Fetal brain development is an important determinant of neuropsychological performance in children. Any alterations in the intrauterine environment at different stages of pregnancy, such as maternal metabolic disorders, can lead to the development of chronic conditions in the offspring. Therefore, maternal diabetes, especially gestational diabetes mellitus, is an important factor in the development of pathological changes, such as miscarriage, fetal macrosomia, or neurodevelopmental disorders. During pregnancy, the hyperglycemic intrauterine environment adversely affects fetal brain development. A growing body of scientific research indicates that prenatal environmental factors, by affecting fetal brain development, can contribute to the appearance of autism spectrum disorders. According to the latest estimates from the International Diabetes Federation (2021), approximately 21.1 million live births worldwide (16.7%) have been affected by some form of hyperglycemia during pregnancy. The condition is more prevalent in low- and middle-income countries, where access to obstetric care is limited. The following factors have been identified as potential risk factors for gestational diabetes: advanced maternal age, overweight and obesity, family history of diabetes, and any form of diabetes. The purpose of this review is to summarize recent studies evaluating the effect of prenatal and maternal risk factors such as maternal pre-pregnancy diabetes, gestational diabetes, and obesity on the risk of developing autism spectrum disorder in offspring.

Keywords: Autism Spectrum Disorder • Diabetes Mellitus • Obesity, Maternal
Introduction

According to the International Statistical Classification of Diseases and Related Health Problems (ICD-10), autism spectrum disorder (ASD) is a pervasive developmental disorder identified by the presence of abnormal and/or impaired development that manifests before the age of 3 years. It includes the following areas: social interaction, communication, and restricted repetitive behavior [1,2].

The Diagnostic and Statistical Manual of Mental Disorders 5 (DSM-5) criteria for ASD include a specifier recommending consideration of the potential role of medical conditions, genetic conditions, and environmental factors associated with atypical neurological development [3].

Autism, or ASD, is a diverse group of conditions related to brain development [4]. The disorder includes domain deficits in social interactions, communication, and learning. There are also atypical patterns of actions and behaviors, specific response to stimuli, and focus on details [3,4].

The etiology of ASD remains unknown, and genetic and environmental factors are involved in its complex pathogenesis [4-6]. Sources report that late-onset disintegrative disorder in children (a type of ASD) may be associated with subacute sclerosing panencephalitis, tuberous sclerosis, leukodystrophy, or lipid storage diseases [2].

It is postulated that ASD is a multifactorial disease, in which genetic factors and environmental factors exert an influence during the critical period of brain development. Perinatal and intrapartum conditions have a significant effect on the development of autism. The risk was shown to increase when children experienced antenatal and intrapartum complications [5,6].

Maternal exposure to gestational diabetes mellitus (GDM) is an independent risk factor for long-term neuropsychiatric diseases in offspring, including ASD [7]. A link has been sought between diabetes of various types and the incidence of obesity in pre-pregnant women and perinatal periods [8]. GDM is a disorder of glucose tolerance that manifests itself during pregnancy. It is estimated to be one of the more common complications of pregnancy and a cause of fetal developmental disorders. The causes of GDM are attributed to an abnormal tissue response to insulin, resulting in increased synthetic hormone. Factors causing the body’s improper use of glucose are hormones produced by the placenta, which exhibit antagonistic effects to insulin. This is mainly contributed by lactogen, growth hormone, prolactin, corticotropin-releasing hormone, and progesterone. GDM develops in the second and third trimesters of pregnancy. Complications of pregnancy associated with GDM include intrauterine death, preterm delivery, fetal hypoxia, and malformations. Risk factors for GDM include excessive body mass index (BMI), physical inactivity, family history of diabetes, history of GDM, metabolic diseases, low levels of high-density lipoprotein, and polycystic ovary syndrome [9].

This article focuses on the search for data on the possible effect of diabetes and obesity in the mother on the development of ASD in the offspring. We reviewed the current state of knowledge, based on the available literature on the subject. We analyzed articles published in English from the years 2014 to 2024. The evidence in the articles suggests the susceptibility of the offspring to the influence of maternal carbohydrate metabolism during pregnancy in the context of the development of ASD. We attempted to classify articles according to the type of metabolic disorder described. Studies of the effect of maternal diabetes on autism in offspring have yielded inconsistent results. A systematic literature review and meta-analysis in 2018 confirmed the risk of autism in children. Based on case-control studies, it was calculated that mothers with diabetes have a 62% higher risk of ASD in their children than mothers without diabetes [10].

This article aims to review the association between maternal obesity, diabetes during pregnancy, and childhood ASD.

Epidemiology

According to the World Health Organization (WHO), autism affects 1 in 100 children. It is believed that these figures are underestimated. Studies in middle- and low-income countries are lacking. There are an estimated 52 million people worldwide with ASD. Some sources say that among North American, Europe, and Asia, 1% of the population have ASD [4].

ASD is more common in boys than in girls. In 2016, at 11 Autism and Developmental Disabilities Monitoring Network centers, 18.5 out of 1000 children aged 8 years had ASD. Boys were 4.3 times more likely than girls to develop the condition. The fewest children were identified in Colorado (13.1), while the most were identified in New Jersey (31.4). Non-Latino Caucasian, Black, and Asian/Pacific Islander children had similar results of 18.5, 18.3 and 17.9 per 1000, respectively. There were fewer cases of children of Hispanic origin having ASD, at 15.4 per 1000 [11]. Numerous studies worldwide indicate an increased risk of ASD in boys [4,12-15]. It has been calculated that in the United States the prevalence of ASD for children 8 years old increased by 63.7% from 2008 to 2016. In the same group, it was observed that per 1000 children in 2000, ASD affected 10.3 boys and 2.9 girls, and in 2016, 29.7 boys and 6.9 girls. Over 16 years, the ratio by sex of 3.6:1 for boys increased to 4.3:1 [16]. The exception is a study in Taiwan assessing that...
the risk for autism was generally higher for girls. However, it increased for boys born by cesarean delivery under general anesthesia [17]. A screening study from China conducted among children aged 6 to 12 years included a group of 140,000, of whom 363 children were confirmed to have ASD based on the DSM-5. The prevalence rate in the entire population was 0.29%, implying an estimated prevalence of 0.70% [15].

A summary of studies published in 2014 yielded the following data: in Canada, according to various sources, 10 to 16.2 children per 1000 population aged 6 to 9 years were diagnosed as having ASD. In the United States, according to the DSM-IV assessment, 16.8 and, according to the DMS-V, 18.5 cases were detected, while in Mexico, 8.7 cases per 1000 children were found. In China, depending on the region and detection criteria, the values ranged from 1.9 to 26.2 per 1000 children. Data from Japan indicate 19 to 93 cases; from India, 1.5 to 5 cases; from Nepal, 3.4 cases; from Bangladesh, 0.8 cases; and from Vietnam, 10.8 cases per 1000 children. In Australia, data from national reports in 2005-2006 reported a rate of 14.1, while in 2010-2011 it was already 25.2 cases per 1000 children. In European countries, variations in the number of cases per 1000 children are as follows: Sweden, 17.4; Poland, 5.2 to 5.4; Germany 6.0; Denmark, 12.6; Finland, 7.7; France, depending on the region, 4.8 to 7.3; Iceland, 31.3; Italy, 4.2 to 11.5; and Spain, between 11.8 and 15.5 [16].

In the Middle East, the fewest cases were detected in Iran, at 1.1 per 1000 children and Oman, at 2.0. Qatar reported 11.4, and Lebanon, 15.3 cases per 1000 children [16].

A study involving 8820 children in Chandigarh, India, between the ages of 1.5 and 10 years found a prevalence of autism of 2.25 per 1000 tested, which is consistent with results across India [18].

According to a 2021 review of global epidemiological studies on autism conducted in 37 countries, the average prevalence of autism in 26 high-income countries was 0.97%. The following figures were given for European countries per 10 000 inhabitants: Iceland, 268; United Kingdom, 116; Ireland, 100; Germany, 38; Poland, 35; Sweden, 154; Norway, 42; Finland, 76; Greece, 115; Italy, 95; France, 73; and Portugal, 9. In the United States, the prevalence of ASD was estimated at 185, in Canada at 152, and in Mexico at 87 per 10 000 people. Data from China is 70; Taiwan, 20; Indonesia, 12; Bangladesh, 8; India, 111; Oman, 20; Iran, 18; Israel, 76; and Qatar, 114 per 10 000 population [19].

### Maternal Obesity During Pregnancy as a Risk Factor for ASD in Offspring

Obesity in mothers during pregnancy can generate many complications. The most common of these is GDM, which is caused by insulin resistance related to hormonal balance during pregnancy. In addition, there is also a higher incidence of pre-eclampsia, gestational hypertension, macrosomia, venous thromboembolism, spontaneous miscarriage, and thrombosis. Obesity increases the risk of perinatal complications, the need for cesarean delivery, and the occurrence of epidural failure. Accompanying insulin resistance, inflammation, and oxidative stress in mothers with obesity can lead to placental dysfunction and negatively affect the fetus. There is a risk of spina bifida, umbilical hernia, and heart defects. Attention is also drawn to the long-term effects of obesity in the form of metabolic disorders and obesity in the offspring [20].

The mechanisms linking ASD to obesity are not yet understood. It has been suggested that they may be related to chronic inflammation and the accompanying increased expression of cytokines [21]. Human and animal experiments indicate that maternal obesity negatively affects the development of the sympathetic nervous system in offspring [22]. Excessive gestational weight gain has been found to affect the risk of ASD in offspring, but results have been inconsistent [23]. Therefore, studies have looked for an association between parental BMI and these disorders [24]. Available studies looking for an association of excessive gestational weight gain in the mother linked this phenomenon to an increased risk of ASD in the offspring. Studies indicate that low gestational weight gain in the mother is not associated with an increased risk of ASD. Some studies evaluating the association of maternal BMI or weight before pregnancy with ASD risk have confirmed that there does not appear to be an association [25].

The search for a link between gestational weight gain and the onset of autism in offspring points to the involvement of leptin. Too high levels of this hormone can cause placental dysfunction. Studies in women with different BMIs before pregnancy have shown different plasma leptin levels. It has been suggested that elevated leptin levels can cause excessive weight gain during pregnancy. Excessive gestational weight gain did not increase the risk of autism in mothers with underweight or normal weight. The study found that being overweight or overweight before pregnancy may not be associated with autism risk in offspring [26].

Another study showed that the mother’s total gestational weight gain was higher in the group with ASD. Inadequate gestational weight gain was not associated with ASD risk, maternal overweight or obesity before pregnancy was not associated with ASD, and there was little association of obesity with ASD. The association of gestational weight gain with ASD seemed stronger among mothers who were overweight or obese before pregnancy [27].

A Spanish study of 1827 preschool-aged children found that parental BMI was not associated with autism symptoms. The
percentages of mothers and fathers with obesity were 8% and 12%, respectively. Maternal and paternal obesity was associated with an increase in symptoms related to attention deficit hyperactivity disorder (ADHD). Maternal obesity before pregnancy was associated with decreased verbal performance in preschool-aged offspring, but was not associated with ASD risk in offspring [28].

An analysis in Finland found that mothers without diabetes with an elevated BMI (moderate to severe obesity) before pregnancy had a risk of multiple disorders in their offspring, such as obesity, ADHD, and intellectual disability. No association of maternal obesity with ASD was found in this case either [29]. Other data show that maternal obesity before pregnancy was associated with a statistically significantly increased chance of having a child with ASD [30]. Another study included 1118 children with ASD and 606 with ASD and ADHD, finding an increased risk of ASD in children of mother with underweight or obesity. The risk of having a child with ADHD and ASD was elevated for mothers with obesity and severe obesity [31].

In another case, the association between maternal BMI and the incidence of ASD was nonlinear. It has been suggested that extreme values of maternal BMI may be associated with a moderate increase in the risk of ASD in offspring. A relationship was found between maternal BMI and ASD risk according to maternal age. An excessively low BMI generated ASD risk among mothers over 30 years of age. Maternal obesity before pregnancy was associated with ASD risk in offspring regardless of age [32].

An analysis of a Danish birth cohort showed that the incidence of ASD was associated with maternal underweight or obesity. In contrast, the incidence of ASD combined with ADHD was increased in overweight women and increased with the degree of obesity, compared with that of mothers with a normal BMI [33].

Overweight and obesity appear to be associated with a variety of neurodevelopmental disorders in offspring [34]. Swedish results at the population level showed that maternal BMI was associated with ASD. However, sibling and paternal BMI analyses challenged this conclusion, as maternal BMI may be a surrogate marker for other familial risk factors. The study points to excessive or insufficient gestational weight gain as a risk factor for ASD [35].

A woman’s obesity appears to indirectly influence autism in offspring. The mother’s weight before pregnancy was not directly related to autism in the offspring in this case. A cohort study showed that children born to mothers with obesity or underweight before pregnancy had a risk of having very low birth weight babies. Rapid growth and increasing weight after birth generated a risk of autism more than double that of children with gradual growth. The rapid increase in head circumference in these children carried a risk at 5 times that of children with normal development [36].

A woman’s pre-pregnancy BMI (ie, overweight and especially obesity) has been shown to be associated with ASD in offspring. A significant association was observed between an increase in the mother’s BMI before conception and the severity of autistic traits in the offspring in early adulthood. Mothers with a BMI of 30 kg/m² or more before pregnancy had an odds ratio of more than 2.0 per child with typical symptoms of autism [21].

**Maternal Coexistence of Diabetes and Obesity as a Risk Factor for ASD in Offspring**

Diabetes and obesity often occur simultaneously. It is estimated that in the United States, one-third of women of childbearing age have obesity, 4% to 9% have diabetes before pregnancy, and 2% to 10% develop diabetes during pregnancy [37].

It is estimated that 20% to 40% of women in developed countries have obesity before pregnancy. Abnormalities in the mother’s metabolic processes are associated with the risk of ASD in offspring. Using data from 89 children with ASD and 700 typically developing children, the association of maternal metabolic processes and branched-chain amino acids with the child’s ASD risk by sex was examined. ASD risk was higher in mothers with obesity and/or type 2 diabetes (T2DM) with elevated branched-chain amino acid levels. The risk increased even higher when the child’s sex was male [38].

Patients with a diagnosis of pregestational diabetes mellitus (PGDM) and GDM were included in a study of mother-child pairs, in which 8.8% of children with ASD had mothers with GDM, and 4.5% of children with normal development had mothers with GDM. Analysis of the birth cohort showed that the combination of obesity and diabetes in the pregnant woman was associated with an increased risk of ASD in the offspring, compared with when these conditions were present separately. An additional compounding factor for ASD in this case was the mother’s intellectual disability. In addition, ASD was more prevalent among boys, babies with premature low birth weight, and babies born by cesarean delivery. The study also confirmed a slightly increased risk of ASD associated with only maternal obesity. A slightly increased risk of ASD associated with only maternal diabetes was also indicated [37].

A nationwide study in Finland showed that PGDM treated with insulin combined with severe obesity significantly increased the risk of ASD. Out of 649 043 births, 20.7% of women had overweight, 7.67% had obesity, and 3.66% had severe obesity.
PGDM affected 0.63%, while GDM affected 15.7%. Women who had PGDM and severe obesity had a 6-fold higher risk of giving birth to a child with ASD than did mothers without diabetes and mothers of normal weight. This study did not find a significant relationship between GDM and ASD. However, other studies indicate such a relationship in mothers with obesity and severe obesity, which may depend on the diagnostic criteria, screening procedures, and treatment guidelines for GDM.

Maternal obesity before pregnancy has been shown to slightly increase the risk of psychiatric disorders in the child [39].

A cohort study conducted in Finland from the Drugs and Pregnancy database on 6 470 099 mother-child pairs analyzed the relationship of the incidence of psychiatric disorders in children of mothers according to the type of diabetes and obesity. They identified 4000 mothers (0.62%) with fetuses exposed to pre-pregnancy type 1 diabetes (T1DM) treated with insulin, 3724 mothers with T2DM (0.57%), and 98 242 (15.18%) mothers with GDM. Severe obesity affected 3.7% of cases, and moderate obesity affected 7.7%. The mental health of children up to age 11 was observed, and 2346 (0.36%) cases of ASD were identified. It was calculated that among offspring with detected disorders caused by the mother’s diabetes, 76% had behavioral and emotional disorders, 61% had ADHD, 57% had autism, and 55.5% had sleep disorders. The disorders occurred simultaneously in many cases [29].

A study showed that children of mothers with obesity with BMI ≥30 kg/m² had the highest probability of ASD, and 29% of these children had a diagnosis of ASD alone, while 33% of the children had a diagnosis of ASD and gastrointestinal disturbances. In mothers with diabetes, 8.8% of children had ASD, and 10% had ASD and gastrointestinal disturbances. It should be added that among mothers with obesity, 14% had diabetes at the same time (14%), 11% had diabetes and asthma, and 8.9% had pre-eclampsia, which affected the obtained results [40]. Another study indicated that maternal obesity and PGDM were linked with the risk of ASD in offspring. Mothers with obesity and GDM also had a significantly increased risk of autism in their children [41].

Children of mothers with severe obesity with T2DM were less likely to have ASD than were children of women with T1DM treated with insulin in pre-pregnancy. In contrast, mothers with severe obesity with T2DM had a higher risk of ill offspring than did women with a similar BMI with gestational diabetes. Diabetes in normal-weight mothers carried no risk of psychiatric disorders in offspring [29].

GDM as a Risk Factor for ASD in Offspring

The incidence of GDM has increased significantly in recent years [42,43]. The incidence of ASD appears to be more frequent in the offspring of mothers with GDM, compared with the general population [42]. Incidence rates for the condition oscillate between 1.7% and 15.7%, depending on ethnicity, maternal age, and diagnostic criteria used [44]. According to a study, 8% of children with autism had mothers with GDM, compared with 2% in the control group [42]. In contrast, data from Israel indicate that 0.04% of mothers with GDM gave birth to children with ASD, with 0.01% of children of non-diabetic mothers having ASD [7]. Based on clinical data from Qatar from 176 960 children, including 844 with ASD aged 5 to 12 years, it was found that 8.9% of autistic children were exposed to maternal GDM [45]. The prevalence of prenatal factors that may contribute to the development of ASD in Taiwan, including GDM, was investigated among 323 children with ASD and their siblings (257 children) without the disorder and 1504 children of typically developing controls. It is suggested that 1.24% of children with ASD had mothers with GDM, while 0.4% of healthy siblings had mothers with GDM. In the control group, 0.7% of the children had mothers with GDM [46]. According to data from Japan, 3% of those in the ASD group were exposed to GDM, compared with 1.3% of the control group [47].

A retrospective study conducted in California on the impact of fetal exposure to previously detected T2DM and gestational diabetes diagnosed at 26 weeks of gestation included 322 323 singleton births at 28 to 44 weeks of gestation. Among them, 3388 children with ASD were identified [48]. It was shown that GDM detected in the mother at 26 weeks of gestation was significantly associated with the risk of ASD in the offspring, with an increase of 42%. Comparing GDM detected at 26 weeks of gestation with T2DM, GDM generated a higher risk of symptoms characteristic of ASD [48].

A study using a questionnaire completed by trained interviewers in Bangladesh of 385 children aged 2 to 18 years in 22 schools for children with autism confirmed the association of specific diseases in pregnancy, such as diabetes, on the incidence of autism in children [49].

One study showed that GDM was positively associated with the birth of a child with ASD, compared with children with either disabilities or developmental disorder and the control group. The highest risk, more than double that of the control group, was detected in mothers with obesity and GDM [30].

Another study also confirmed the association of GDM exposure to the fetus with ASD risk. The study showed that the incidence of ASD in children of mothers with GDM was higher than in the population of Polish children aged 0 to 18 years, at 8 in 1007 children, or 0.8% [50].

Literature is also available that questions the effect of gestational diabetes on the risk of ASD in offspring. A Spanish study...
on 36 ASD cases found that prenatal exposure to GDM was not associated with an increased risk of ASD, compared with uncomplicated pregnancies [51]. In contrast, another study found no statistically significant risk of ASD in cases of GDM, particularly those diagnosed after 26 weeks of gestation not requiring treatment, or GDM unrelated to obesity [52]. Another study found that GDM diagnosed after 26 weeks of gestation was not associated with a higher risk than was no diabetes [53].

Children aged 2 to 5 years were studied in 3 subgroups: those with ASD, those with ASD associated with delayed development, and healthy children from the general population. The study included 2564 mothers. A total of 246 (10%) had diabetes during pregnancy, 65 (3%) had diabetes before pregnancy, and 181 (7%) had GDM. The analysis showed that diabetes of any type was not associated with ASD but was associated with developmental delays unrelated to autism. The association of maternal BMI and diabetes in relation to ASD was also not confirmed. A total of 386 (15.1%) mothers had any hypertension during pregnancy and it was associated with ASD risk [54].

### T1DM as a Risk Factor for ASD in Offspring

T1DM or T2DM is estimated to affect 1% of pregnant women, and the incidence continues to rise, with indications that the risk of ASD is higher with T1DM [44,52,55].

T1DM in pregnant women increases the risk of preterm birth. A study was conducted on 1.5 million Swedish children. Of these, 24941 had a diagnosis of ASD, 8003 children were born to mothers with T1DM, and 81915 were born prematurely. It was estimated that about 22% of the total risk of ASD in offspring was due to preterm birth. The risk of ASD in offspring of mothers with T1DM diagnosed before conception was 140-fold higher than that of healthy mothers. The study did not confirm that increasing glycated hemoglobin levels before or during pregnancy could further increase the risk of ASD in offspring [55].

Of 419423 children diagnosed, 621 of their mothers had T1DM, 9453 had T2DM, 11922 had GDM diagnosed at 26 weeks of gestation, and 24505 had GDM diagnosed after 26 weeks of gestation. A total of 5827 children were diagnosed with ASD, which was observed from age 6 to 9 years. The unadjusted annual incidence rates per 1000 people for each type of diabetes were as follows: T1DM, 4.4; T2DM, 3.6; GDM up to 26 weeks, 2.9; and GDM after 26 weeks, 2.1 [53].

A study in Taiwan included 877233 children, of whom 338 were exposed to T1DM, 8749 to T2DM, and 90200 to GDM in fetal life. The most significant impact on the development of ASD in a child was T1DM, followed by T2DM, and the least significant was GDM [56].

The results of another large study conducted in Sweden seem to contradict the above reports. The study compared the risk of ASD with T1DM, T2DM, and GDM in the mothers during the prenatal period, as well as in the father. Each type of diabetes was associated with an increased risk of ASD. The highest risk of ASD in offspring was carried by prenatal exposure to T2DM diabetes in 3.2%, T1DM in 2.9%, GDM in 2.8%, non-specified PGDM in 2.5% [57].

### Diabetes as a Risk Factor for ASD in Offspring

Diabetogenic hormones increase during pregnancy, resulting in persistent relative insulin resistance in the mother. Elevated hormone levels persist until delivery. Such a condition increases the risk of poor glycemic control and the onset of hyperglycemia or gestational diabetes [58].

The occurrence of neurodevelopmental disorders has been shown to be associated with exposure in utero to each type of diabetes, with the association being stronger for pre-pregnancy diabetes (T1DM and T2DM) than for GDM [52].

Diabetes that occurs in women before pregnancy (PGDM) adversely affects the fetus and the course of pregnancy [44]. The greatest risk of a variety of disorders in the offspring, including ASD, is carried by diabetes diagnosed in the mother before pregnancy of T1DM, followed by T2DM, and least of all GDM in mothers with obesity [29].

A case-control study conducted in Egypt among students at 1 school confirmed diabetes history in the mother as a factor for ASD in offspring [59]. A 2015 meta-analysis indicated that the risk of ASD in offspring can vary depending on the type of diabetes. Numerous studies have noted that the risk of ASD in offspring of mothers with GDM is lower than that of mothers with PGDM. Studies also indicate that offspring of mothers with GDM and PGDM, compared with controls, have reduced cognitive abilities, poorer fine and gross motor skills, and reduced activity levels [60].

### Future Directions

The cited literature clearly indicates the large role of diabetes and obesity in the mother for the risk of ASD in the offspring. There is presently a search for the (molecular) mechanisms that cause neuronal changes in the fetus that determine the normal development of the child’s brain and possible causes of autism. It seems that the role of abnormal glucose levels significantly affects neuronal migration, among other things.
Abnormal neuronal migration and lack of neuronal connectivity are observed in individuals with ASD. The involvement of glucose and insulin in catalyzing these processes is sought. It is reported that even transient hyperglycemia can affect epigenetic changes and cause neuronal dysfunction. Substances that interfere with neuronal migration or cause epigenetic changes can be glycation end products, inhibiting Ras-related C3 botulinum toxin substrate 1 (Rac1) activation and gua- sine triphosphatase [61,62]. The contribution of relin, alpha-2-macroglobulin levels and plasminogen activator inhibitor to impaired neuronal connectivity has also been implicated [61].

There is a need to study prenatal and neonatal risk factors, as well as to create uniform assessment and surveillance systems and guidelines to monitor the phenomenon, which will allow reliable comparison of data from different regions of the world [4,16,61].

Recent decades have seen a sharp increase in autism cases in the population. Researchers are trying to explain the reasons for this. The growing number of people with ASD poses both economic and health challenges for societies. When assessing the scale of the problem globally, obstacles are encountered in the form of inconsistent methodological criteria and qualification procedures or case definitions. The assessment of individual cases may be affected by autism awareness resulting also from socioeconomic, ethnic, racial, and cultural disparities. Prevalence data depend on geographic areas, countries, and source of information [16].

The increase in the prevalence of ASD is commonly attributed to changing diagnostic criteria for ASD. The most recent manual on clinical diagnostic criteria is the fifth edition of the Diagnostic and Statistical Manual (DSM-5), which was published in 2013. The use of these revised diagnostic criteria may have an impact on the prevalence of ASD [63].

There are doubts as to whether the 2000-2014 data from the Centers for Disease Control and Prevention, which reported a 2.5-fold increase in the incidence of ASD, should be attributed to changes in diagnostic tools and more screening or a true increase in the incidence [64].

Conclusions

Different types of diabetes and the severity of obesity appear to play a significant role in the pathogenesis of ASD. There is a need for the screening of pregnant women and those planning to conceive, as well as prenatal monitoring of carbohydrate levels and hormonal levels. There is a need to verify the causes of the increase in the incidence of ASD. There is a need to standardize the criteria for the diagnosis of ASD and make it accessible in all regions of the world. The increase in the number of obese and/or diabetic pregnant women is not only a challenge for modern medicine, but also an economic challenge. Children of women with carbohydrate metabolism disorders are at risk of neural complications, which may underlie the development of ASD. There is a lack of research that clarifies unequivocally which type of diabetes poses the greatest risk to the brain of the developing fetus. There is a lack of clear information on what other perinatal and prenatal factors, as well as social and economic factors, generate the risk of ASD in the offspring of women with coexisting diabetes and/or obesity.

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