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The Importance of Selenium in Children's Health and Reproduction

O.A. Senkevich and Y.G. Koval'skiy

Abstract

The authors report selenium deficiency in pregnant women, which can lead to an increase in the frequency and severity of early and late gestosis, fetal hypotrophy, hypoxia, and increased risk of miscarriage. The provision of selenium in children depends on the degree of maturity and physical development, decreases with fetal hypotrophy, prematurity, artificial feeding, and hypoxia. The content of selenium in breast milk of women who gave birth prematurely, contains selenium three times less than in normal childbirth, which contributes to a high risk of alimentary-dependent conditions in premature infants.

Keywords: selenium, children, pregnant women, reproduction, placenta, breast milk

1. Introduction

Pregnancy is a period of important conditional physiological changes, when the fetus requires a regular and balanced diet provided by the mother's food and its physiological reserves. The intrauterine period, infancy, and early age are considered to be a critical period in terms of the impact of nutrition on subsequent development [1].

For the growth and development of the fetus, normal pregnancy requires a constant consumption of sufficient amounts of nutrients, at least 40 of them are essential for pregnant and lactating women. Nutritional status of pregnant and lactating women is of great importance for the health of the child, as the development of the body is most actively carried out up to 18 months of life.

2. Value of selenium deficiency in the perinatal period

Adequate provision of selenium for pregnant women, premature infants, children of different ages, and adolescents living in environmentally unfavorable conditions and constituting a risk group for selenium deficiency is particularly relevant [2–7].

Nefedova et al. [8] believe that the fact of selenodetic in healthy women in Western Siberia is associated with the possible formation of "anomalous biogeochemical province" endemic selenium. In pregnancy, even with the physiological course, this deficit is exacerbated, which is an understandable increased

expenditure of trace elements [9–11]. In women at risk of termination of pregnancy, selenium deficiency is most pronounced, with pregnancy ending in miscarriage at different stages [12–15].

The level of selenium (serum, erythrocytes, urine, and hair) in healthy adult blood donors, residents of Khabarovsk in the preconception (indicator group), at the end of physiological pregnancy (group “healthy pregnant”) and breast milk on the 7–10 day after birth in mothers of healthy newborns and preterm infants was established.

There were no differences in the content of selenium in the serum of young healthy nonpregnant women, donor volunteers (residents of Khabarovsk, examined by random sampling), healthy pregnant women at the end of physiological pregnancy, and healthy puerperas. The provision in these groups was found to be average (82–85 $\mu\text{g/l}$), which is approximately 80% of the optimal level, since the interval of normal serum concentrations of selenium is on average 115 $\mu\text{g/l}$. At the end of physiological pregnancy and normal childbirth, the level of selenium in the blood serum was determined within $85.4 \pm 4.8 \mu\text{g/l}$ and 82.6 ± 6.1 , respectively, which is lower than optimal for pregnant women. In premature births, serum levels of selenium were significantly lower—58.2 $\mu\text{g/l}$, which is about half of the optimum. There are no significant differences in the content of selenium in the blood inversion in smoking and nonsmoking women in the indicator group (82.8 ± 16.2 and $83.1 \pm 15.9 \mu\text{g/l}$) or in the group of healthy pregnant women (84.7 ± 15.8 and $85.8 \pm 16.1 \mu\text{g/l}$), in the presence of significant differences in the hair of smoking and nonsmoking pregnant women (383.0 ± 24.4 and $436.6 \pm 28.2 \mu\text{g/l}$, respectively, $p < 0.01$).

During pregnancy, there is a significant reduction in the excretion of selenium in the urine, which ensures the maximum possible provision of the fetus with a trace element [16]. Excretion of selenium in urine is constant and is normally 40–50% of intake [17]. Losses of selenium with urine in the group of healthy pregnant women in Khabarovsk ranged from 12.6 to 40.8 $\mu\text{g/l}$, on average 23, 68 $\mu\text{g/l}$, which differs with a high degree of reliability from the standards described in the literature.

A direct relationship of average strength between the level of selenium in the hair and urine of pregnant women indicate unidirectional changes in the concentration of selenium in the hair and urine of pregnant women.

Also in the process of gestation, there is a change in the content of selenium in serum and in hair, and there is a highly reliable feedback of average strength between the content of selenium in serum and hair of pregnant women.

In young healthy nonpregnant women, the existence of a direct relationship of moderate strength ($r = 0.4804$) is established between selenium content in blood plasma and urine, and there is a highly significant direct strong correlation between selenium in serum and erythrocytes ($r = 0.9552$), direct medium strength correlation between selenium in urine and selenium in erythrocytes ($r = 0.4348$). At the same time, during the physiological course of the gestational process in healthy mothers, serum selenium levels and urine selenium values have a direct relationship of average strength.

The loss of selenium in urine was significantly lower ($p < 0.05$) in women with a long labor period ($18.6 \pm 0.76 \mu\text{g/l}$) and higher in mothers with planned operative labor ($24.9 \pm 1.3 \mu\text{g/l}$), and there was no dependence on the gestation period. Probably, the detected changes are associated with the development of oxidant stress; since the intensive physical activity (childbirth) determines the acceleration of metabolic processes, leading to significant oxidant stress of the body, a specific mechanism is included that ensures the preservation of selenium by reducing its excretion in the urine [18].

On the territory of the Khabarovsk, there is a shortage of selenium, because in the food diet of the inhabitants of the region, local products prevail, including from

household plots, which, in conditions of natural low maintenance, contain little of this element. The problem is also aggravated by the fact that our study of the diet of young children showed a significant deficit in the consumption of the surveyed residents of Khabarovsk products—sources of selenium, which undoubtedly exacerbates the population deficit.

In the epidemiological study of the provision of selenium in children permanently living in Khabarovsk, we have established a significant variability in the provision of selenium in different age groups of children with maximum security in adolescents 12–17 years; the minimum level of selenium was detected in children aged 2 years of life, due to the peculiarities of nutrition and food preferences of children of different ages. In general, selenium deficiency was observed in 18% of the surveyed children; only 28% of children found trace element content at the lower limit of the norm. The data obtained by us on the provision of selenium for children and residents of Khabarovsk are comparable with the data obtained by us from the adult population [19].

3. Placenta as an indicator of fetal selenium content

The placenta is a unique, complex organ composed of the maternal and fetal parts. It is a full component of the system and performs numerous functions, the violation of which leads to a wide range of pregnancy complications [20]. The placenta has a wide range of functions that go in both ways; directly from maternal body to placenta; from placenta to fetus, and vice versa, from the fetus to the mother and placenta [21–23]. Placental dysfunction is a threat to the development and life of the fetus, and subsequently the newborn. In physiological pregnancy, the mother's body retains its homeostasis and provides the fetus with everything necessary for normal development [24, 25]. Studies in recent years [3, 26] show that the inability of the system of “mother-placenta-fetus” to maintain adequate exchanges between mother and fetus (feto-placental insufficiency) leads to violation of fetal development and homeostasis of the mother. The value of the placenta is as an indicator of fetal selenium content [20–22, 27].

It is known that the mass of the newborn is largely determined by serum selenium [21, 28]; however, the unknown is the correlation between the content of selenium level with the mass of the placenta and fetus. During the multivariate correlation analysis, it was found that there is an average strength positive correlation ($R = 0.53$; $p = 0.002$) between the body weight of premature infants born at gestation 28–36 weeks, and the content of selenium in the placenta.

Therefore, the higher the level of selenium in the placenta, the greater the body weight of the newborn. The same relationship was established in the group of children with psrp ($R = 0.57$; $p = 0.03$). The obtained results do not confirm the data of Zadrozna et al. [27], which did not reveal this dependence. A negative correlation between the placental-fetal coefficient and the content of selenium in the placenta ($R = -0.55$; $p = 0.002$) was established [29].

In the study of the accumulation of selenium in the placenta, depending on the duration of pregnancy, it was found that the minimum content was observed at the gestation period of 28–33 weeks and significantly different from the indicators in other groups [29]. The content of selenium in the placenta of women in countries such as Russia, Ukraine, Poland, Spain, and Turkey is in the range of 0.15–1.65 mg/kg [21–23, 30, 31], which is significantly higher.

There is a positive correlation between the average strength between the levels of selenium in the placenta and the evaluation of the newborn on the Apgar scale both at the first ($R = 0.45$; $p = 0.02$) and fifth minute ($R = 0.36$; $p = 0.005$), which

indicates a better tolerability of labor stress in newborns with a greater reserve of this microelements antioxidant properties in the placenta.

Clinical and diagnostic parallels were made to assess the role of trace element imbalance in the placenta in the formation of pathological conditions of newborns in the early neonatal period by calculating the relative risk (RR) with 95% confidence interval.

It was found that the deficiency of selenium significantly and statistically significantly affects the low score on the Apgar scale in the first minute in newborns—more often 2.5 times (OR with CI 1.1–2.5) for selenium. A low content of selenium in the placenta in newborns significantly increases the risk of neutrophilia with OR—1.6 (CI 1.1–3.0). The above parallels occur reliably in all study groups. In the pathogenesis of RDS, selenium deficiency probably plays a significant role because its deficiency not only in the hair of the mother and newborn, but also in the placenta at gestation 28–33 weeks forms the risk of RDS and is 1.9 CI 1.06–4.3.

It was found that the most pronounced imbalance of trace elements in the groups of premature infants with a gestation period of 28–33 weeks and in newborns with intrauterine growth retardation syndrome, which is a manifestation of immaturity in the first and decompensation of the function of passive transport systems in the second case. Clinically, this will be manifested by a violation of acute neonatal adaptation with a low Apgar score, severe general condition, and the presence of markers of inflammatory response.

In serum and placenta, selenium to some extent mimics the behavior of zinc and behaves opposite to copper [32]. It is interesting to note that active smoking gave higher levels of selenium and zinc in the placenta [32]. Both elements play an important role in protecting against oxidant stress: selenium in glutathione peroxidase and zinc in superoxide dismutase. Smoking causes severe chemical stress to the placenta, so a higher concentration of zinc and selenium in the placenta in smokers may reflect the activation of protective mechanisms.

When smoking, the transfer of selenium from the blood to the placenta is increased, so that the level of selenium in the blood decreases; while the level of selenium in the placenta of smoking women is much higher than in nonsmokers. In general, the results suggest that in women smokers, selenium transport appears to be part of a protective mechanism against chemical stress.

Selenium concentrations during pregnancy are reduced in serum and placenta by the end of pregnancy and increased in the placenta of smokers. Since selenium status negatively affects the level of zinc in breast milk [33, 34], the concentration of selenium can affect the level of zinc in the placenta and serum by changing the distribution of zinc. Low concentrations of serum selenium and low copper concentrations in the placenta are associated with higher weight of newborn, which reflects the correlation between serum selenium and copper of the placenta. A strong positive correlation in serum between selenium and copper correspond to such in milk [34], although the reason for this is not clear. An inverse correlation between the activity of aromatase and placenta selenium was revealed, which indicates the protective effect of selenium by acting on the activity of the enzyme to transfer nutrients to the fruit. The authors conclude that it is essential to identify the relationship between selenium, zinc, and copper. Thus, the protective role is established selenium during pregnancy, especially in smokers (**Table 1**).

It is shown that Cd and Pb placenta have an inverse correlation with the body weight of the newborn [25, 36], and these elements are selenium antagonists which reduce their toxic effect [36–38].

In Croatia, Alexiou et al., [35], indicated that Se in the placenta predicts birth of a child of normal weight, not intrauterine growth restriction. In this work, a significant positive relationship between placental Se and neonatal weight in a

Indicator	Level	Region
Se placenta	810 ± 20 µg/kg	Spain*
Se umbilical cord blood	74 ± 7 µg/kg (51–104)	
Se mothers blood	90 ± 15 (57–118)	
Se mothers hair	600 ± 370 (220–1500)	
Se newborn hair	1040 ± 480 (400–2530 µg/kg)	
Placenta	200 µg/kg raw weight	Austria**
Placenta		Italy***
Placenta (normal pregnancy) (36)	150 ± 30 (100–240 МКГ/КГ)	Croatia**** (significant differences with the norm P < 0.05)
Placenta intrauterine growth restriction cases (49)	140 ± 20 (100–200 МКГ/КГ)	

*Lorenzo Alonso et al. [22, 23].
 **Iyengar and Raphuman [31].
 ***Capellia et al. [21].
 ****Alexiou et al. [35].

Table 1.
Indicators of selenium in the placenta.

subgroup of pregnant women with body weight appropriate for this gestational age was established. This is consistent with a number of important functions that can be used to influence fetal development. On the other hand, the lack of communication in the IWRM group may mean that the element is not causally related to the condition and further research is needed on this issue. In studies Alexiou et al. [35] studied trace elements (zinc, cobalt, selenium, rubidium, bromine, gold) in the human placenta and in the liver of the newborn at birth, the authors note that the average concentration of essential trace elements (zinc, cobalt and selenium) were significantly higher in the liver than in the placenta, while interchangeable trace elements (rubidium, bromine, gold) were found in significantly higher concentrations in the placenta than in liver tissue.

Since the decrease in serum selenium level during pregnancy occurs monotonously, it is possible to calculate the optimal concentration of selenium in the serum of pregnant women: by the end of the first trimester—104 to 109 µg/l, the second—98 to 103 µg/l, to the third trimester—95 to 100 µg/l [39], these indicators can be used to assess the normal selenium status during pregnancy.

There is evidence of a connection with selenium deficiency weak labor activity, a significantly greater number of complications during delivery and lower development indicators of newborn [40]. The risk group for selenium deficiency is also pregnant women living in ecologically unfavorable regions and women with cardiovascular diseases [30]. In several case studies, the authors attributed the poor pregnancy outcome and reproductive failure, including recurrent pregnancy loss, with abnormal concentrations of selenium [12]. Other authors [13, 41, 42] also found a positive correlation between the increased risk of miscarriage in women with low selenium concentrations. Selenium reduction is a predictor of preterm birth and low birth weight, and correction of selenium deficiency during pregnancy eliminates the risk of low birth weight [28].

The concentration of selenium in the placenta at different periods of gestation was studied in children—residents of the Khabarovsk territory [43]. Estimating the central values of the studied microelements, the norm was understood as variants within one standard deviation; the boundaries of the norm were the values between the 25th and 75th centiles or (M + 2δ), and the pathological values beyond these limits were considered. To calculate the extreme limits of normal values, the

Group	n	Average	m	Deviation interval
28–33 gw	14	0.217 ^{*,***}	0.01	0.160–0.278
34–36 gw	17	0.341 [*]	0.03	0.176–0.588
Fetal growth Retardation	14	0.271 ^{**}	0.01	0.196–0.343
Control group	20	0.370 ^{***}	0.04	0.233–0.626

^{*}Significant difference between 28 and 33 weeks and 34 and 36 weeks ($p = 0.0017$).

^{**}Significant difference between 28 and 33 weeks and DLD don ($p = 0.0013$).

^{***}Significant difference between 28 and 33 weeks and full term ($p = 0.00003$).

Table 2.
Selenium concentration (mg/kg) in the placenta at different gestational ages.

method of standard deviations was used—the lower limit—5 SD corresponds to 5 percentiles; the upper limit +5 SD—95 percentiles. Based on this, for all cases with values below 5 percentile, we consider a sign of a significant decrease in the parameter, and with values more than 95 percentile—it is stated as an increase (Table 2).

It is interesting to note that with the increase in gestation period, there is a more significant variability of selenium content, which does not occur in the group of children with intrauterine growth retardation syndrome.

Our findings suggest that passive transport through the placenta of selenium has insufficiently mature mechanisms of its regulation in the early stages of embryogenesis.

The study revealed that the level of selenium in the placenta has a dynamics similar to that in the hair of the mother and the newborn and in breast milk [29, 44].

The placenta is an organ that reflects the features of the course of the intrauterine period, the environmental situation, and the infectious background, and it is also an indicator of the content of vital for the normal carrying of the newborn trace elements.

4. Breast milk is the only natural source of selenium for infants

The only natural vitamin and mineral complex is breast milk; it contains all the necessary bioelements and is most adapted to the assimilation of the child. However, the composition of breast milk, both qualitatively and quantitatively, depends on the time of onset of labor, significantly different from normal indicators for premature termination of pregnancy and fetal growth retardation syndrome. Of particular importance for solving the problem of nutrition of small children [45] is a detailed explanation of the properties of breast milk and determining the value of each of its components [46].

Given that the child in the first months of life receives selenium exclusively from mother's milk and baby food does not always contain it in sufficient quantities, of particular importance is the provision of this micronutrient women, both during pregnancy and during lactation. The estimated need for selenium in premature infants is 20–25 µg/l in breast milk or infant formula (15 µg/l for full-term infants) [47, 48]. The main source of selenium for a child is breast milk, but many children, especially those receiving long-term inpatient treatment, are artificially fed and have lower values of selenium content [49]. In addition, the presence of selenium deficiency in the mother is a common cause of element deficiency in the newborn [50, 51]. The content of selenium in breast milk varies widely [9, 50, 52]. Thus, in the study of selenium in breast milk of women living in the United States, it was determined that the average selenium content is 18 µg/l, and the maximum level

reached 60 µg/l [52]; a direct correlation was found between the level of selenium in milk and serum. In premature births, the milk of women living in New Zealand contains an average of 20 µg/l selenium [53]. The question of the needs of newborns in selenium is not finally resolved, but most researchers recommend enriching the mixture for children with selenium in an amount corresponding to its content in breast milk. But the supply of children with selenium in an amount equal to its content in breast milk is not equivalent, because in breast milk, mixtures contain different chemical forms of selenium with different levels of bioavailability and toxicity. Currently, baby foods mainly use selenite, which easily interacts with the ascorbic acid contained therein, forming an inactive elemental selenium; as a result, such products are inert against selenium [54]. Therefore, it seems natural to normalize the level of selenium in the mother feeding a newborn baby breast milk.

The content of selenium in breast milk during normal pregnancy and physiological childbirth by a conditionally healthy fetus and at low birth weight (**Table 3**) was determined in Khabarovsk [55].

When compared with the data of the WHO/MAGATE collaborative study [56], there were significant differences in the content of selenium in the milk of healthy women living in Khabarovsk (**Table 3**). The most pronounced deficiency of selenium in breast milk is preterm labor and fetal growth retardation syndrome; its level is 2 and 20 times lower than optimal, respectively, and from the first days of life of a small child, its insufficiency is formed with a significant ($p < 0.05$) maximum decrease in premature infants with a gestation period of 34–36 weeks and children with fetal growth retardation syndrome.

Selenium supply in newborns in the control group is optimal (**Table 4**).

At low weight, the actual provision of newborn children with selenium did not meet the standards of physiological needs of the body. The extremely low figures for the actual supply of selenium in natural feeding are detected in underweight children with the syndrome of fetal growth retardation and prematurity. Low level of selenium from the first days of life of a small child forms its negative balance. Given that the need for selenium at low weight is higher, and breast milk [55], as the only source

Trace elements	Control group, n = 20	28–33 weeks of gestation, n = 20	34–36 weeks of gestation, n = 20	Fetal growth retardation, n = 19	Literature data
Se	0.02 ± 0.007	0.01 ± 0.004**	0.009 ± 0.002*	0.001 ± 0.0002**	0.019 ± 0.001

*Significance of differences between the control group and childbirth at 34–36 weeks, $p < 0.05$.
 **Significance of differences between the control group, 28–33 weeks of labor, 34–36 weeks of labor, and fetal growth retardation syndrome, $p < 0.05$.

Table 3.
 Content of Se (mg/kg) in milk (M + m).

Groups	n	Se
The norm of physiological needs*		0.01
Childbirth 28–33 w	n = 20	0.003
Childbirth 34–36 w	n = 20	0.003
Childbirth a delay syndrome fetal growth retardation	n = 19	0.0003
Control group	n = 20	0.013

*Norms of physiological needs for energy and nutrients for different groups of the population of the Russian Federation were approved on December 18, 2008 (Mr 2.3.1.2432–08).

Table 4.
 Actual consumption of bioelements (mg/day).

Indicators	All subjects 13 months after delivery, n = 20	Lactation, 13 months after birth, n = 7	No lactation, 13 months after delivery, n = 13	Immediately after birth
Se	359.9 ± 18.9*	314.6 ± 20.2*	388.7 ± 25.1	524.6 ± 55.0*

*Significant differences (<0.05) between groups.

Table 5. Selenium content ($\mu\text{g}/\text{kg}$) in women's hair after childbirth ($M + m$).

of selenium for the newborn, contains less than optimal selenium, such a child is provided with the necessary trace element only 25–30% in premature birth and only 3% of the need for the birth of a child with delayed fetal development syndrome.

In the study of selenium content (**Table 5**) in the hair of 20 healthy women who gave birth to healthy children, a few months after birth, and an average of $13.05 + 0.54$ months after birth (variability from 10 to 18 months) there were significant differences in the content of selenium in women's hair 13 months after birth depending on the duration of lactation. At the time of the study, seven women continued to feed their children with breast milk, the rest of the lactation ended 6 months or more ago [3].

In women with preserved lactation function by 13 months after birth, the level of selenium is significantly lower than in women who breastfed children for a shorter time.

Regardless of the presence of lactation, all subjects had significantly lower levels of selenium than immediately after birth. Thus, in the above observations, there is no recovery of selenium concentration after childbirth, typical for the content of selenium in blood serum. There were no significant differences in the content of selenium in the hair of women a year after childbirth with or without the use of vitamin complexes during lactation ($368.9 + 22.13$ and $336.4 + 38.7 \mu\text{g}/\text{kg}$, respectively).

5. Children

Premature infants with low selenium levels have a higher rate of early neonatal morbidity [57]. The risk group for selenium deficiency includes children receiving long-term hemodialysis [18], with respiratory distress syndrome [58]; and children born in a state of chronic intrauterine hypoxia [59], with bronchopulmonary dysplasia [60]. In premature infants, selenium deficiency is associated with hypoxia and respiratory diseases [61]. In all these cases, the appointment of selenium is accompanied by a positive therapeutic effect. Confirmation of the diagnosis of chronic intrauterine hypoxia in the fetus can be a decrease in the content of selenium in the blood less than $25 \mu\text{g}/\text{l}$ [62]. Based on a multicenter randomized trial conducted by Darlow et al. in 2003, including a meta-analysis of other studies in this area, the additional use of selenium in the diet of preterm infants contributed to a reduction in the incidence of septic complications, which allowed the authors to recommend the use of selenium in the diet of preterm infants [53].

However, the optimal selenium supply of a newborn child is not currently determined; in the literature, there are different indicators of the norm from $65\text{--}75 \mu\text{g}/\text{l}$ [63] to $191 \mu\text{g}/\text{l}$ [64] in whole umbilical cord blood. In a study by Anya et al. [65] in the reference group of healthy newborns normal level of selenium was established in children younger than 1 month of life within the median $64 \mu\text{g}/\text{L}$. Parfenova and Reshetnik [66] in the whole blood of preterm infants 27–32 weeks of gestation the level of selenium equal to $112.4 + 5.3 \mu\text{g}/\text{l}$ was determined.

Group	28–33 gw	34–36 gw	Fetal growth retardation	Control
n	23	34	21	20
Mother	0.3 ± 0.04* 0.02–0.7	0.4 ± 0.06* 0.02–1.4	0.4 ± 0.07* 0.02–0.8	0.6 ± 0.05 0.4–1.1
Children	0.5 ± 0.07* 0.04–0.9	0.6 ± 0.05* 0.08–1.01	0.2 ± 0.04*** 0.01–0.6	0.8 ± 0.05 0.2–0.96

*Significant difference ($p < 0.05$) between study and control groups.

**Significant difference ($p < 0.05$) when comparing the indicators of the fetal growth retardation group with the groups of 28–33 weeks and 34–36 weeks.

Table 6.

Selenium content (mg/kg) in the hair of mother–newborn pairs in prematurity and delay syndrome fetal growth retardation ($M \pm m$, min–max).

The concentration of selenium in serum is reduced in children with intraventricular hemorrhage [67], diseases of the respiratory system [68], and diseases of the gastrointestinal tract [69]. However, first of all, premature children are a risk group for selenium deficiency, aggravated by living in environmentally unfavorable conditions [3, 61, 70]. Many of them, especially those receiving long-term inpatient treatment, are artificially fed, and baby foods contain predominantly sodium selenite—an inorganic compound of selenium with high toxicity, low bioavailability and not always in sufficient quantities. Selenium obtained from mother's milk is better absorbed than selenium nutrient mixtures [71]. It is recommended to add selenium to the nutrition of mothers, as well as cows whose milk is used for the preparation of nutrient mixtures [63].

The mass of the newborn is largely determined by serum selenium and is inversely correlated. This also confirms the assumption that selenium is actively transported to the fetus in the quantities required by the embryo (**Table 6**).

Selenium levels in the hair of low-weight newborns and their mothers are statistically significantly different from the levels of selenium in the hair in the control group, with the highest degree of confidence established in the group with fetal growth retardation.

The deeper the immaturity and the lower the gestation period, the lower the level of selenium in the hair of premature infants and their mothers; the level of selenium in the hair of the group with fetal growth retardation decreased more than four times and was significantly lower than in the group of premature infants with the deepest immaturity.

Reduction of selenium in the hair of newborns increases the chance of oppression syndrome, muscle dystonia, and neutrophilia. RDS is often formed when selenium deficiency in the pair “mother–newborn” in prematurity.

Direct dependence of the average strength between the level of selenium in the hair and urine of pregnant women indicates unidirectional changes in the concentration of selenium.

Also in the process of gestation, there is a change in the content of selenium in serum and hair and a highly reliable feedback of average strength ($r = 0.538$) between the content of selenium in serum and hair of pregnant women.

6. Conclusion

Selenium deficiency in pregnant women can lead to an increase in the frequency and severity of early and late gestosis, fetal hypotrophy, and hypoxia, and has an impact on the duration of pregnancy and the rate of growth of fetal body weight. Selenium deficiency in pregnant women can lead to an increase in the frequency

and severity of early and late gestosis, fetal hypotrophy and hypoxia, and also affects the duration of pregnancy and the rate of growth of fetal body weight, the formation of a deficiency in the fetus and newborn [15].

The study found a deficiency of selenium in women in preterm labor and is due not only to the lack of an element in the diet and the environment, but also an excess of selenium antagonists—Mn, Cd, Pb, and Fe.

The provision of selenium depends on the degree of maturity and physical development of newborns, decreases with fetal development syndrome, prematurity, artificial feeding, and hypoxia. The content of selenium in breast milk of prematurely born women provides only 25–30% of the needs of the element in premature infants. Breast milk of prematurely born women contains selenium three times less than in normal childbirth, which contributes to a high risk of developing alimentary-dependent conditions in premature infants [72].

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