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Markers of Oxidative Stress in Diabetic Mothers and Their Infants During Delivery

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Summary

Oxidative stress is probably a pathophysiological process leading to disadvantageous outcomes in diabetic pregnancies. We aimed to map a complex of potential markers of oxidative stress in this condition. Diabetic mothers had significantly higher concentrations of thiobarbituric acid reactive substances in the plasma [TBARS] both before ($p<0.0001$) and after ($p<0.001$) delivery and also their newborns showed higher values of TBARS ($p<0.0001$) in comparison with the control group. Diabetic mothers also showed lower concentrations of reduced glutathione in erythrocytes [GSH] both before ($p<0.05$) and after ($p<0.01$) delivery and their infants also had lower levels of GSH ($p<0.0001$). We found a lower total antioxidative capacity of plasma [AOC] before delivery ($p<0.05$) in the diabetic group in comparison with the control group. Newborns of diabetic mothers had higher plasmatic concentrations of apolipoproteine B [apo B] ($p<0.05$), higher erythrocyte glutathione peroxidase [GPx] activity ($p<0.05$) and lower pH ($p<0.001$) in the umbilical cord blood, when compared with infants of control non-diabetic mothers. We conclude that pregestational and gestational diabetes mellitus represent increased oxidative stress for both mother and her infant. TBARS in plasma are a valuable marker of oxidative stress in this condition. Disruption of glutathione peroxidase/glutathione pattern can be involved in pathophysiology of enhanced oxidative stress in diabetic pregnancies.

Key words

Diabetes mellitus • Gestational diabetes mellitus • Delivery • Oxidative stress • Gender

Introduction

Diabetes in pregnancy increases perinatal morbidity and mortality of both mother and her newborn. The prevalence of pregestational diabetes (type 1 or type 2) has been estimated at 0.2-0.5 % of all pregnancies in the USA (Feig and Palda 2002) and 0.2-0.4 % in European countries (Linn and Bretzel 1997). About 65 % of these cases can be attributed to type 2 diabetes. The prevalence of gestational diabetes mellitus (GDM) in Europe varies between 0.15 and 4 % of all pregnancies (Linn and Bretzel 1997). GDM is a potent risk factor for

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the development of permanent diabetes later in life (Hoffman *et al.* 1998).

Although hyperglycemia is clearly recognized as the primary culprit in the pathogenesis of diabetic complications, even maximum glycemic control is associated with the development of complications. There is growing evidence that oxidative stress can play an important role in the pathogenesis of DM, GDM (Matteucci and Giampietro 2000) and in the development of maternal and fetal complications of diabetic pregnancies (Viana *et al.* 1996, Baynes and Thrope 1999, Kinalski *et al.* 2000, Stitt 2001). Reactive oxygen and nitrogen species may cause chemical alterations of biomolecules (lipids, proteins, DNA, carbohydrates) and the redox state of the whole organism is maintained by a complex of mechanisms. Scavenging enzyme activities (Cu-Zn superoxide dismutase, SOD; glutathione peroxidase, GPx) reflect antioxidant defence status. Glutathione is the most abundant intracellular antioxidant. Malondialdehyde (MDA) is an end-product of lipid peroxidation. Among the different analytical methods established, the reaction with thiobarbituric acid (thiobarbituric acid reactive substances, TBARS) is the most widely used (Jentzsch *et al.* 1996). Although the reaction is not specific for MDA only, it was shown that the majority of TBARS originate in lipid peroxidation (Lefevre *et al.* 1998). Measurement of TBARS can be useful in determination of fetal hypoxia during labour (Wang *et al.* 1996).

Several studies found associations among diabetes in pregnancy and different markers of oxidative stress (Carone *et al.* 1993, Kinalski *et al.* 2001) and confirmed that supplementation with some antioxidants (vitamins E and C) could be beneficial in the treatment of diabetes (Jain *et al.* 2000) and in the prevention of teratogenic effect of diabetes (Cederberg *et al.* 2001). Management of oxidative stress is considered, along with tight glycemic control, to be beneficial both preconceptionally and during pregnancy. However, there is no consensus on the pathophysiological events underlying oxidative stress in diabetic patients, especially in pregnancy.

In the present study, we aimed to map a pattern of oxidative stress and cardiovascular risk markers in diabetic mothers and their newborns during labor. Furthermore, we also aimed to elucidate the influence of the manner of delivery (Caesarean section) and gender of the newborns on selected markers of oxidative stress in newborns and their mothers.

Methods

We enrolled 18 diabetic mothers and infants (group D) and 18 healthy pregnant age-matched mothers and infant pairs (group C). The group D consisted of 12 mothers with type 1 diabetes mellitus (diagnosed before the pregnancy, DM subgroup) and 6 mothers with gestational diabetes mellitus (diagnosed on the basis of oral glucose tolerance test results during pregnancy, GDM subgroup). All infants from the control group were delivered vaginally at term and were appropriate for the gestational age. Twelve infants from the diabetic group were delivered by Caesarean section (SC diabetic subgroup) at term and the remainder delivered vaginally (V diabetic subgroup) in term. The additional control group included 11 healthy pregnant women + infant pairs with a negative diabetic history and their normal pregnancies were terminated by Caesarean section (SC control group). Further details are summarized in Table 1.

Table 1. Basic characteristics of control (group C) and diabetic (group D) groups.

	Control group (n=18)	**Diabetic group (n=18)**
Mothers´ age (years)	27 (22-38)	27 (22-43)
Gestational age (months)	40.5 (37-42)	38 (36-41)
Newborns´ weight (g)	3600 (2750-4950)	3550 (2400-4900)
Newborns´ length (cm)	50.0 (45-54)	49.5 (45-52)
Apgar 1	10 (9-10)	10 (5-10)
Apgar 5	10 (9-10)	10 (9-10)
Newborn males (count)	8	7

Data are expressed as median (range).

Heparinized venous blood samples were withdrawn from mothers before delivery (uterine outlet < 3 cm) and after delivery of the placenta. Heparinized blood from newborns was obtained immediately following delivery from the umbilical cord vein. Plasma and erythrocyte mass were obtained by centrifugation (2400 x *g*, 5 min, 4 °C). Butylated hydroxytoluene (BHT) was added to the plasma for TBARS analysis (8 µl BHT to 400 µl plasma; final concentration of BHT in the

reaction solution was 3 mmol/l) prior to freezing. For reduced glutathione determination erythrocyte mass was used, superoxide dismutase activity was determined in erythrocyte lysate (add 3 ml 0.9 % NaCl to 500 µl of blood and centrifuge 660 g/10 min at 4 °C, remove supernatant and repeat 4 times; after final removal of supernatant add 1.5 ml of chilled distilled water, leave for 20 min and freeze) and glutathione peroxidase activity was determined in whole blood. Plasma, erythrocyte mass and erythrocyte lysate were stored at –80 °C until analysis (maximum of 2 months after freezing, stability of all measured analytes was tested) that was performed in batches.

Levels of superoxide dismutase (SOD), glutathione peroxidase (GPx), reduced glutathione (GSH), thiobarbituric acid reactive substances (TBARS) and total antioxidant capacity (AOC) were measured at the Department of Clinical Biochemistry and Hematology of the Medical Faculty in Pilsen, Charles University. To determine these levels (except TBARS) commercial kits from RANDOX (SOD, GPx, AOC; Crumlin, North Ireland) and OXIS (GSH, Portland, USA) were used. The levels of TBARS were measured using the thiobarbituric acid reaction (Jentzsch *et al.* 1996).

The basic parameters of lipid metabolism (serum levels of total cholesterol [TC], HDL-cholesterol [HDL], LDL-cholesterol [LDL] and triglycerides [TG]) were determined using commercial kits from Roche (Mannheim, Germany), apolipoproteins A [apo A] and B [apo B] were determined with Tina Quant kit by Roche Diagnostics (Mannheim, Germany). A Hitachi 712 Analyzer (Roche, Germany) was used for analysis. Glycated hemoglobin (HbA_{1c}) was determined using the HPLC method (Tosoh Corp., Tokyo, Japan) and fructosamine by photometric analysis (Roche, Manheim, Germany).

To determine antibodies against oxidized LDL (oxLDLAb), the oLAb ELISA kit from eliTec (Raabs, Austria) was used. This method uses microtiter wells coated with Cu^{2+}-oxidized LDL where after binding of oxLDLAb from serum, anti-human IgG peroxidase conjugate is used for their detection. The SPECTRA I microplate reader (SLT Labinstruments, Austria) was used to measure this ELISA method.

For the statistical evaluation of normally and log-normally distributed results, the unpaired t-test and paired t-test or ANOVA were used. For non-normally distributed results nonparametric Mann-Whitney U-test or Wilcoxon signed rank test were used. $P<0.05$ value was considered statistically significant, data are expressed (if not stated otherwise) as median (range). All computations were performed by R 1.7.1 (Free Software Foundation, http://www.r-project.org/) and Statview (SAS Institute Inc.Cary, NC, USA) software.

Results

Differences between DM subgroup (n=12) and GDM subgroup (n=6)

No differences were found between values of mothers and newborns from the DM and GDM subgroups, except for significantly higher titers of oxLDLAb in the GDM group before and after delivery and in newborns ($p<0.05$) and significantly lower AOC levels after delivery ($p<0.05$) in the same group (Table 2). Thus, in further statistical evaluation subgroups, DM and GDM were considered (except for oxLDLAb values before and after delivery and in newborns and AOC levels after delivery) as one group (group D). We were not able to demonstrate any differences between DM and GDM in markers of diabetes compensation (HbA_{1c}, fructosamine).

Changes in mothers during delivery (Table 3)

Although there were no statistical differences between the values of all measured parameters before and after delivery in the control group, there was a significant increase in SOD ($p<0.05$) and a decrease in AOC ($p<0.01$) after delivery in the diabetic group. We also found a decrease in AOC ($p<0.05$) (without an increase in SOD or other measured parameters) following delivery in the SC (Caesarean section) control group.

Differences between SC diabetic subgroup (n=12) and V diabetic subgroup (n=6)

Diabetic mothers who underwent SC had higher SOD activities [1312.5 (1167-1642) vs. 1185 (1066-1333); $p<0.05$] and lower AOC [1.16 (0.98-1.56) vs. 1.42 (1.15-1.64); $p<0.05$] after delivery in comparison with diabetic mothers who delivered vaginally. Newborns of diabetic mothers delivered by SC showed lower AOC [1.36 (1.21-1.55) vs. 1.54 (1.46-1.67); $p<0.05$] than those delivered vaginally. In addition, there was a lower concentration of AOC after delivery in the SC control group in comparison with vaginally delivering control mothers [1.26 (1.15-1.44) vs. 1.43 (1.27-1.84); $p<0.01$].

Table 2. Oxidative stress markers (DM and GDM subgroups).

	SOD (U/g Hb)	GPx (U/g Hb)	GSH (mmol/l ery)	AOC (mmol/l)	TBARS (µmol/l)	oxLDLAb (U/l)
Before DM	1206 (1057-1444)	62.4 (48.3-88.1)	1.66 (1.48-2.14)	1.28 (1.15-1.53)	3.1 (2.17-4.4)	809.8 (339.8-1071.1)
Before GDM	1184.5 (1000-1398)	63.25 (49-90.8)	1.75 (1.34-2.14)	1.345 (1.27-1.52)	3.65 (2.4-5.23)	2199.5 (937.5-5150)
After DM	1308 (1103-1622)	61.15 (47-99.8)	1.715 (1.45-2.25)	1.155 (0.98-1.64)	3.09 (1.4-3.7)	685 (316.4-1064.1)
After GDM	1190 (1066-1642)	63 (49.7-75.2)	1.82 (1.37-2.13)	1.415 (1.22-1.49)	3.49 (2.32-4.61)	2231.95 (915-4380)
Infant DM	1273 (1171-1436)	49.3 (37.7-69.5)	1.88 (1.39-2.3)	1.43 (1.29-1.56)	4.6 (3.69-5.2)	449 (180.5-943.4)
Infant GDM	1178 (1066-1688)	48.55 (46.1-55.6)	1.81 (1.62-2.02)	1.5 (1.21-1.67)	4.2 (3.61-4.98)	1395.55 (970-4305)

Data are expressed as median (range). DM = type I diabetes mellitus group; GDM = gestational diabetes mellitus group; before = before delivery; after = after the delivery; infant = immediately after delivery (umbilical cord blood).

Table 3. Oxidative stress markers.

	SOD (U/g Hb)	GPx (U/g Hb)	GSH (mmol/l ery)	AOC (mmol/l)	TBARS (µmol/l)	oxLDLAb (U/l)
Before C	1182.5 (957-1439)	64.25 (39.6-102.6)	1.92 (1.5-2.77)	1.395 (1.29-1.74)	1.965 (1.38-3.45)	410 (98.3-6050)
Before D	1200.5 (1000-1444)	62.4 (48.3-90.8)	**1.67** ↓ (1.34-2.14)	1.315 (1.15-1.53)	**3.105** ↑↑↑ (2.17-5.23)	937.5 (339.8-5150)
After C	1173 (952-1376)	75 (41.4-102.8)	2.04 (1.59-2.57)	1.43 (1.27-1.84)	1.99 (1.52-2.81)	401.5 (115-6500)
After D	1251.5 (1066-1642)	61.15 (47-99.8)	**1.72** ↓↓ (1.37-2.25)	1.21 (0.98-1.64)	**3.29** ↑↑↑ (1.4-4.61)	878.9 (316.4-4380)
Infant C (total)	1226 (1045-1525)	44.9 (34.7-55)	2.305 (1.62-2.85)	1.46 (1.33-1.97)	3.28 (2.45-4.09)	273.5 (7.18-4960)
Infant C (male)	1329 (1129-1382)	44.9 (34.7-54.8)	2.27 (2.15-2.55)	1.48 (1.33-1.87)	3.44 (2.93-4.09)	260 (106-998)
Infant C (female)	1197 (1045-1525)	45.1 (38.6-55)	2.37 (1.62-2.85)	1.46 (1.35-1.97)	3.28 (2.45-3.84)	432 (7.18-4960)
Infant D (total)	1244 (1066-1688)	**49.3** ↑ (37.7-69.5)	**1.87** ↓↓↓ (1.39-2.3)	1.47 (1.21-1.67)	**4.4** ↑↑↑ (3.61-5.2)	696 (180.5-4305)
Infant D (male)	1334 (1171-1688)	46.3 (37.7-50.6)	1.78 (1.62-2.06)	1.46 (1.21-1.55)	4.22 (3.69-4.98)	569.1 (180.5-1805)
Infant D (female)	1213 (1066-1407)	53.1 (43.3-69.5)	1.88 (1.39-2.3)	1.49 (1.29-1.67)	4.4 (3.61-5.2)	943.4 (381-4305)

Data are expressed as median (range). C = control group; D = diabetic group; before = before delivery; after = after the delivery; infant = immediately after delivery (umbilical cord blood). ↑ (↓) - significantly higher (lower) than control group ($p<0.05$), ↑↑ (↓↓) - significantly higher (lower) than control group ($p<0.01$), ↑↑↑ (↓↓↓) - significantly higher (lower) than the control group ($p<0.001$)

Differences between SC diabetic subgroup (n=12) and SC control subgroup (n=11)

Diabetic mothers who delivered by SC showed increased concentrations of TBARS both before [3.06 (2.17-4.4) vs. 1.71 (1.24-2.19); p<0.0001] and after [2.89 (1.4-4.2) vs. 1.74 (1.01-2.71); p<0.01] delivery and their infants also demonstrated higher levels of TBARS [4.25 (3.69-5.2) vs. 3.26 (2.8-4.66); p<0.001]. Infants born to diabetic mothers who underwent SC had lower GSH [1.84 (1.39-2.06) vs. 2.27 (1.59-2.47); p<0.01] and AOC [1.36 (1.21-1.55) vs. 1.46 (1.4-1.62); p<0.05) in comparison with infants of non-diabetic mothers that delivered by SC.

Differences between group D (n=18) and group C (n=18)

Diabetic mothers had significantly higher concentrations of TBARS both before (p<0.0001) and after (p<0.001) delivery and their newborns exhibited higher values of TBARS (p<0.0001) in comparison with the control group (Fig. 1). Diabetic mothers also showed lower concentrations of GSH both before (p<0.05) and after (p<0.01) delivery and their infants also had lower levels of GSH (p<0.0001). We found lower AOC before delivery (p<0.05) in the diabetic group and lower AOC after delivery (p<0.001) but only in the DM subgroup (and there was no significant difference in the GDM subgroup) in comparison with the control group. Newborns of diabetic mothers had higher concentrations of apo B (p<0.05), higher GPx activity (p<0.05) and lower pH (p<0.001) in umbilical cord blood, when compared with infants of control non-diabetic mothers (Tables 1, 3 and 4).

Table 4. Lipoprotein concentrations.

	chol (mmol/l)	HDL (mmol/l)	LDL (mmol/l)	apo A (g/l)	apo B (g/l)
Before C	7.4 (3.9-9.3)	1.53 (0.98-1.80)	4.74 (1.42-6.84)	1.86 (1.51-2.98)	1.53 (0.53-1.87)
Before D	7.0 (5.2-8.8)	1.46 (1.38-1.81)	4.07 (1.84-5.86)	1.79 (1.52-2.27)	1.29 (0.24-2.00)
After C	7.4 (4.1-9.1)	1.67 (1.13-1.80)	4.06 (1.87-6.67)	1.81 (1.62-2.37)	1.41 (0.48-1.86)
After D	6.6 (4.7-9.0)	1.54 (1.29-1.88)	3.86 (2.17-5.79)	1.72 (0.59-2.33)	1.31 (0.85-2.00)
Infant C (total)	1.7 (1.2-2.6)	0.83 (0.52-1.27)	0.80 (0.24-1.53)	0.80 (0.62-0.97)	0.24 (0.06-0.64)
Infant C (male)	1.6 (1.2-2.4)	0.83 (0.52-1.27)	0.61 (0.24-1.22)	0.83 (0.63-0.94)	0.24 (0.06-0.32)
Infant C (female)	1.8 (1.6-2.6)	0.85 (0.55-1.03)	0.9 (0.52-1.53)	0.79 (0.62-0.97)	0.23 (0.08-0.64)
Infant D	2.3 (1.4-3.9)	0.77 (0.73-0.96)	1.15 (0.57-1.43)	0.89 (0.53-1.06)	0.32 ↑ (0.19-0.79)
Infant D (male)	2.4 (1.4-3.7)	0.87 (0.77-0.96)	0.99 (0.74-1.24)	0.92 (0.53-1.06)	0.32 (0.19-0.74)
Infant D (female)	2.2 (1.6-3.9)	0.76 (0.73-0.85)	1.15 (0.57-1.43)	0.83 (0.81-1.03)	0.31 (0.25-0.79)

Data are expressed as median (range). C = control group; D = diabetic group; before = before the delivery; after = after delivery; infant = immediately after delivery (umbilical cord blood). ↑ (↓) - significantly higher (lower) than the control group (p<0.05).

Differences between male and female newborns (Tables 1 and 3)

No differences were found between male and female newborns in the control group. However, significantly lower activities of GPx in male newborns (p<0.05) from the diabetic group was demonstrated when compared with their female counterparts from the same group.

Differences between mother and infant (Tables 3 and 4)

Infants from the control group showed higher concentrations of GSH (p<0.0001) and lower activities of GPx (p<0.0001) in comparison with their mothers. Infants from the D group, on the contrary, had only lower activities of GPx (p<0.001), but no differences were found in GSH levels in comparison with their mothers. Infants from both D and C groups had higher concentrations of TBARS (p<0.0001 for both groups) than their mothers. Infants from group D, though not from group C, showed higher AOC (p<0.01) and lower oxLDLAb (p<0.01), when compared with their mothers. Mothers from both groups had higher levels of all measured parameters of lipid metabolism (TC, HDL, LDL, TG, apo A and apo B; p<0.01 for both groups, all parameters) than their infants.

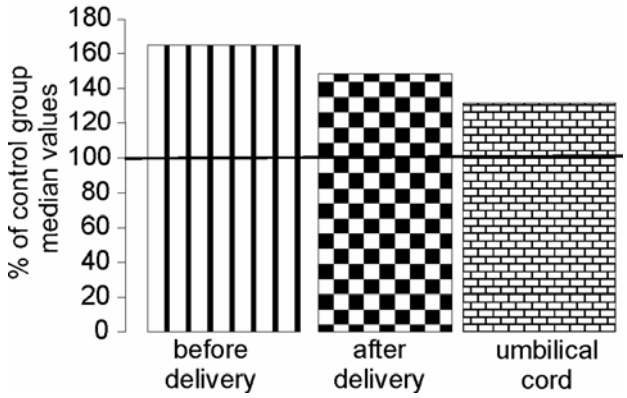

Fig. 1. TBARS concentrations in group D and group C. Values are expressed as percents of median.

Discussion

Management of oxidative stress is a great challenge for both researchers and clinicians. In this study, we describe a complex of potentially clinically valuable markers of oxidative stress in diabetic pregnancies.

We found substantially increased levels of TBARS in diabetic mothers and in their infants (Fig. 1). This is in accordance with other authors (Carone *et al.* 1993, Kamath *et al.* 1998, Kinalski *et al.* 2001, Orhan *et al.* 2003) and implies that TBARS are one of the promising clinical markers of oxidative stress. We have proved (data not shown) that glucose concentrations of up to 45 mmol/l do not interfere with TBARS determination by the method we have used. Diabetic mothers were usually closely compensated (HbA_{1C} levels in the 3rd trimester 6.6[5.5-9.5] %, upper reference limit 6.0 %) and glucose concentrations higher than the concentration mentioned above are improbable. Infants of diabetic mothers have normoglycemia or even severe hypoglycemia immediately after labor so that hyperglycemia cannot be expected. In the present study, diabetic mothers and their infants showed decreased concentrations of GSH in comparison with healthy pregnant mothers and their newborns. This agrees with the concept of oxidative stress in diabetic pregnancies, but it is in disagreement with Kinalski *et al.* (2001) who found significantly higher levels of GSH in umbilical cord blood of infants of mothers suffering with pregestational and gestational diabetes mellitus. Few studies have measured GSH in human (Kharb 2000) and animal (Sakamaki *et al.* 1999, Damasceno *et al.* 2002) diabetic pregnancies, but these agree with our results.

However, the higher activities of GPx in newborns of diabetic mothers remain more controversial. Orhan *et al.* (2003) found higher GPx activities in mothers with diabetes mellitus type 1 before delivery, but no changes in their newborns. The main shortcoming of this study was the very small sample (n=3). We have previously described (Rajdl *et al.* 2002) and confirmed in the present study a physiological pattern of GSH and GPx in mothers during delivery and in their newborns. This pattern is characterized by relatively (in comparison with their infants) higher activities of GPx (thus providing a better chance to decompose lipid hydroperoxides) and lower GSH concentrations (thus a lower intracellular reduction potential, probably caused by GSH consumption by GPx) in mothers. On the other hand, infants of healthy mothers have relatively low activities of GPx (we interpret this as a sign of a lower need of lipid hydroperoxide decomposition) and relatively high concentrations of GSH (relatively lower consumption of GSH by GPx). Low activities of GPx in infants should not be explained as a mark of antioxidant enzymatic system immaturity, as adult activities of GPx are attained in healthy infants between 28 and 30 weeks of pregnancy (Candlish *et al.* 1995). We hypothesize that the burden of lipid hydroperoxides decomposition is shifted towards mothers in physiologic pregnancies, but this mechanism is disrupted in diabetic mothers and it results in relatively (compared to newborns born to healthy mothers) higher activities of GPx and lower GSH levels in newborns born to diabetic mothers. This pattern in infants of diabetic mothers is associated with enhanced lipid peroxidation (elevated TBARS levels).

Interestingly, in this study we found significantly higher GPx activities in female newborns of diabetic mothers in comparison with their male counterparts. This difference (95 % CI: 2.0 to 18.1) is more difficult to interpret. The fact that female newborns have relatively better chance to decompose lipid hydroperoxides with comparable (or even non-significantly higher) intracellular reduction potential (GSH concentration) led us to hypothesize that female children of diabetic mothers have a relatively better defence against oxidative stress. This could be important especially during the perinatal period that is characterized by enhanced oxidative stress (Wang *et al.* 1996, Neefjes *et al.* 1999, Rajdl *et al.* 2002). There are some hints that the male gender can be disadvantageous for very low birth weight infants (Stevenson *et al.* 2000) and even healthy male infants are more prone to lower Apgar score at 5 min and have a higher perinatal morbidity and mortality (Thorngren-

Jerneck and Herbst 2001). Lavoie and Chessex (1997) showed better viability, higher glutathione reductase activity and higher total intracellular glutathione content in female-derived cells (umbilical endothelial cells, cells from tracheal aspirates). Parmigiani *et al.* (2000) concluded that normal female newborns have lower levels of reactive oxygen metabolites.

The effect of the mode of delivery (Caesarean section vs. vaginal labor) does not seem to be conclusive in this study. However, we have proved higher (in comparison with diabetic mothers that delivered vaginally) SOD activities after delivery exclusively in diabetic mothers undergoing SC. This fact indicates that women with diabetes undergoing Caesarean section may have a relative advantage, in comparison with diabetic mothers delivering vaginally, of better decomposition of superoxide radicals and hence a better chance to cope with oxidative stress during the peripartum period. We are not aware of any study determining the influence of caesarean section on parameters of oxidative stress in diabetic women. There are some data about the influence of Caesarean section on oxidative stress markers in umbilical cord blood that indicate no influence of delivery mode in physiological infants (Parmigiani *et al.* 2000), but Caesarean section may be advantageous for preterm infants (Yigit *et al.* 1998). The differences between SC control group and SC diabetic subgroup can be ascribed to differences between D and C groups.

In this study, we confirmed that there is a relative predominance of HDL-cholesterol in physiological neonates (Hardell and Carlson 1978), but there is a non-significant tendency to higher LDL cholesterol and significantly increased apo B in diabetic newborns. These findings support the hypothesis that diabetes in a mother influences the lipid metabolism of her offspring.

Conclusions

Pregestational and gestational diabetes mellitus represent increased oxidative stress for both mother and her infant. TBARS are valuable markers of oxidative stress in this condition. Disruption of glutathione peroxidase/glutathione patterns can be involved in the pathophysiology of enhanced oxidative stress in diabetic pregnancies. Male vs. female newborns of diabetic mothers have lower GPx activities immediately after birth.

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