus. *Trans* isomers were incorporated not only into lipids of cord blood (plasma and RBC) afferent to fetus but also into TPL of parietal tissue and vessels of umbilical cord. Moreover, inverse correlation found between 9*c*13*t* + 9*t*12*t* isomer mix and AA levels in artery TPL suggested that these *trans* 18:2 isomers might enhance LC-PUFA deficiency of fetus. However, this hypothese must be supported by further investigations since it concerns mix of different *trans* 18:2 isomers.

Finally, TFA intake level of these French pregnant women, assessed to 3.1 gram per day, seems to be sufficiently low not to affect negatively weight and head circumference of newborn.

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Illustrations
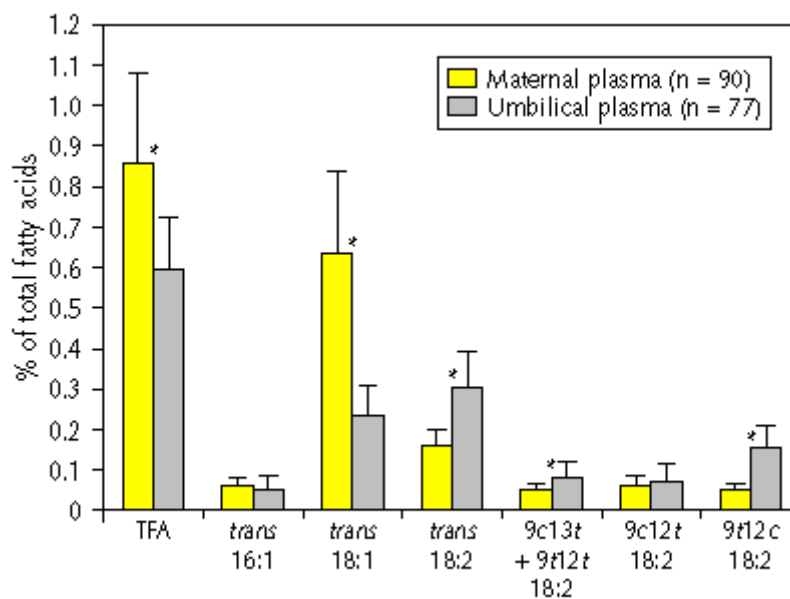
Figure 1. Trans *fatty acid (TFA) composition (% weight) of maternal and umbilical plasma total lipids (* p < 0.001).*
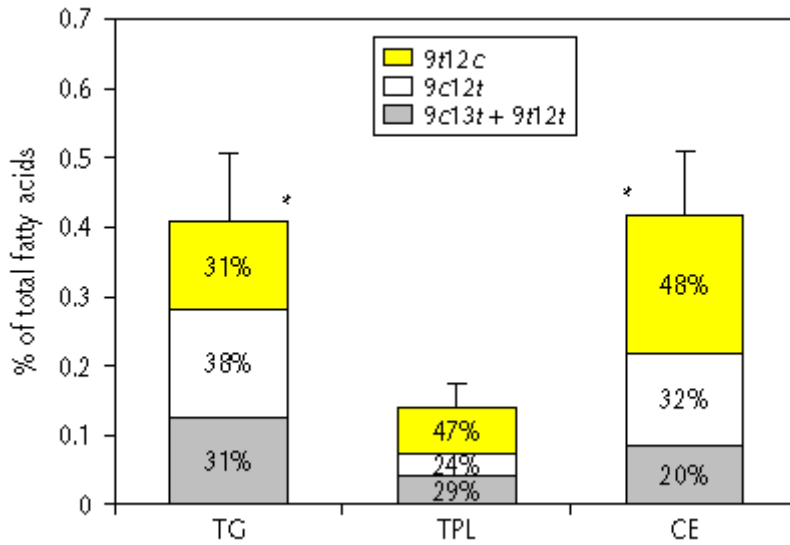
Figure 2. *Proportion of the different* trans *18:2 isomers in triglycerides (TG) (n = 32), total phospholipids (TPL) (n = 62) and cholesterol esters (CE) (n = 40) of umbilical plasma (* p < 0.001).*
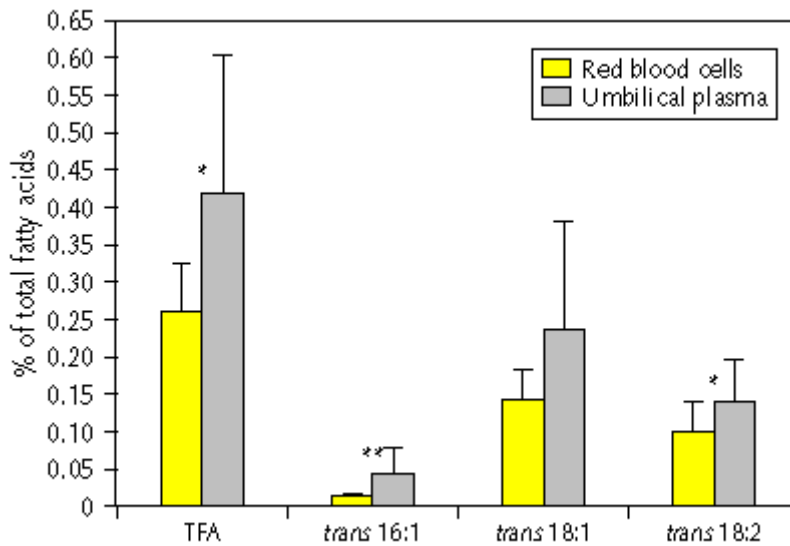
Figure 3. Trans *fatty acid (TFA) composition (% weight) of umbilical red blood cell (n = 11) and plasma total phospholipids (n = 62) (* p < 0.01, ** p < 0.001).*
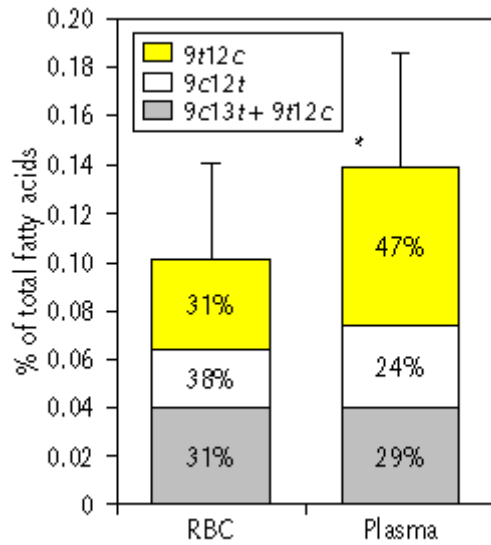
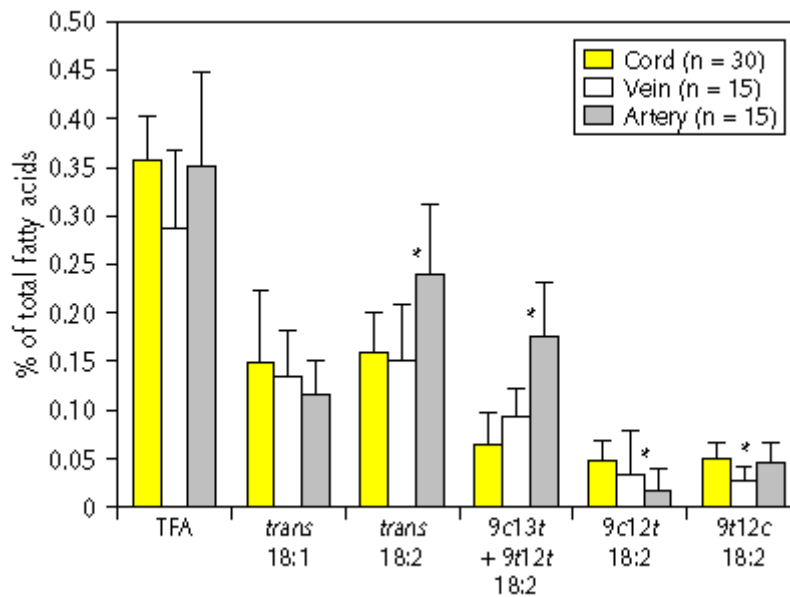Figure 4. *Trans 18:2 isomer pattern of umbilical red blood cell (RBC) (n = 11) and plasma (n = 62) total phospholipids (* p < 0.01).*

Figure 5. *Trans fatty acid (TFA) composition (% weight) of cord wall, vein and artery total phospholipids (* p < 0.001).*
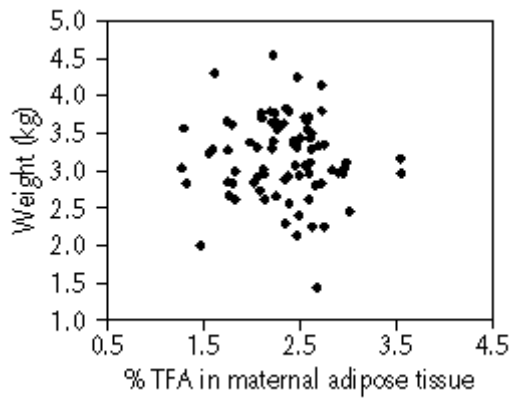
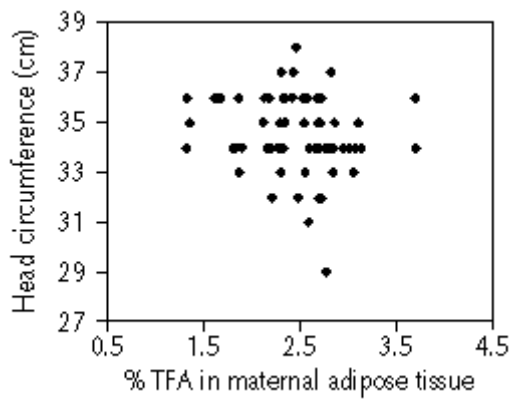Figure 6. *Correlation between newborn weight (kg) and TFA percentage in maternal adipose tissue (n = 81).*

Figure 7. *Correlation between newborn head circumference (cm) and TFA percentage in maternal adipose tissue (n = 68).*
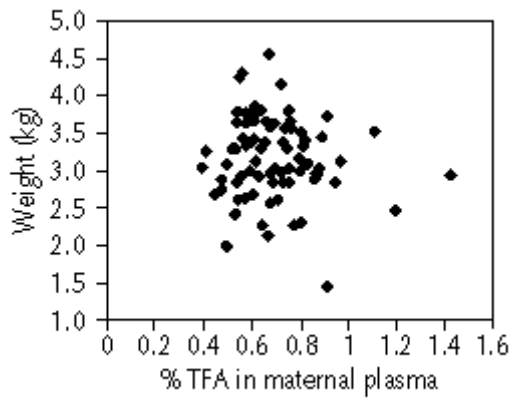
Figure 8. *Correlation between newborn weight (kg) and TFA percentage in maternal plasma (n = 81).*
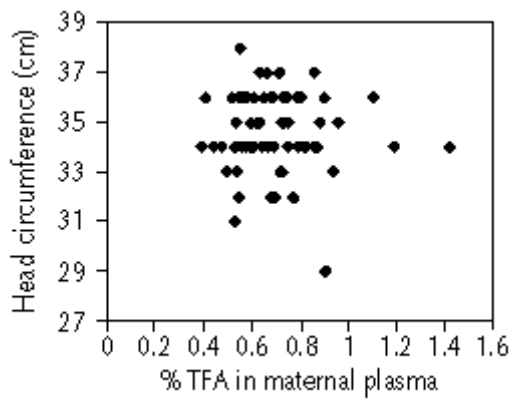

Figure 9. *Correlation between newborn head circumference (cm) and TFA percentage in maternal plasma (n = 68).*