

## THE INFLUENCE OF COMPONENTS OF DIET ON THE SYMPTOMS OF ADHD IN CHILDREN

### WPŁYW SKŁADNIKÓW DIETY NA OBJAWY ADHD U DZIECI

Klaudia Konikowska, Bożena Regulska-Ilow, Dorota Różańska

Department of Dietetics, Wrocław Medical University, Poland

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#### STRESZCZENIE

U większości dzieci przyczyna występowania ADHD nie jest do końca znana, a jego etiologia jest wieloaspektowa. Konwencjonalne leczenie tego schorzenia oparte jest na połączeniu terapii behawioralnej, psychologicznej oraz farmakoterapii. Farmakoterapia wykazuje wysoką skuteczność w leczeniu ADHD, jednak często wiąże się ona z występowaniem niepożądanych efektów ubocznych, takich jak: brak apetytu, utrata masy ciała, zahamowanie wzrostu, bóle brzucha, bóle głowy, problemy z zasypianiem, wzrost ciśnienia krwi. W ostatnich latach można zauważać wzrastające zainteresowanie zastosowaniem dietoterapii w leczeniu ADHD, zwłaszcza kiedy standardowa farmakoterapia nie przynosi poprawy stanu zdrowia. Zarówno dieta kobiety ciążarnej, karmiącej, jak i dziecka może mieć wpływ na rozwój i pogłębianie objawów zespołu hiperkinetycznego. Istnieje wiele dowodów, które wskazują, że ADHD jest związany z czynnikami żywieniowymi. Przewlekłe niedobory niektórych składników mineralnych, takich jak: cynk, żelazo, magnez, jod mogą mieć znaczący wpływ na rozwój i pogłębianie objawów ADHD u dzieci. Szczególną rolę w diecie kobiety w ciąży, karmiącej oraz dziecka odgrywają także kwasy tłuszczyzny omega-3, a głównie DHA, które są niezbędne do prawidłowego rozwoju i czynności mózgu u dziecka. Ich przewlekły niedobór może przyczyniać się do zwiększonego ryzyka występowania ADHD u dzieci. Autorzy kilku badań wykazali korzystny wpływ eliminacji produktów spożywczych zawierających syntetyczne dodatki do żywności, m.in. barwniki i konserwanty na zachowanie u dzieci z ADHD. Pozytywne efekty przyniosła także eliminacja z diety produktów będących bogatym źródłem salicylanów. Zaobserwowano ponadto, że spożycie produktów z niskim indeksem glikemicznym przyczynia się do zmniejszenia objawów choroby u niektórych osób nadaktywnych. Dostarczenie odpowiedniej podaży składników odżywcznych i mineralnych oraz ewentualne wyeliminowanie niektórych produktów spożywczych z diety jest szczególnie istotne w okresie rozwoju i intensywnego wzrostu dziecka. Omówiono zagadnienia dotyczące roli diety w leczeniu dzieci, u których rozpoznano ADHD oraz znaczenia niedoborów niektórych składników mineralnych i dugołańcuchowych wielonienasyconych kwasów tłuszczyzny w przebiegu ciąży i rozwoju dziecka. Istnieje potrzeba dalszych badań w tym zakresie, w celu oceny potencjalnej skuteczności diety w leczeniu objawów ADHD.

#### ABSTRACT

In most children with ADHD the cause of the disease is not exactly known, and its etiology is multifactorial. The conventional treatment is based on the combination of behavioral and psychological therapy and the pharmacotherapy. The pharmacotherapy has a high effectiveness in ADHD treatment, but it is often associated with undesirable side effects, such as: loss of appetite and weight, growth inhibition, abdominal pain, headaches, sleeping problems and increased blood pressure. In the recent years, much attention was devoted to the issue of an appropriate diet in this disease, especially when the standard pharmacotherapy is not effective. The diet of pregnant and lactating woman, and child may have an impact on the development and deepening of the hyperkinetic syndrome. There is much evidence to indicate that it is linked to nutritional factors. Chronic deficiencies of certain minerals such as zinc, iron, magnesium and iodine and insufficient dietary intake of long-chain polyunsaturated fatty acids may have a significant impact on the development and deepening of the symptoms of ADHD in children. A crucial role in the diet of pregnant and lactating women, and child plays also polyunsaturated *omega-3* fatty acids, mainly DHA, which are necessary for proper development and function of brain. Their chronic deficiency may contribute to increase risk of ADHD in children. The authors of several studies also demonstrated the positive impact of the elimination food products containing synthetic food additives, like artificial food dyes and preservatives on the behavior of

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**Correspondence address:** Bożena Regulska-Ilow, Zakład Dietetyki, Wrocławski Uniwersytet Medyczny, 51-616 Wrocław, ul. Parkowa 34, tel. +48 71 348-25-70, e-mail: bozena.regulska-ilow@am.wroc.pl

children with ADHD. The beneficial effects brought also the elimination of food products, that are rich in salicylates. It was found that the intake of food products with a low glycemic index helps to reduce symptoms in some hyperactive children. Providing an appropriate supply of nutrients and minerals and elimination of certain food products from diet is especially important during intensive growth and development of the child. In this article the issues concerning the role of the diet in treatment of the children with diagnosed ADHD and the importance of deficiency of certain minerals and long-chain polyunsaturated fatty acids in pregnancy and child growth was discussed. There is a need for further studies in this area to evaluate the potential effectiveness of the diet in treating the symptoms of ADHD.

## ETIOLOGY OF ADHD

ADHD (*Attention Deficit Hyperactivity Disorder*) is one of the most frequently neurobehavioral disorder diagnosed among children in school-age. It affects about 5-10% of the population of children in school-age. The occurrence of ADHD is significantly more frequent among boys than girls [14]. ADHD is characterized by severe attention deficit disorder, excessive motor activity and problems in learning. The disease manifests itself also by hyperactivity, impulsiveness and distraction. Children with ADHD may be also impatient. They easily distracted under the influence of external stimuli and they do not discharge the commands properly.

The etiology of ADHD is not exactly known. The latest researches show the multifaceted causes of the disease. The risk of ADHD is associated with the genetic, environmental and psychosocial factors [32].

Environmental factors contribute significantly to development of ADHD. Complications during pregnancy and delivery have an especially important meaning. The increased risk of ADHD during the prenatal period is connected with environment pollution and intake by a mother the food products containing preservatives and contaminated with pesticides [32]. The disease occurred more frequently in those children, whose mothers smoked cigarettes and abused alcohol during pregnancy [27]. During the delivery there may become complications, such as: hypoxia and child's brain ischemia, what can influence on the modification of gene expression. After the child birth the risk factors of ADHD are: hypoxia, fever, exposure to certain chemicals such as lead and mercury, and organic pollutants [32].

Nutritional factors like: the synthetic food dyes, preservatives, refined sugars, insufficient intake of long-chain polyunsaturated fatty acids and some minerals (iron, zinc, magnesium and iodine) are positively correlated with the symptoms of ADHD among some children. Recently an increasing interest in this issues was observed.

The influence of nutritional factors on the symptoms of ADHD was discussed based on a review of the publications.

## TREATMENT IN THE ATTENTION DEFICIT HYPERACTIVITY DISORDER

Hyperactivity disorder is a genetic trait connected with an abnormal brain maturation. The imaging studies among children with ADHD showed abnormalities in the prefrontal cortex, basal ganglia and cerebellum, which were the causes of executive dysfunction. According to Shaw et al. [35] ADHD does not show a deviation from typical development of the cerebral cortex but its delayed development. The most probably is, that among people with this disorder occur weakening of activity of neurotransmitter dopamine and norepinephrine, which are involved in the transmission of the impulses in the nervous system [8].

The conventional treatment is based on the combination of behavioral and psychological therapy and the pharmacotherapy, which are often associated with a long-term use of stimulants such as methylphenidate, dexamphetamine and their derivatives [8, 16]. These medicines increase the activity of dopamine and norepinephrine. The stimulants have a high effectiveness in ADHD treatment. However, during the pharmacotherapy often occur undesirable side effects, such as: loss of appetite and weight, growth inhibition, abdominal pain, headaches, sleeping problems and increased blood pressure. Moreover, most of the commonly used medications, which stimulate nerve conduction may lead to addiction [16]. Nowadays, there is a need to search for an alternative tools in ADHD treatment, because in the therapists opinion the medicaments do not bring the intended effects.

Results of the studies show, that modification of the diet composition may have an influence on increasing as well as reducing the symptoms of ADHD, therefore proper diet may be a support for pharmacotherapy. It was observed that hyperactivity and lack of concentration among people with ADHD is positively correlated with the iron, zinc and magnesium deficiency in the diet. It seems that hyperactive behavior in some children can be reduced by supplements intake [9, 23, 24].

## DIET ELIMINATING FOOD ADDITIVES

It was observed that there is a relationship between some food additives and development of ADHD among

children. Although so far there is no clear evidence for the existence of this correlation and it is not clear, whether concerns to all patients, findings of some authors are promising [4, 13, 15].

*Feingold* [15] has published in the 70's the researches, in which he observed the negative effects of artificial food dyes and salicylates on children's behavior. It was the first important trial to apply dietary management in the treatment of ADHD. The author observed improvement in the treatment in more than 50% of children with hyperactivity after the elimination from diet all artificial food dyes and products with naturally occurring salicylates. Modification of the diet made by *Feingold* [15] has concerned the elimination of processed food, products containing artificial food dyes, flavors, sweeteners, preservatives and exclusion many of fruits and vegetables due to the presence of salicylates. The results of further studies on the *Feingold* diet were too ambiguous to confirm its effectiveness.

*Bateman* et al. [4] showed a relationship between intake of preservatives and food dyes and increasing the symptoms of ADHD. The study was conducted for 4 weeks and included 277 children. Some of the children had hyperactivity and the others did not have the symptoms of ADHD. In the first week of the study the artificial food dyes and sodium benzoate were excluded from the diet. Fruit juices with artificial food dyes and sodium benzoate or placebo were given to children in the second and fourth week of the study. Juices contained 20 mg of artificial dyes (sunset yellow, tartrazine, carmoisine and ponceau 4R) and 45 mg of sodium benzoate. Parents observed reduce hyperactivity in that time, when food additives were excluded from diet. They observed also increase hyperactivity in the group, which consumed juices with artificial food dyes and sodium benzoate compared with the control group (according to APHR – aggregated parental hyperactivity ratings). However, clinical studies have not confirmed the subjective assessment of parents (according to ATH - aggregated test hyperactivity). Nevertheless, findings of the study would suggest that artificial food dyes and preservatives may increase hyperactive behavior among some children.

Observations of *Dengate* and *Ruben* [13] confirmed the influence of food additives on the behavior of children with ADHD. The calcium propionate (E 282), which is commonly used in the bread production to prevent mould and bacterial growth, was used in the study. The children were on the elimination diet (without products which contained food additives, natural salicylates, amines and glutamates) for 3 weeks. The diet included pears, selected vegetables, meat, poultry, fish, oils, margarine, eggs, oats, rice, wheat, some processed breakfast cereals and snacks. Selected dairy products and sugar were not excluded from the diet. After the first

stage of the study on this diet, four slices of bread with the calcium propionate were given to children from the study group. Bread without preservative was given to children from the control group. The significant difference between children, whose behavior has deteriorated during the study (52%) compared with those, whose behavior has improved (19%) was observed relative to placebo. The authors observed that symptoms of ADHD, such as: irritability, restlessness, inattention and sleep disturbance among some children may be caused by daily intake of bread with preservative. They suggested that the addition of preservatives to food should be minimized, to reduce their adverse effects on health.

Since 2008 in the European Union the food products, which contain artificial food dyes (sunset yellow, quinoline yellow, azorubine, allura red, tartrazine and ponceau 4R) should have a following information on the label: "can have a detrimental effect on activity and focusing of attention in children" [31]. In 2009 the British government requested that food producers remove the most detrimental artificial dyes from food products. These actions can have a positive influence on the reduction symptoms of ADHD in some patients [21].

## REDUCTION OF SUGAR INTAKE

Role of sugar in the diet of people with Attention Deficit Hyperactivity Disorder is the following problem considered by researchers. Authors of the studies conducted in the 80's proved, the adverse influence of refined sugar on the behavior in children [6, 17]. Very small number of children was involved in these researches, and therefore the results have a limited value and cannot be applied to a wider group. In *Goldman*'s et al. [17] study participated only 8 children. In the research of *Behar* et al. [5] the study group included 21 patients.

*Wolraich* et al. [39], in a double-blind controlled trial, did not observe the influence of dietary sucrose and the sweetener aspartame on behavior or cognitive function in the study group. Children, who participated in the study, were divided into 3 groups and each group received different sweetener in a diet for 3 weeks. One diet was high in sucrose with no artificial sweeteners, second was low in sucrose and contained aspartame, and the third was low in sucrose and contained saccharin as a placebo. Others food additives, for example artificial dyes or preservatives, were also excluded from each diet. The authors did not observe the significant differences in the cognitive impairment or in behavior changes between 3 groups, receiving different sweeteners [39].

*Ludwig*, the pediatrician from the Boston hospital, observed that components of children's breakfast can

have an influence on their behavior [33]. The child, whose breakfast included high glycemic index products, would be distracted and restless at school, and would be less attentive during the lessons. These observations were confirmed by the research of Benton et al. [7], in which 19 children aged 6 – 7 years were included. The children were divided into 3 groups and they had been receiving breakfast with low, medium and high glycemic load. Better results in memory tests after 2-3 hours after breakfast were observed among those children, who received products with low glycemic index compared to other groups. Children from the first group were also less frustrated, and they needed less time for solving tasks.

### N-3 POLYUSATURATED FATTY ACIDS

Deficiency of polyunsaturated fatty acids (PUFA) in diet and the human body contribute, for example to dysfunction of nervous system and brain functioning. N-6 and n-3 long-chain polyunsaturated fatty acids (LC-PUFA) are precursors of eicosanoids like: prostaglandins, leukotrienes and thromboxanes, hormones which are responsible for the regulation of many function in the human organism.

The cell membranes of the central nervous system contain a little amount of linoleic acid (LA) and *alpha*-linolenic acid (ALA), but prevail their long-chain derivatives. Docosahexaenoic acid (DHA) accounts for 40% of LC-PUFA in the brain and for 60% of LC-PUFA in the retina [36]. There are evidences that essential fatty acids play an important role in brain functioning. Experimental studies in rats showed, that ALA deficiency in diet, decreases dopamine neurotransmission in brain [42].

Findings of many studies show, that children with Attention Deficit Hyperactivity Disorder have a low level of n-3 PUFA in organism [18, 20]. Deficiency of eicosapentaenoic acid (EPA) in children with ADHD contributes to: thirst, frequent urination, dry skin and hair and brittleness of nails [12]. Chronic deficiency of DHA in children may lead to occur Zellweger syndrome, peroxisomal disorders or mental retardation [18]. DHA may have a particular impact on the construction and functioning of the cell membranes in central nervous system and retina in children [2].

N-3 and n-6 PUFA cannot be synthesized *de novo* in human organism. They can only be reconstructed by elongation of the carbon chain and insertion the additional double bonds. This is the reason, why daily diet should provide ALA and LA, which are the precursors of n-3 and n-6 LC-PUFA, and which are called the essential fatty acids (EFA).

The sources of ALA (belonging to the n-3 fatty acids) are: flaxseed oil, cold-pressed rapeseed oil, flax and rape seeds, wheat germ and walnuts. DHA and EPA in the human organism are synthesized from *alpha*-linolenic acid. DHA and EPA are included in some oily fish, like: mackerel, halibut, salmon, sardines and herring. DHA, which is included in mother's milk, is the essential fatty acid for infants and young children. The products rich in linoleic acid, which belongs to the n-6 fatty acid, are: sunflower, pumpkin and sesame seeds, cold-pressed soybean, corn and grapeseed oil and most of the nuts [25].

In the clinical studies, where the *Teller* test was used, it was shown, that children fed with milk supplemented with LC-PUFA are better psychomotor developed and have better visual acuity. Increased supply of their precursor - ALA did not improve the function of the eye. It's indicates the need to provide the LC-PUFA in the diet [2].

Experts from the Polish Pediatric Society and the Polish Society for Atherosclerosis Research recommend to pregnant women daily intake of EPA and DHA in total amount of from 1.0 to 1.5 g [37]. Recommended 1 g of DHA and EPA, provide fresh fish, for example: 35.0 g of salmon, 42.3 g of rainbow trout and 57.0 g of mackerel, what was calculated using the Food Composition Tables [26]. According to the Polish Pediatric Society and the Polish Society for Atherosclerosis Research, tuna and salmon should not be consumed more frequent than once a week, because of the fish contamination by mercury and/or dioxins. Supplementation of EPA and DHA is recommended for pregnant women in case when there are deficiencies of these compounds [37]. Food and Drug Administration (FDA) also advise caution in fish and seafood intake during pregnancy. Weekly intake of these products should not exceed 340 g. According to FDA eating more fish by pregnant women may lead to the accumulation of methylmercury, which negatively affects the development of the central nervous system of fetuses [38].

However *Hibbeln* et al. [18] showed, that lack of the dietary LC-PUFA, which are contained in fish and seafood, is more dangerous for the child development than the risk of exposure the child on the residual contamination (for example methylmercury) contained in these products. Among those women, who participated in the study, 12% ate no fish during pregnancy, 65% ate from 1 to 340 g fish per week and 23% ate more than 340 g fish per week. The mothers after delivery responded to questions related to the development and behavior of their children at ages 6, 18, 30, 42 and 81 months. Intake of less than 340 g of fish and seafood per week by pregnant women was associated with higher risk of lower level of intelligence (assessed by *Wechsler* intelligence scale) in their children, compared to those

children, whose mothers consumed more than 340 g of these products per week during pregnancy.

Results of the studies did not prove, that intake more than 3 portions of fish per week during the pregnancy may have a negative influence on child development and behavior in the future. However it was showed, that intake more than 340 g of fish per week had a positive effects on the neurological child development.

*Joshi et al.* [20] showed a beneficial impact of dietary supplementation with linseed oil rich in ALA (a precursor of EPA and DHA) and the vitamin C on the behavior of children with ADHD. Supplementation of linseed oil (equivalent to 200 mg of ALA) and vitamin C in the amount of 2x25 mg was inducted to reduce lipid peroxidation of cell membranes. After 3 months of the study the level of EPA increased on 53% and DHA increased on 30% among children with ADHD, which resulted in a reduction of symptoms such as: impulsivity, restlessness, inattention and learning problems (accessed by the Conners' Parent Rating Scale, CPRS). The importance of this study was reduced because of the lack of a placebo group.

The authors of the studies suggest, that diet rich in n-3 fatty acids may alleviate the symptoms of hyperactivity in some children [18, 20].

The results of *Voight et al.* [41] study did not support the hypothesis, that PUFA influenced on the reduction of ADHD symptoms. Authors showed, that supplementation of DHA did not improve significantly the behavior of hyperactive children. The study included only those children with ADHD, who were treated pharmacologically. The children have been given 345 mg of DHA per day or placebo for 4 months. After that time, the DHA content in phospholipids among children from the study group was 2.6-fold higher than in placebo group. Despite this, there was no statistically significant improvement in behavior in children treated with DHA (according to CPRS and CBCL – Child Behavior Checklist).

## ADHD AND IRON DEFICIENCY

Maternal iron deficiency anemia is associated with an increased risk of preterm birth [34]. In addition, iron deficiency in childhood was reported to affect the development of the central nervous system, leading to mental retardation and behavioral disorders [23]. Symptoms of iron deficiency in children include: loss of appetite, apathy, shortness of breath, fatigue, weakness, irritability, pallor, short attention span and reduced ability to learn.

*Konofal et al.* [23] reported that serum ferritin levels were significantly lower in the children with ADHD than in the control group. Serum ferritin levels were found to be low in 84% of hyperactive children and 18%

of controls. Low serum ferritin levels correlated with more severe ADHD symptoms measured with Conners' Parent Rating Scale and greater cognitive deficits.

In other studies *Konofal et al.* [22] confirmed a significant association between iron deficiency, increased symptoms of ADHD and prevalence of restless legs syndrome (RLS). Serum ferritin levels, previous supplementation in infancy and familiar history of RLS were assessed in 12 ADHD plus RLS children, 10 ADHD children and 10 controls. Symptoms of ADHD were assessed using the CPRS. The authors found that ADHD symptoms were more severe in ADHD plus RLS group versus ADHD. However, this difference was no statistical significant. The mean serum ferritin levels were lower in children with ADHD plus RLS than in ADHD group, although the difference did not reach statistical significance. Children with ADHD had significantly lower mean serum ferritin levels than in controls.

Maternal nutrition plays a crucial role in influencing fetal growth and birth outcomes. Woman's diet should contain sufficient iron supply, otherwise she will not be able to accumulate adequate iron stores during the pregnancy [1]. To prevent iron deficiency pregnancy woman should eat food which contain iron or take supplementation. Food products rich in easily assimilated iron are: meat, fish and seafood. Lower iron bioavailability occurs in legumes (broad bean, peas), vegetables with green leaves (cabbage, salad), almonds, peanuts and walnuts, dried apricots, dactyls, barley, wheat bran. Vitamin C, oligosaccharides and intestinal microflora can increase the bioavailability of non-heme iron from the diet.

## ZINC IN A DIET OF CHILDREN WITH ADHD

Zinc is a cofactor of more than 100 enzymes, including delta-6 desaturase, which is important for the metabolism of essential fatty acids (EFA) and several enzymes involved in the metabolism of neurotransmitters, prostaglandins and melatonin. Zinc deficiency can lead to weakness of immune system function that probably contribute to increased susceptibility to infections. Lower zinc status is often found in pre-school children. In addition, zinc deficiency is one of many causes of slowed growth [11].

Zinc plays an important role in normal growth and sexual development. The nutritional adequacy of zinc depends on intensity of growth during childhood and adolescence. An adequate intake of zinc is the most important during the first year of life than at any other time. The recommended intake of zinc for infants (0-3 months) is 120 µg/kg of body weight/day for girls and

140 µg/kg of body weight/day for boys. Pregnant and lactating women are at increased risk of zinc deficiency. For these reasons, recommended zinc intake is higher for these groups than for non-pregnant women. Food sources of zinc are: oysters, wheat germ, liver, pumpkin seeds, sesame, low fat roast beef, dark chocolate, lamb, buckwheat, cheese, peanuts, rice and eggs [19].

Authors of some studies suggest that zinc deficiency plays a substantial role in the aetiopathogenesis of ADHD [3, 9]. In a study by *Bilici et al.* [9], 202 of the 400 children with ADHD were receiving zinc sulfate in an amount of 150 mg per day for 12 weeks. The control group constituted 198 children. Efficacy of the treatment was evaluated using the Attention Deficit Hyperactivity Disorder Scale (ADHDS), Conners Teacher Questionnaire, and DuPaul Parent Rating of ADHD. The level of the serum zinc and free fatty acids (FFA) were measured using routine biochemical and hematological tests. It was found, that zinc supplementation was significantly more effective than placebo in reducing hyperactivity, impulsivity and socialization scores in children with ADHD. However there were no positive effect on attention deficiency score. More effective treatment of zinc sulfate has been shown in patients of older age and high BMI score with low pretreatment zinc and FFA levels. This study demonstrated that zinc supplementation is well tolerated in children with ADHD. The full therapeutic response ratio in the zinc group (28.7%) were significantly higher than those of the placebo group (20.4%). The researchers suggested that the treatment of zinc in ADHD should be a support therapy as a supplementary medication rather than treatment by itself.

## MAGNESIUM AND VITAMIN B<sub>6</sub> SUPPLEMENTATION

Magnesium is an activator of about 300 enzymes. It takes a part in the metabolism of carbohydrates, nucleic acids and proteins. Mg<sup>2+</sup> stabilizes DNA structures and affects RNA transcription. For these reasons, is involved in control of some central nervous system processes.

Magnesium deficiency can cause an increased neuromuscular hyperactivity, nervousness, anxiety, insomnia, fatigue, migraine headaches, heart arrhythmias and palpitations, eyelid twitching and numbness [10]. An adequate intake of magnesium can reduce the occurrence of its deficiency. Food products rich in magnesium are: wheat bran, cocoa, sunflower, buckwheat, legumes, dark chocolate and nuts.

*Mousain-Bosc et al.* [28] reported a close relationship between magnesium deficiency and ADHD syndrome. In 30 of the 52 hyperactive children aged 0–15 years, there were low erythrocyte magnesium levels.

The patients were treated with Mg<sup>2+</sup>/vitamin B<sub>6</sub> supplementation at a dose of 6 mg/kg of body weight/day for 1 to 6 months. In all patients, symptoms of attention deficit hyperactivity disorder (physical aggressivity, instability, scholar attention, spasm, myoclonia, hypertony) were reduced after the treatment. The criteria for ADHD syndrome were evaluated using the psychometric scales in relation to children age (according to Connor's Rating Scale for Parents and Teachers).

*Nogovitsina and Levitina* also identified decreased plasma and erythrocyte magnesium levels in children with ADHD [29]. Serum magnesium values were assessed in 51 children with ADHD and 15 healthy children. Patients with ADHD had significantly lower magnesium levels in plasma and erythrocytes compared to the healthy group. The authors examined the benefits of magnesium supplementation with vitamin B<sub>6</sub>. For 30 days 31 children with ADHD had been receiving Magne-B<sub>6</sub> (lactate magnesium – 48 mg, pyridoxine chloride – 5 mg). The control group, consisted of 20 hyperactive children, had been receiving multivitamins - 1 teaspoon of syrup per day. On day 30, Magne-B<sub>6</sub> supplementation was significantly more effective than multivitamin treatment in reducing anxiety and impairment of attention and hyperactivity in the total point score according to the *Achenbach* questionnaire.

These studies suggest that magnesium deficiency often occur in children with ADHD. Mg<sup>2+</sup> and vitamin B<sub>6</sub> supplementation can restore normal serum magnesium level and decrease hyperactive behavior in children with ADHD.

## IODINE DEFICIENCY IN ADHD

An inadequate dietary supply of iodine is still a common problem all over the world. Iodine deficiency has been identified by World Health Organization as one of the major public health problem [6]. The introduction of iodine prophylaxis programs may prevent or reduce the symptoms of hypothyroidism during pregnancy and therefore minimize permanent neurological damage in offspring [30].

*Vermiglio et al.* [40] carried out a ten-year prospective study of the neuropsychological development of the offspring of 16 women with moderate iodine deficiency (area A) and of 11 women with minimally iodine-sufficient (area B). Hyperkinetic syndrome was diagnosed in 11 of 16 children from area A (68.7%), but in none from area B. Total intelligence quotient score was lower in children from area A ( $92.1 \pm 7.8$ ) compared to children from area B ( $110 \pm 10$ ). Intelligence was measured by the Wechsler Intelligence Scale for Children, 3rd edition (WISC-III). The results of these studies have shown that

iodine deficiency and hypothyroidism are both prenatal and postnatal risk factors for ADHD in children.

## CONCLUSIONS

There are many factors influencing the occurrence of hyperkinetic syndrome, including genetics, environmental and psychosocial variables. Some of the ADHD symptoms are associated with nutritional factors, such as preservatives, synthetic food dyes, refined sugars, lack of long-chain polyunsaturated fatty acids and minerals in the diet.

The occurrence of iron, zinc, magnesium, iodine and LC-PUFA deficiency in children with ADHD often correlate with inappropriate nutrition of pregnant and lactating women. In pregnancy diet an adequate supply of polyunsaturated fatty acids is especially important. Omega-3, and especially DHA are required for proper function of central nervous system in children.

This study identifies a strong association between certain minerals deficiency and the occurrence of ADHD. Deficiencies of macro- and micronutrients in the diet during pregnancy poses a risk to the health of mother and fetus. Low ferritin levels in children can have a negative impact on the development of central nervous system. This may lead to mental retardation. Children whose diet does not contain enough amounts of zinc are lower and less mentally developed. Magnesium deficiency may contribute to increased neuromuscular hyperactivity, nervousness, anxiety, insomnia and fatigue, while inadequate intake of iodine in pregnant women's diet can lead to hypothyroidism during pregnancy and permanently damage the nervous system of the fetus.

There are many controversies around the role of certain food additives and salicylates in the children's diet with ADHD. In the studies, various elimination diets were analyzed. In most of them foods containing artificial dyes and preservatives, and some vegetables and fruits in which naturally occurring salicylates were excluded. Results of research suggest that food additives and salicylates may aggravate hyperactive behavior children. It is worth to consider using an elimination diet, but only some children will respond well to this type of diet.

In conclusion, the child's diet should be balanced and completely cover the requirements for essential nutrients, vitamins and minerals. In states of deficiency increased intake of omega-3 and certain minerals supplementation, with their physician is safe and the number of adverse effects is small compared with the side effects caused by pharmacotherapy. Studies on eating habits in ADHD, although they are preliminary, show many benefits of using proper diet.

## REFERENCES

1. Abu-Saab K., Fraser D.: Maternal nutrition and birth outcomes. *Epidemiol. Rev.* 2010, 32, 5-25.
2. Achremowicz K., Szary-Sworst K.: Wielonienasycone kwasy tłuszczone czynnikiem poprawy stanu zdrowia człowieka. *Żywność. Nauka. Technologia. Jakość.* 2005, 3, 23-35.
3. Akhondzadeh S., Mohammadi M.-R., Khademi M.: Zinc sulfate as an adjunct to methylphenidate for the treatment of attention deficit hyperactivity disorder in children: A double blind and randomized trial [ISRCTN64132371]. *BMC Psychiatry.* 2004, 4, 1-6.
4. Bateman B., Warner J.O., Hutchinson E., Dean T., Rowlands P., Gant C., Grundy J., Fitzgerald C., Stevenson J.: The effects of a double blind, placebo controlled, artificial food colourings and benzoate preservative challenge on hyperactivity in a general population sample of preschool children. *Arch. Dis. Child.* 2004, 89, 506-511.
5. Behar D., Rapoport J.L., Adams A.A., Berg C.J., Cornblath, M.: Sugar challenge testing with children considered behaviorally "sugar reactive." *J. Nutr. Behav.* 1984, 1, 277-288.
6. Benoist B., Andersson M., Egli I., Takkouche B., Allen H.: Iodine status worldwide. WHO Global Database on Iodine Deficiency. Department of Nutrition for Health and Development World Health Organization. Geneva 2004.
7. Benton D., Maconie A., Williams C.: The influence of the glycaemic load of breakfast on the behaviour of children in school. *Physiol. Behav.* 2007, 92, 717-724.
8. Biederman J.: Attention-deficit/hyperactivity disorder: a selective overview. *Biol. Psychiatry.* 2005, 57, 1215-1220.
9. Bilici M., Yıldırım F., Kandil S., Bekaroğlu M., Yıldırımtı S., Değer O., Ülgen M., Yıldırın A., Aksu H.: Double-blind, placebo-controlled study of zinc sulfate in the treatment of attention deficit hyperactivity disorder. *Progr. Neuro-Psychoph.* 2004, 28, 181-190.
10. Blach J., Nowacki W., Mazur A.: Wpływ magnezu na reakcje alergiczne skóry. *Postępy Hig. Med. Dosw.* 2007, 61, 548-554.
11. Brown K.H., Peerson J.M., Rivera J., Allen L.H.: Effect of supplemental zinc on the growth and serum zinc concentrations of prepubertal children: a meta-analysis of randomized controlled trials. *Am. J. Clin. Nutr.* 2002, 75, 1062-71.
12. Chalon S.: The role of fatty acids in the treatment of ADHD. *Neuropharmacology.* 2009, 57, 636-639.
13. Dengate S., Ruben A.: Controlled trial of cumulative behavioural effects of a common bread preservative. *J. Paediatr. Child. Health.* 2002, 38, 373-376.
14. Faraone S.V., Sergeant J., Gillberg C., Biederman J.: The worldwide prevalence of ADHD: is it an American condition? *World Psychiatry* 2003, 2, 104-113.
15. Feingold B.F.: Dietary management of juvenile delinquency. *Int. J. Offender Ther.* 1979, 23, 73-84.
16. Findling R.L.: Evolution of the treatment of attention-deficit/hyperactivity disorder in children: a review. *Clin. Ther.* 2008, 30, 942-957.

17. Goldman J.A., Lerman R.H., Contois, J.H., Udall J.N.: Behavioral effects of sucrose on preschool children. *J. Abnorm. Child. Psych.* 1986, 14, 565–577.
18. Hibbeln J.R., Davis J.M., Steer C., Emmett P., Rogers I., Williams C., Golding J.: Maternal seafood consumption in pregnancy and neurodevelopmental outcomes in childhood (ALSPAC study): an observational cohort study. *Lancet.* 2007, 369, 578-585.
19. Jarosz M., Bulhak-Jachymczyk B.: Normy żywienia człowieka. Podstawy prewencji otyłości i chorób niezałącznych. Warszawa, PZWL 2008.
20. Joshi K., Lad S., Kale M., Patwardhan B., Mahadik P., Patni B., Chaudhary A., Bhave S., Pandit A.: Supplementation with flax oil and vitamin C improves the outcome of Attention Deficit Hyperactivity Disorder (ADHD). *Prostag. Leukot. Ess.* 2006, 74, 17–21.
21. Kanarek R.B.: Artificial food dyes and attention deficit hyperactivity disorder. *Nutr. Rev.* 2011, 69, 385-391.
22. Konofal E., Cortese S., Marchand M., Moren M.C., Arnulf I., Lecendreux M.: Impact of restless legs syndrome and iron deficiency on attention-deficit/hyperactivity disorder in children. *Sleep. Med.* 2007, 8, 711-715.
23. Konofal E., Lecendreux M., Arnulf I., Mouren M.C.: Iron deficiency in children with attention deficit/hyperactivity disorder. *Arch. Pediatr. Adolesc. Med.* 2004, 158, 1113–1115.
24. Konofal E., Lecendreux M., Deron J., Marchand M., Cortese S., Zaïm M., Mouren M.C., Arnulf I.: Effects of iron supplementation on attention deficit hyperactivity disorder in children. *Pediatr. Neurol.* 2008, 38, 20–26.
25. Kozłowska-Wojciechowska M., Makarewicz-Wujec M.: Dieta – nieodzowny element terapii w zespole metabolicznym. W: Mamcarz A. (red.). Zespół metaboliczny. Warszawa, Medical Education 2008, 307-318.
26. Kunachowicz H., Nadolna I., Przygoda B.: Tabele składu i wartości odżywczej żywności. Warszawa, PZWL 2001.
27. Mick E., Biederman J., Faraone S.V., Sayer J., Kleinman S.: Case-control study of attention-deficit hyperactivity disorder and maternal smoking, alcohol use, and drug use during pregnancy. *J. Am. Acad. Child. Adolsc. Psychiatry.* 2002, 41, 378–385.
28. Mousain-Bosc M., Roche M., Rapin J., Bali J.P.: Magnesium Vit B6 intake reduces central nervous system hyperexcitability in children. *J. Am. Coll. Nutr.* 2004, 23, 545S-548S.
29. Nogovitsina O.R., Levitina E.V.: Neurological aspects of the clinical features, pathophysiology, and corrections of impairments in attention deficit hyperactivity disorder. *Neurosci. Behav. Physiol.* 2007, 37, 199-202.
30. Program eliminacji niedoboru jodu w Polsce na lata 2009-2011. Minister Zdrowia. Warszawa, 2009. Internet: (accessed 12.11.2011) [http://woda.bonart.com.pl/pdf/Ministerstwo\\_Zdrowia.pdf](http://woda.bonart.com.pl/pdf/Ministerstwo_Zdrowia.pdf)
31. Rozporządzenie Parlamentu Europejskiego i Rady (WE) Nr 1333/2008 z dnia 16 grudnia 2008 r. w sprawie dodatków do żywności. Dziennik Urzędowy Unii Europejskiej. L 354, 31.12.2008. Internet: (accessed 08.11.2011) <http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:2008:354:0016:0033:pl:PDF>
32. Sadowska L.: ADHD - Attention Deficit Hyperactiv Disorder – genetyczne przeznaczenie czy zaburzenie rozwojowe dające się korygować w procesie wykowawczym. *Med. Środ.* 2006, 9, 73-80.
33. Scholastic News Online. Turn off the TV to fight fat and ADHD: television commercials can affect your child's diet, and in turn, his learning. Internet: (accessed 07.11.2011) <http://www.scholastic.com/resources/article/turn-off-the-tv-to-fight-fat-and-adhd/>.
34. Scholl T.O.: Iron status during pregnancy: setting the stage for mother and infant. *Am. J. Clin. Nutr.* 2005, 81(suppl), 1218S-22S.
35. Shaw P., Eckstrand K., Sharp W., Blumenthal J., Lerch J.P., Greenstein D., Clasen L., Evans A., Giedd J., Rapoport J.L.: Attention-deficit/hyperactivity disorder is characterized by a delay in cortical maturation. *P. Natl. Acad. Sci. USA.* 2007, 104, 19649-19654.
36. Singh M.: Essential fatty acids, DHA and human brain. *Indian J. Pediat.* 2005, 72, 239-242.
37. Stanowisko ekspertów Polskiego Towarzystwa Pediatrycznego (PTP) i Polskiego Towarzystwa Badań nad Mięźdzycą (PTBnM) dotyczące przeciwdziałania stanom niedoborowym wielonienasyconych kwasów tłuszczowych omega-3 w żywieniu kobiet w ciąży, niemowląt i dzieci w Polsce. Czynniki Ryzyka. 2007, 1, 4.
38. US Department of Health and Human Services, US Environmental Protection Agency. What you need to know about mercury in fish and shellfish (brochure). Advice for: women who might become pregnant, women who are pregnant, nursing mothers, young children. Washington, DC, 2004. EPA-823-R-04-005 Internet: (accessed 12.11.2011) <http://www.fda.gov/Food/ResourcesForYou/Consumers/ucm110591.htm>
39. Wolraich M.L., Lindgren S.D., Stumbo P.J., Stegink L.D., Appelbaum M.I., Kiritsy M.C.: Effects of diets high in sucrose or aspartame on the behavior and cognitive performance of children. *New Engl. J. Med.* 1994, 330, 301–307.
40. Vermiglio F., Lo Presti V.P., Moleti M., Sidoti M., Tortorella G., Scaffidi G., Castagna M.G., Mattina F., Violi M.A., Crisà A., Artemisia A., Trimarchi F.: Attention deficit and hyperactivity disorders in the offspring of mothers exposed to mild-moderate iodine deficiency: a possible novel iodine deficiency disorder in developed countries. *J. Clin. Endocrinol. Metab.* 2004, 89, 6054-6060.
41. Voigt R.G., Llorente A.M., Jensen C.L., Fraley J.K., Berretta M.C., Heird W.C.: A randomized, double-blind, placebo-controlled trial of docosahexaenoic acid supplementation in children with attention-deficit/hyperactivity disorder. *J. Pediatr.* 2001, 139, 189-196.
42. Zimmer L., Delion-Vancassel S., Durand G., Guilloteau D., Bodard S., Besnard J.-C., Chalon S.: Modification of dopamine neurotransmission in the nucleus accumbens of rats deficient in n-3 polyunsaturated fatty acids. *J. Lipid. Res.* 2000, 41, 32–40.

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