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The Effect of Low Alcohol Consumption during Pregnancy on the Metabolic Processes of Women and Their Alcohol-Exposed Babies

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Abstract

The aim of this study was to examine the effect of low or very low amounts of alcohol consumption on the LPO-AOD systems of pregnant women and their infants after birth, and the effect of that exposure on infant, growth, health, and development.

Methods and Results: A sample of 201 pregnant women (mother-child dyads) was recruited for the study. Pregnant women were categorized into three groups according to the amount of alcohol they consume: 1) non-drinking, 2) very low drinking, and 3) low drinking. Small amounts of alcohol consumption caused dysfunction of the LPO-AOD system and the development of OS in women, and had negative effects on infants.

The biomarkers of potentially harmful LPO, such as thiobarbituric acid reactants (TBARs), were higher in very low and low drinking mothers. The activity of the AOD system was lower among mothers who drank alcohol. Alcohol consumption decreased levels of retinol, SOD activity, GSH, and GR activity. Higher rates of pathological conditions, delayed development, and slower growth were observed among infants who were prenatally exposed to alcohol.

Conclusion: Identification and preventive interventions are needed for pregnant women who use alcohol in any amount. (**International Journal of Biomedicine. 2021;11(4):519-525.**)

Key Words: lipid peroxidation • antioxidant defense • pregnant women • infant • alcohol

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Abbreviations

AOD, antioxidant defense; **CDs**, conjugated dienes; **GPO**, glutathione peroxidase; **GSH**, reduced glutathione; **GSSG**, oxidized glutathione; **GST**, glutathione-S-transferase; **GR**, glutathione reductase; **LPO**, lipid peroxidation; **OS**, oxidative stress; **SOD**, superoxide dismutase; **TBARs**, thiobarbituric acid reactants.

Introduction

The period of organogenesis in embryonic development is critical because alcohol exposure during this time has long-term effects. One of the reasons for the teratogenic effect of alcohol is its rapid penetration through the placenta and blood-brain barrier, where alcohol can have effects that are more serious than those of many other substances, including exposure to maternal smoking and illicit drug use.[1-5]

There is evidence that the fetus is exposed to the same alcohol level as the mother. The effects of alcohol on the fetus are extremely destructive and damaging. The severity of the damage depends on many factors: maternal age, social environment, amount of alcohol consumed, frequency of alcohol consumption, duration of maternal alcohol abuse, and other factors.[6-17]

The biotransformation of ethanol is a typical reaction of toxification, which yields metabolites that are more toxic than

ethanol.[4,17,18] As shown in numerous studies, even small doses of ethanol and its metabolites, especially acetaldehyde, lead to congenital malformations of the child, directly or indirectly, through disruption of maternal biochemical mechanisms, and result in fetal alcohol syndrome.[1,4,8]

It is known that an imbalance in the LPO-AOD system leads to the development of OS, which is accompanied by a decrease in the body's resistance to adverse responses to the external and internal environment.[19-26] This imbalance can be assessed by measuring oxidized and reduced forms of glutathione, the activity of enzymes affecting glutathione metabolism (SOD, GR, GPO, and GST), substances that protect from OS (α-tocopherol and retinol), and products that result from oxidation (CDs and TBARS).

Few studies have reported the relationship between alcohol consumption and activities of components of the LPO-AOD system among mothers and infants, and how prenatal alcohol exposure affects infant growth, health, and development by the age of one year.

The aim of this study was to examine the effect of low or very low amounts of alcohol consumption on the LPO-AOD systems of pregnant women and their infants after birth, and the effect of that exposure on infant, growth, health, and development.

Materials and Methods

Study participants

Pregnant women consecutively enrolled in prenatal care at the Irkutsk Regional Perinatal Center (IRPC) between June 2012 and June 2014 were recruited to participate. Those women who initiated prenatal care at ≤10 weeks of gestation were eligible to participate. Since the study included mother-child dyads, if pregnancy was terminated or did not result in a live birth, the participant was removed from the study.

A total of 204 pregnant women were approached about participating in the study. Two refused to participate, resulting in a sample of 202 pregnant women (mother-child dyads) recruited for the study. All participants were admitted for delivery to IRPC. One delivery resulted in the neonate's death due to severe fetal distress and hypoxia within 24 hours of delivery, resulting in a final sample of 201 mother-child dyads.

Procedures and data collection

At the first prenatal visit, the women underwent standard medical procedures, including a prenatal care medical exam, were prescribed folic acid, and were provided with information about a healthy lifestyle during pregnancy (risks of alcohol use, tobacco use, and secondhand smoke). All participants completed a face-to-face interview with a study investigator and a brief intervention recommended for alcohol use.[27] At 30-32 weeks of gestation, maternal laboratory tests were completed. At delivery, a neonatologist examined neonates and samples of cord blood were collected. One-to-three days after delivery, mothers completed a face-to-face interview with the study investigator. Follow-up data on infant development were collected from child medical charts.

Collected data included maternal medical history and birth outcomes. Infant medical information noted by a developmental pediatrician at 6 and 12 months was extracted from the hospital's patient records. Characteristics of interest included socio-demographic characteristics, such as maternal age at the time of the birth and marital status; maternal medical history, including number of previous deliveries (parity) and chronic conditions; infectious diseases, such as HIV; pregnancy complications and mode of delivery (vaginal delivery or cesarean section); sex of the neonate, and; the neonate's birth weight, gestational age at birth, and evaluation (APGAR Score) at birth by a neonatologist.

Two face-to-face structured interviews were conducted with each participant by a study investigator. The interviews utilized measures developed by the Prevent FAS Research Group.[28] The first 20- to 30-minute interview was conducted at the time of the first prenatal visit to collect socio-demographic information and to assess women's drug use, alcohol use, smoking, and other risk factors prior to pregnancy. The second interview was conducted on the first to the third day after delivery to collect information about alcohol use and smoking during pregnancy, and took approximately 20 minutes to complete. The postnatal timing of reports was selected due to recent research data suggesting that pregnant women's reports about their consumption during pregnancy are more affected by biases than are their retrospective, after-pregnancy reports about drinking during pregnancy.[29] Several additional measures were implemented to further improve accuracy and elicit truthful self-reports. All interviews were conducted in a clinical setting in private. Participants were alcohol-free when interviewed and were reassured of confidentiality. Questions were worded clearly.

Alcohol exposure

Participants were asked to provide detailed reports about their alcohol consumption during 40 weeks of pregnancy. Following guidance from Sobell & Sobell (2003), participants were provided with a calendar and asked to memorize personal events that might be associated with alcohol use, such as holidays and birthdays. Although self-reports about alcohol consumption may be affected by desirability bias, they are considered to be reasonably accurate among volunteers recruited in health care settings when confidentiality is protected.[30-33]

The concept of "one drink" as a unit of consumption was not familiar to women in Siberia. Therefore, similar to beverage- and container-specific approaches that have been used in Russia[8] and other countries,[34,35] a beverage-specific approach was used to determine standardized alcohol content and volume of alcohol consumption. Participants were provided with a card that showed pictures of alcoholic beverages and containers that are common in Russia, and were asked about the type of beverage, type of container, and a number of containers consumed during each month of pregnancy. This information was converted into ethanol volume. The total amounts of alcohol consumed during the first half (1-20 weeks of gestation) and the second half (21-40 weeks) of pregnancy were calculated for each participant and utilized in data analysis. For reporting clarity, these data were then converted to U.S. standard drink units (i.e., 14 grams of pure alcohol).[36] The women were categorized as having very

low alcohol consumption if they drank less than 750 ml of beer, dry wine, or champagne, and were categorized as having low alcohol consumption if they consumed between 750 ml and 3850 ml of beer, dry wine, or champagne. The control group was women who never consumed alcohol during pregnancy.

Parameters of LPO-AOD system

Between 30 and 32 weeks gestation, fasting blood was drawn to measure indicators of LPO-AOD system function among pregnant women.

The SOD, GR, GST, and GPO activity was measured in erythrocytes using a commercially available kit. Fluorometry was used to measure GSH and GSSG levels in hemolysate.[37] Retinol and α-tocopherol levels were detected in plasma by fluorometry.[38] The concentration of CDs absorbance was detected on plasma heptane extracts at 232 nm.[39] The coefficient of molar absorption ($K=2.2 \cdot 10^5\ M^{-1}\ S^{-1}$) for conversation of absorption units of μmol/L was used. TBARs levels were detected by fluorometry in μmol/L.[40]

Umbilical cord blood samples were obtained from 140(69.7%) of 201 infants; 66(70.1%) of 93 infants in Group 1; 53(70.1%) of 75 infants in Group 2, and; 21(63.6%) of 33 infants in Group 3. We examined the same markers of the LPO-AOD system in infants as we did in their mothers.

Child developmental evaluation

Child development data, including infant height, weight, head and chest circumferences, congenital malformations, rickets, and developmental milestones, were extracted from the children's medical charts. Following routine medical procedures and standard protocols, neonates were evaluated at birth (n=201) by their clinic's neonatologists. At 6 and 12 months of age, infants (n=201) were evaluated by a pediatrician and a pediatric neurologist at the infant's local pediatric clinic. Anemia was diagnosed based on blood hemoglobin (≤12 g/dL for newborns; <10 g/dL for infants aged between 6 and 12 months). Rickets was diagnosed by clinical evaluation by a pediatrician. Congenital malformations included congenital heart disease, retroperitoneal tumor, adrenal gland tumor, malformations, fetal alcohol syndrome spectrum disorders, and cerebral palsy. Pediatric neurologists evaluated all children in the first 2-4 days of life and at 6 and 12 months to evaluate psychomotor development according to developmental milestones.

Statistical processing was carried out using the STATISTICA Version 10 (StatSoft, USA). The normality of distribution of continuous variables was tested by Shapiro-Wilk test. For descriptive analysis, results are presented as mean±standard deviation (SD), median (Me) (interquartile range [IQR]). Multiple comparisons were performed with one-way ANOVA and Tukey's HSD Post-hoc Test. Kruskal-Wallis test was used to compare means of 3 groups of variables not normally distributed. Categorical variables were analyzed using the chi-square test with the Yates' correction. A value of P<0.05 was considered significant.

The study was carried out in compliance with Ethical Principles for Medical Research Involving Human Subjects, Adopted by the 18th WMA General Assembly, Helsinki, Finland, June 1964, and amended by the 64th WMA General Assembly, Fortaleza, Brazil, October 2013. The study was approved by the Ethics Committee of the Scientific Center for Family Health and Human Reproduction Problems. Written informed consent was obtained from each patient.

Results

Clinical characteristics of pregnant women and infants at birth

We analyzed the clinical characteristics of the surveyed women and their newborns (Table 1). A total of 201 pregnant women aged 15 to 42 (29.1±6.0) enrolled in the study at their initiation of prenatal care at 7-to-10 weeks (7.1±0.5) of gestation. There were no differences in age, marital status, or other socio-demographic characteristics between women who reported consuming alcohol and those who reported no alcohol use during pregnancy. Among participants, 69.2% were married and 41.8% had a higher education than a school diploma or secondary education. A total of 37.3% and 34.8% reported smoking prior to pregnancy and during pregnancy. None were positive for HIV.

Table 1.
Characteristics of the surveyed pregnant women

Variable	Group 1 (n=93)	Group 2 (n=75)	Group 3 (n=33)	Statistics
Age, yrs	29.7±0.6	28.3±0.7	29.0±1.0	P=0.0000 P_{1-2}=0.0000 P^{1-3}=0.0000 P_{2-3}=0.0000
Weight, kg	68.2±1.2	69.1±0.3	68.8±0.4	P=0.0000 P_{1-2}=0.0000 P^{1-3}=0.0018 P_{2-3}=0.2144
Height, cm	166.3±0.7	166.1±0.3	166.7±0.6	P=0.0000 P_{1-2}=0.0610 P^{1-3}=0.0017 P_{2-3}=0.0000

In this sample (n=201), 93 participants reported no alcohol consumption during pregnancy (Group 1), and 108 reported consuming alcohol in some amount. Based on the total amount of alcohol reported during pregnancy, participants were categorized into three groups: no alcohol use during pregnancy (Group 1, n=93), very low (≤2 drinks) alcohol use (Group 2, n=75), and low (>2 drinks) alcohol use (Group 3, n=33). The average amounts of alcohol consumed were 1.28±0.48 drinks for Group 2 and 5.91±2.67 drinks for Group 3.

With regard to tobacco use, 15.1% of Group 1, 42.7% of Group 2, and 72.7% of Group 3 women reported smoking daily or occasionally, less than 10 and not more than 20 cigarettes a day, during pregnancy. Women with higher alcohol consumption during pregnancy smoked more often than did those with lower alcohol consumption during pregnancy (Group 1 vs. Group 2, P=0.005; Group 1 vs. Group 3, P=0.001; Group 2 vs. Group 3. P=0.04).

There were no significant differences between the heights, weights, and head and chest circumferences of the infants in any of the three groups (Table 2).

Table 2.

Characteristics of infants who were prenatally exposed to alcohol and infants with no alcohol exposure.

Variable	Group 1 (n=93)	Group 2 (n=75)	Group 3 (n=33)	Statistics
	M±SD; Me (IQR)			
Height, cm	50.9±2.9	50.1±4.3	49.7±4.4	P=0.1937 P_{1-2}=0.3532 P^{1-3}=0.2541 P_{2-3}=0.8652
Weight, kg	3.3(2.9-3.7)	3.2(2.8-3.7)	3.04(2.6-3.6)	P>0.05
Head circumference, cm	33.9±1.5	33.1±3.7	33.6±2.2	P=0.1494 P_{1-2}=0.1259 P^{1-3}=0.8405 P_{2-3}=0.6357
Chest circumference, cm	32.7±2.6	31.7±3.6	31.9±3.1	P=0.0973 P_{1-2}=0.0952 P^{1-3}=0.4090 P_{2-3}=0.9484

Development of infants at 6 and 12 months of age

Congenital malformations were significantly more common in children with low prenatal alcohol exposure at both ages, compared with the children with very low exposure (Table 3). Rickets was also significantly more common at 12 months in Group 3 children than in Group 2 children. Hypoxic-ischemic central nervous system damage in newborns was noted in 2.2% of infants from Group 1, 8% of infants from Group 2, and 24.4% of infants from Group 3.

Table 3.

Congenital malformations and other pathological conditions of infants at 6 and 12 months of age

Variable		Group 1 (n=93) n (%)	Group 2 (n=75) n (%)	Group 3 (n=33) n (%)	Statistics
6 months	Congenital malformations	4(4.3)	7(9.3)	10(30.3)	P=0.0001
	Rickets	18(19.4)	16(21.3)	7(21.2)	P=0.9436
	Anemia	22(23.7)	18(24.0)	8(24.2)	P=0.9970
12 months	Congenital malformations	4(4.3)	7(9.3)	10(30.3)	P=0.0001
	Rickets	10(10.8)	8(10.7)	7(21.2)	P=0.2477
	Anemia	12(12.9)	14(18.7)	8(24.2)	P=0.2881

Children in Groups 2 and 3 had significantly lower weights, heights, and head circumferences at 6 and 12 months of age, and significantly larger fontanelles at 12 months of age than did children in Group 1 (Table 4). Infants with very low exposure had significantly lower heights and weights than did infants with no exposure at 6 and 12 months.

In analyzing the physical and psychomotor development of children 6 and 12 months of age, those with low prenatal alcohol exposure were more likely to have psychomotor development delayed for their age. Thus, they had significantly more motor development delay at 6 and 12 months than did children with no exposure (Group 1 vs. Group3 at 6 months. *P*=0.023; Group 1 vs. Group 3 at 12 months, *P*=0.015).

Table 4.

Anthropometric data in the infants at the first year of life

Variable		Group 1 (n=93)	Group 2 (n=75)	Group 3 (n=33)	Statistics
		M±SD; Me (IQR)			
6 months	Weight, kg	7.7±0.5	6.9±0.5	6.8±0.6	P=0.0000 P_{1-2}=0.0000 P^{1-3}=0.0000 P_{2-3}=0.6251
	Height, cm	70.2±1.1	67.8±1.1	67.6±1.6	P=0.0000 P_{1-2}=0.0000 P^{1-3}=0.0000 P_{2-3}=0.7027
	Head circumference, cm	43.7±1.4	45.9±0.42	40.7±0.9	P=0.0000 P_{1-2}=0.0000 P^{1-3}=0.0000 P_{2-3}=0.0000
	Large fontanelle, cm	1.5(1.5-1.5)	1.5(1.5-1.5)	1.5(1.5-1.5)	NaN
12 months	Weight, kg	12.3±0.9	10.3±1.2	10.0±1.0	P=0.0000 P_{1-2}=0.0000 P^{1-3}=0.0000 P_{2-3}=0.3510
	Height, cm	81.2±2.0	78.1±1.8	77.6±2.0	P=0.0000 P_{1-2}=0.0000 P^{1-3}=0.0000 P_{2-3}=0.4301
	Head circumference, cm	48.8±0.9	51.4±0.5	44.5±1.3	P=0.0000 P_{1-2}=0.0000 P^{1-3}=0.0000 P_{2-3}=0.0000
	Large fontanelle, cm	1.0(0.5-1.0)	1.0(0.5-1.0)	1.0(0.5-1.0)	NaN

Characteristics of LPO-AOD process

The level of CDs was significantly lower in women of Group 2 than in women of Group 1 (*P*=0.003). The concentrations of TBARs in Groups 2 and 3 were significantly higher than those in Group 1 (Table 5).

There were no statistically significant differences in α-tocopherol levels between the groups. However, retinol concentrations were significantly lower in both drinking groups than in the non-drinking group. Dysregulation in the glutathione system was indicated by significantly lower GSH values and GR activity, and higher GSSG and GST levels, among women of Group 1 than in women of Group 3.

Table 6 indicates the levels of markers related to the LPO-AOD system in infants who were prenatally exposed to alcohol and infants with no alcohol exposure. CDs were statistically significantly higher in infants from Groups 2 and 3 than in infants from Group 1. In the AOD system, the values of α-tocopherol and SOD activity were significantly lower in Groups 2 and 3 than in Group 1. In terms of changes in the glutathione system, there were significantly lower levels of

GSH in Group 2 and GR in Groups 2 and 3 than in Group 1. Lower GR in Groups 2 and 3 was accompanied by a decrease in the activity of the other glutathione-dependent enzymes, GST and GP.

Table 5.

LPO-AOD system components in the pregnant women of the study groups

Variable	Group 1 (n=93)	Group 2 (n=75)	Group 3 (n=33)	Statistics
	M±SD; Me (IQR)			
CDs, µmol/L	2.12 (1.61-2.68)	1.19 (1.26-2.50)	1.84 (1.50-2.40)	P_{1-2}=0.003 P^{1-3}=0.088 P_{2--3}=0.989
TBARs, µmol/L	0.83 (0.61-1.12)	1.19 (0.7-1.57)	1.18 (0.87-1.54)	P_{1-2}<0.001 P^{1-3}<0.001 P_{2--3}=0.951
α-tocopherol, µmol/L	6.49 (5.48-7.77)	5.46 (4.10-8.07)	6.33 (4.77-9.88)	P_{1-2}=0.243 P^{1-3}=0.167 P_{2--3}=0.053
Retinol, µmol/L	0.75 (0.59-0.92)	0.58 (0.431-0.70)	0.57 (0.48-0.67)	P_{1-2}<0.001 P^{1-3}<0.001 P_{2--3}=0.769
SOD, U/mgHb	1.69±0.15	1.63±0.19	1.64±0.19	P=0.0658 P_{1-2}=0.0670 P^{1-3}=0.3275 P_{2--3}=0.9585
GSH, mmol/L	2.11 (1.85-2.41)	1.96 (1.81-2.24)	2.09 (1.72-2.39)	P_{1-2}=0.014 P^{1-3}=0.412 P_{2--3}=0.334
GSSG, mmol/L	1.81 (1.5-2.13)	1.92 (1.75-2.26)	1.97 (1.78-2.25)	P_{1-2}=0.023 P^{1-3}=0.025 P_{2--3}=0.717
GR, µmol/min/L	951 (752-1168)	764 (459-965)	855 (541-1097)	P_{1-2}<0.001 P^{1-3}=0.068 P_{2--3}=0.434
GST, µmol/min/L	809 (678-951)	966 (852-1450)	852 (457-1423)	P_{1-2}<0.001 P^{1-3}=0.068 P_{2--3}=0.434
GPO, µmol/min/L	279 (216-316)	256 (197-308)	275 (206-321)	P_{1-2}=0.260 P^{1-3}=0.895 P_{2--3}=0.364

Table 6.

LPO-AOD components in infants who were prenatally exposed to alcohol and infants with no alcohol exposure

Variable	Group 1 (n=66)	Group 2 (n=53)	Group 3 (n=21)	Statistics
	M±SD; Me (IQR)			
CDs, µmol/L	1.34 (1.12-1.67)	1.73 (0.98-2.13)	1.68 (1.52-2.13)	P_{1-2}=0.006 P^{1-3}<0.001 P_{2--3}=0.396
TBARs, µmol/L	1.32 (1.06-1.61)	1.35 (1.16-1.64)	1.31 (0.90-1.44)	P_{1-2}=0.976 P^{1-3}=0.764 P_{2--3}=0.771
α-tocopherol, µmol/L	6.98 (5.84-8.17)	5.88 (5.22-7.05)	6.08 (5.21-7.15)	P_{1-2}=0.006 P^{1-3}=0.029 P_{2--3}=0.823
Retinol, µmol/L	0.41±0.10	0.43±0.11	0.42±0.10	P_{1-2}=0.385 P^{1-3}=0.876 P_{2--3}=0.460

Table 6 (continued).

LPO-AOD components in infants who were prenatally exposed to alcohol and infants with no alcohol exposure

Variable	Group 1 (n=66)	Group 2 (n=53)	Group 3 (n=21)	Statistics
	M±SD; Me (IQR)			
SOD, U/mg Hb	1.74±0.08	1.64±0.14	1.69±0.13	P_{1-2}<0.010 P^{1-3}=0.030 P_{2--3}=0.109
GSH, mmol/L	2.26±0.40	2.04±0.28	2.15±0.29	P_{1-2}=0.014 P^{1-3}=0.223 P_{2--3}=0.139
GSSG, mmol/L	1.97 (1.66-2.20)	1.96 (1.78-2.29)	1.96 (1.59-2.06)	P_{1-2}=0.781 P^{1-3}=0.197 P_{2--3}=0.151
GR, µmol/min/L	857.5 (731-985)	652 (456-851)	699 (456-1085)	P_{1-2}<0.001 P^{1-3}<0.001 P_{2--3}=0.277
GST, µmol/min/L	1244 (1056-1478)	995 (852-1431)	1133 (813-1455)	P_{1-2}=0.012 P^{1-3}=0.041 P_{2--3}=0.961
GPO, µmol/min/L	280 (231-318)	231 (199-291)	245 (233-316)	P_{1-2}=0.002 P^{1-3}=0.031 P_{2--3}=0.396

Discussion

Our findings indicate that even a small amount of alcohol consumed during pregnancy can cause serious metabolic changes in mothers and their newborn babies. In particular, alcohol exposure leads to an imbalance of redox exchange and dysfunction of the LPO-AOD system. The concentrations of TBARs were 60.4% higher in the very low and low drinking groups than in the control group of non-drinkers. The increase of TBARs indicates the decline of AOD that can be described as the OS development.[22]

Retinol concentration decreased in both drinking groups, compared with the control group**.** This reduction may result in a corresponding reduction in the antioxidant effect of retinol. The role of retinol as a prohormone may also be affected by oxidation, prompting its development into retinoic acid, a true hormone, which is involved in the regulation of gene expression.[21,41] Further, retinoic acid has morphogenetic action, and its deficiency can lead to fetal malformations.

SOD activity was significantly lower in very low drinkers than in non-drinkers. Even a small reduction in SOD activity is an important signal of the metabolic shift in the direction of prevailing pro-oxidant processes, because of the high content of the enzyme in the red blood cell.[21] Women who drank very low amounts of alcohol had lower GSH and GR values, and higher GSSG and GST values, than did those who drank no alcohol. The reduced form of glutathione participates in neutralizing oxidants and in transporting substances across membranes, and has an antitoxic effect.[24,42-44] The decreased GSH and GR levels in women who consumed alcoholic beverages had a negative effect on their health.[45] Reduced activity of GR in women who consumed even very low doses of alcohol, in turn, indicates the protective function of the enzyme, conversion of GSSG to GSH.

Infants prenatally exposed to small and moderate amounts of alcohol had higher rates of pathological conditions, smaller heights and weights, larger fontanelles, and smaller head circumferences at birth and 6 and 12 months of age, as well as delayed psychomotor development than those with no alcohol exposure. In terms of the LPO-AOD system of newborns, changes similar to those that occurred in their mothers were found, especially for SOD, GSH, and GR. The decrease in GSH concentration and activity of the enzymes involved in its metabolism, which were found in infants of Group 2, has a negative effect on the balance in the LPO-AOD system. It is reported that α-tocopherol activated γ-glutamyl cysteine synthetase, which leads to regulation of glutathione biosynthesis. In this case, α-tocopherol has an indirect effect on the AOD system.[1]

The assessments of the LPO-AOD system for women who consumed alcoholic beverages in the prenatal period indicate increased OS, even when consuming low doses. Thus, the results suggest that even a small amount of alcohol drunk by women during pregnancy can cause serious metabolic changes in the newborn body, leading to an imbalance of redox exchange and dysfunction of the LPO-AOD system.

Conclusion

The use of even small doses of alcohol during pregnancy can cause dysfunction of the LPO-AOD system and the development of OS and can have negative effects on infant growth, health, and development. Specialists and researchers in the medical and social fields must address the prevention of any amount of alcohol use in pregnant women.

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Conflict of Interests

The authors declare that they have no conflicts of interest.

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