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Dietary patterns and blood fatty acid composition in children with attention-deficit hyperactivity disorder in Taiwan

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Abstract

Nutritional factors may be relative to attention-deficit hyperactive disorder (ADHD), although the pathogenic mechanism is still unknown. Based on the work of others, we hypothesized that children with ADHD have altered dietary patterns and fatty acid metabolism. Therefore, the aim of this study was to evaluate dietary patterns and the blood fatty acid composition in children with ADHD in the Taipei area of Taiwan. The present study found that 58 subjects with ADHD (average age 8.5 years) had significantly higher intakes of iron and vitamin C compared to those of 52 control subjects (average age 7.9 years) ($P < 0.05$). The blood total protein content in subjects with ADHD was significantly lower than that in control subjects ($P < 0.05$). On the other hand, children with ADHD had significantly higher blood iron levels compared to the control children ($P < 0.05$). Additionally, plasma γ-linolenic acid (18:3 n-6) in children with ADHD was higher than that in control children ($P < 0.05$). Concerning the composition of other fatty acids in the phospholipid isolated from red blood cell (RBC) membranes, oleic acid (18:1n-9) was significantly higher, whereas nervonic acid (24:1n-9), linoleic acid (18:2n-6), arachidonic acid (20:4n-6), and docosahexaenoic acid (22:6n-3) were significantly lower in subjects with ADHD ($P < 0.05$). Our results suggest that there were no differences in dietary patterns of these children with ADHD except for the intake of iron and vitamin C; however, the fatty acid composition of phospholipid from RBC membranes in the ADHD children differed from that of the normal children.

Keywords: Attention-deficit hyperactivity disorder (ADHD); Dietary pattern; Blood fatty acid composition

1. Introduction

Attention-deficit hyperactivity disorder (ADHD) is the term used to describe children who are inattentive, impulsive, and hyperactive [1]. These behaviors may severely affect school performance, family relationships, and social interactions. ADHD is thought to affect 3–5% of the Chinese school-age population, and boys are more commonly identified with ADHD than are girls [1]. Moreover, recent systemic reviews have also reported the ADHD prevalence to be as great as 2–18% [2]. In Taiwan, 2% of school-age children have been diagnosed as having ADHD [3]. At present, the cause of ADHD is unknown, but it is thought to be biological and multifactorial [4]. For example, various studies have suggested that genetic factors, neurotransmitter imbalances, lead toxicity, food sensitivities, or nutritional problems might affect behavior in children with ADHD [5–9].

It is known that nutritional factors such as metabolism of glucose or fatty acid as well as deficiencies of tryptophan, vitamins, or minerals may affect brain function [10–14]. Furthermore, some studies have focused on essential fatty acid (EFA) metabolism in children with ADHD. EFAs play important structural roles as components of all cell membranes and affect their biological properties. EFAs serve as precursors to substrates in the biosynthesis of eicosanoids, which mediate a wide variety of functions in every cell in the body [15].

In Taiwan, few studies have focused on the relationships between ADHD and nutritional factors. Dietary patterns are endemic and present in the local culture in a specific region

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or country. Therefore, it is considered that the results of foreign research might not reflect the actual situation in Taiwan, although many papers have discussed ADHD and nutrition, especially fatty acid metabolism.

In the present study, dietary patterns were evaluated and the fatty acid composition of lipids from plasma and red blood cell (RBC) membranes in control subjects and in children with ADHD were compared in the Taipei area of Taiwan.

2. Methods and materials

2.1. Subject selection

A total of 58 children with ADHD, 4–12 years of age, participated as volunteers from Taipei Medical University Hospital and Taiwan Adventist Hospital. In addition, 52 control subjects were recruited from local kindergartens and elementary schools. The children with ADHD, who were diagnosed through diagnostic interviews by psychiatrists with the Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition (DSM-IV) as having ADHD, and their parents were interviewed at the hospital. Investigators sent each family a pamphlet outlining the purpose of the study and the steps required for each subject: completion of questionnaires by parents, a 3-day diet record, and a blood test. The protocol was approved by the Taipei Medical University Hospital. Investigations of the control subjects were completed at the respective schools.

2.2. Anthropometric assessment

After subjects agreed to participate in this study, investigators evaluated the anthropometric data of children with ADHD and the control children, when their parents had completed the questionnaires. Anthropometric data included height, weight, chest perimeter, and triceps skinfold thickness.

2.3. Dietary assessment

Completion of the 3-day diet record was explained in detail to parents by using food models to illustrate serving sizes. Each family could choose 3 continuous days for recording, and the record was sent back by mail. A computer was used to analyze these records for food components with a software program (Food Composition Analyzer, November 1997 edition, provided by Dr. Wen-Harn Pan from the Institute of Biochemical Sciences, Academia Sinica, Taipei, Taiwan, R.O.C.).

2.4. Biochemical analysis

2.4.1. Blood sample preparation

After 8 hours of fasting, 7 mL of venous blood was drawn from each subject and immediately divided into different tubes, including a tube containing EDTA (1 mL), a tube without anticoagulant (2 mL), and a tube containing heparin (4 mL). The different tubes of blood samples were used to measure the different biochemical data as follows.

2.4.2. Whole blood analysis

Whole blood samples in tubes containing EDTA were used to analyze the levels of hematocrit (Hct), hemoglobin (Hb), RBCs, white blood cells (WBCs), and lymphocytes using a the blood autoanalyzer (ADVIA 120; Bayer, Tarrytown, NY, USA).

2.4.3. Serum analysis

Blood in tubes without anticoagulant was centrifuged at $1400 \times g$ for 10 min at 4 °C (Kubota 5800; Kubota, Tokyo, Japan). Then, the serum was reserved for analyses using a Hitachi 7170 autoanalyzer (Hitachi, Tokyo, Japan), including albumin, total protein (TP), blood lipid profile, blood urea nitrogen (BUN), creatinin, GOT, GPT, alkaline phosphatase (ALK-P), billirubin, electrolytes (Na^+, K^+, and Cl^-), and minerals (Ca, P, and Fe).

2.4.4. Fatty acid analysis

Blood in tubes containing heparin was used for fatty acid analysis. Blood was centrifuged at $1400 \times g$ for 10 minutes at 4°C. The RBC pellet was resuspended in phosphate-buffered saline (PBS) and washed twice with PBS. After centrifugation, the upper layer was discarded. The RBC pellet was resuspended to 50% of its original volume and stored at −80°C. Plasma was also stored at −80°C for fatty acid analysis.

Phospholipids were isolated from RBC membrane by the method of Edward et al. [16]. Then, lipids were extracted from plasma using the method of Bligh and Dyer [17] and evaporated to dryness under nitrogen. Lipids were hydrolyzed and methylated with boron fluoride and methanol as reported by Holman et al. [18]. The fatty acid methyl esters were separated by gas chromatography (Hewlett Packard 5890 Series II–type; Hewlett Packard, Andover, MA) with a 25×0.32–mm capillary column (DB23, J&W Scientific, Folsom, CA, USA) and a flame ionization detector. Nitrogen was the carrier gas. The initial oven temperature of 160 °C was held for 4 minutes and increased at a rate of 2.5 °C per minute until the final temperature of 225 °C was reached. The total gas-chromatographic run was 60 minutes.

2.5. Statistical analysis

All data are expressed as the mean ± SD. Data were analyzed by means of the Student t test to determine the variance between subjects with ADHD and control subjects. Statistical significance was assigned at the level of $P = 0.05$.

Table 1
Characteristics and anthropometric data in control subjects and subjects with attention-deficit hyperactivity disorder (ADHD)[1]

	Control subjects	ADHD[2]
Sample size	52	58
Ratio of boys to girls	40/12	53/15
Age (y)	7.9 ± 2.0	8.5 ± 2.2
Height (cm)	128.7 ± 15.4	128.5 ± 11.6
Weight (kg)	30.6 ± 11.2	29.8 ± 9.3
BMI (kg/m^2)	18.5 ± 3.8	18.0 ± 2.3
Chest perimeter (cm)	63.4 ± 10.4	65.7 ± 11.6
Triceps skinfold (mm)	15.8 ± 6.1	14.3 ± 7.3

[1] Values are mean ± SD. The data did not differ significantly ($P > 0.05$) between the groups by Student's *t*-test.

[2] Thirty-four percent of children with ADHD in this study took medicines to improve their symptoms. Methylphenidate (Ritalin) was a major one among these medicines.

3. Results

Characteristics and anthropometric data of subjects in the study are summarized in Table 1. Subjects in the control and ADHD groups had similar values with regard to sample size, age, height, weight, BMI, chest perimeter, and triceps skinfold thickness. Of the subjects with ADHD, 34% were on medication to control their ADHD symptoms.

Table 2
Dietary intake from the 3-day dietary records in control subjects and subjects with attention-deficit hyperactivity disorder (ADHD)

	Control subjects	ADHD subjects
Energy (kcal/day)	1685 ± 524	1742 ± 556
Protein (g/day)	50 ± 21	54 ± 24
Tryptophan (mg/day)	614 ± 271	739 ± 348
Fat (g)	67 ± 32	65 ± 31
Polyunsaturated fatty acids		
Linoleic acid (g/day)	13.7 ± 8.0	16.2 ± 8.0
Linolenic acid (g/day)	1.1 ± 0.8	1.2 ± 0.7
Monounsaturated fatty acids		
Oleic acid (g/day)	23.6 ± 12.0	22.3 ± 11.5
Saturated fatty acids	22.7 ± 11.5	19.8 ± 12.7
Cholesterol (mg/day)	314 ± 171	311 ± 264
Carbohydrate (g/day)	213 ± 78	225 ± 92
Dietary fiber (g/day)	2.4 ± 1.0	2.5 ± 1.3
Energy composition		
Protein (% of energy)	13.5 ± 4.4	13.4 ± 5.5
Fat (% of energy)	35.1 ± 11.3	33.3 ± 9.9
Carbohydrate (% of energy)	51.0 ± 13.0	51.7 ± 11.9
Calcium (mg/day)	462 ± 260	438 ± 423
Phosphorus (mg/day)	878 ± 345	850 ± 389
Iron (mg/day)	7.4 ± 2.9	10.6 ± 9.3*
Sodium (mg/day)	1387 ± 1137	1862 ± 1191
Vitamin A (IU/day)	4426 ± 3757	3646 ± 3470
Vitamin E (mg α-TE/day)	6.2 ± 3.3	9.3 ± 4.9
Vitamin B_1 (mg/day)	0.77 ± 0.41	0.96 ± 0.66
Vitamin B_2 (mg/day)	1.00 ± 0.45	1.24 ± 1.03
Niacin (mg/day)	11.8 ± 5.5	14.8 ± 12.5
Vitamin C (mg/day)	69.1 ± 49.3	106.6 ± 76.6*

Values are mean ± SD.
* Significantly different from control subjects ($P < 0.05$).

Table 3
Blood parameters in control subjects and subjects with attention-deficit hyperactivity disorder (ADHD)

	Control subjects	ADHD subjects
Albumin (g/L)	44.8 ± 1.9	44.7 ± 2.8
Total protein (g/L)	75.0 ± 3.2	72.6 ± 4.0*
Triglyceride (mmol/L)	0.72 ± 0.56	0.58 ± 0.23
Total cholesterol (mmol/L)	4.2 ± 0.6	4.3 ± 0.7
LDL-C (mmol/L)	2.4 ± 0.5	2.5 ± 0.6
HDL-C (mmol/L)	1.5 ± 0.2	1.6 ± 0.3
RBC (× 10^{12}/L)	4.6 ± 0.4	4.8 ± 0.5
Hemoglobin (g/L)	128.7 ± 8.7	131.5 ± 13.9
Hematocrit (proportion of 1.0)	0.37 ± 0.03	0.38 ± 0.04
Blood urea nitrogen (mmol/L)	4.8 ± 1.0	5.0 ± 1.0
Creatinine (μmol/L)	48.6 ± 8.0	50.4 ± 8.8
GOT (μkat/L)	0.44 ± 0.10	0.44 ± 0.14
GPT (μkat/L)	0.23 ± 0.08	0.22 ± 0.05
Alkaline phosphatase (μkat/L)	7.5 ± 1.6	7.1 ± 1.9
Bilirubin (μmol/L)	7.2 ± 4.3	8.2 ± 2.7
WBC (× 10^9/L)	7.9 ± 2.1	7.6 ± 1.6
Lymphocyte (proportion of 1.0)	0.37 ± 0.1	0.41 ± 0.07
Sodium (mmol/L)	145.5 ± 59.7	139.2 ± 6.2
Potassium (mmol/L)	4.4 ± 0.4	4.8 ± 0.5
Chloride (mmol/L)	103.4 ± 2.1	103.1 ± 3.6
Calcium (mmol/L)	2.4 ± 0.1	2.4 ± 0.1
Phosphorus (mmol/L)	1.6 ± 0.1	1.5 ± 0.2
Iron (μmol/L)	16.1 ± 6.8	19.7 ± 6.4*

Values are mean ± SD.
* Significantly different from control subjects ($P < 0.05$).

Data of the 3-day dietary intake are shown in Table 2. Except for intakes of iron and vitamin C, there were no significant differences between the two groups in the intakes of other nutrients. The ADHD groups consumed more iron and vitamin C than the control group by 43% and 53%, respectively.

Table 3 shows the blood parameters in the two groups. When compared with control subjects, subjects with ADHD showed significantly lower concentrations of total protein. However, the iron content in the blood was significantly higher in the ADHD group compared with that in the control subjects.

Results of the analysis of plasma fatty acid composition expressed as area percentages are given in Table 4. Except for the mean content of 18:3n-6, there was no significant difference between the two groups in the mean content of other fatty acids. That is, mean contents of 18:3n-6 (γ-linolenic acid, GLA) in the ADHD group were significantly higher. None of the other fatty acid indexes differed. However, 24:1n-9, 22:4n-6, 20:5n-3, and 22:5n-3 were not detected in plasma.

Results of the analysis of phospholipid isolated from RBC membranes, expressed as area percentages, are given in Table 5. The mean contents of 18:1n-9 (oleic acid) was significantly elevated in the ADHD group when compared with the control group. However, mean concentrations of

Table 4
Fatty acid composition of lipids from plasma in control subjects and subjects with attention-deficit hyperactivity disorder (ADHD)

	Control subjects	ADHD subjects
	Area %	
Saturated fatty acids		
14:0	0.95 ± 0.47	0.86 ± 0.27
16:0	18.24 ± 4.14	17.28 ± 2.27
18:0	9.41 ± 2.14	8.88 ± 2.42
20:0	0.89 ± 0.42	1.07 ± 0.75
Monounsaturated fatty acids		
18:1n-9	21.74 ± 3.10	20.72 ± 6.87
20:1n-9	1.18 ± 0.49	1.17 ± 0.47
24:1n-9	ND	ND
Polyunsaturated fatty acids (n-6)		
18:2n-6	33.81 ± 6.90	33.66 ± 6.94
18:3n-6	1.48 ± 0.57	1.97 ± 1.28*
20:3n-6	1.22 ± 0.41	1.47 ± 1.15
20:4n-6	6.55 ± 1.08	6.78 ± 1.87
22:4n-6	ND	ND
Polyunsaturated fatty acids (n-3)		
18:3n-3	1.81 ± 0.88	1.82 ± 0.53
20:5n-3	ND	ND
22:5n-3	ND	ND
22:6n-3	3.91 ± 1.58	5.07 ± 4.26
Totals and ratios		
Σn-6 fatty acids	43.18 ± 7.07	43.83 ± 6.97
Σn-3 fatty acids	5.41 ± 2.05	5.99 ± 1.57
Σn-6: Σn-3	9.35 ± 4.71	8.42 ± 4.49

Values are mean ± SD.
* Significantly different from control subjects ($P < 0.05$).
ND = not detected.

Table 5
Fatty acid composition of phospholipid isolated from red blood cell membranes in control subjects and subjects with attention deficit hyperactivity disorder (ADHD)

	Control subjects	ADHD
	Area %	
Saturated fatty acids		
14:0	1.08 ± 0.60	1.58 ± 0.45
16:0	22.96 ± 3.45	23.90 ± 1.34
18:0	15.75 ± 2.30	16.02 ± 2.34
20:0	5.38 ± 2.07	5.51 ± 0.99
Monounsaturated fatty acids		
18:1n-9	15.36 ± 2.18	18.32 ± 5.65*
20:1n-9	1.30 ± 2.22	1.86 ± 0.68
24:1n-9	1.35 ± 0.86	1.08 ± 0.86*
Polyunsaturated fatty acids (n-6)		
18:2n-6	20.08 ± 2.05	16.04 ± 0.36*
18:3n-6	0.11 ± 0.61	0.26 ± 0.12
20:3n-6	1.05 ± 0.82	1.35 ± 0.35
20:4n-6	11.51 ± 3.32	9.91 ± 1.35*
22:4n-6	0.96 ± 1.25	0.90 ± 0.21
Polyunsaturated fatty acids (n-3)		
18:3n-3	0.09 ± 0.02	0.05 ± 0.08
20:5n-3	0.18 ± 0.11	0.15 ± 0.02
22:5n-3	1.32 ± 0.23	1.48 ± 0.97
22:6n-3	2.08 ± 0.94	1.35 ± 0.37*
Totals and ratios		
Σn-6 fatty acids	34.01 ± 3.82	28.19 ± 4.63
Σn-3 fatty acids	3.58 ± 1.56	2.53 ± 1.34*
Σn-6: Σn-3	9.65 ± 1.82	11.07 ± 2.35*

Values are mean ± SD.
* Significantly different from control subjects ($P < 0.05$).

24:1n-9 (nervonic acid), 18:2n-6 (linoleic acid, LA), 20:4n-6 (arachidonic acid, AA), and 22:6n-3 (docosahexaenoic acid, DHA) were significantly lower in the ADHD group. There was no difference in the mean concentration of total n-6 fatty acids between the two groups. The mean content of total n-3 fatty acids was significantly lower, whereas the mean ratio of total n-6 to n-3 was significantly higher in ADHD group.

4. Discussion

Many studies have suggested a variety of etiologies for ADHD; thus ADHD appears to be multifactorial. Children with ADHD in Taiwan were the subjects of this study, and correlations between ADHD and nutritional factors such as dietary patterns, blood biochemical analysis, and fatty acid composition in plasma or RBCs were investigated.

There were no significant differences in characteristics between ADHD and control subjects. Height, weight, chest perimeter, and triceps skinfold thickness in children with ADHD were also similar to those in the control children.

According to the results of the 3-day diet record, the intake or ratios to total energy of three major nutrients did not differ between the two groups. The intake pattern of fatty acids in children with ADHD was also similar to that of the control children. Although the ratio of protein was within the range of the recommendation of the Recommended Daily Allowance (USA RDA) or the Recommended Daily Nutrient Allowance (Taiwan, R.O.C.; RDNA), the ratio of fat was greater than the recommended range (25–30%) not only in control subjects but also in children with ADHD.

It generally thought that children with ADHD should expend more energy in physical activity that will affect the total calorie expenditure. However, in this study, there was no difference in the calorie intakes and the anthropometric data between the control and ADHD children. Therefore, it is necessary to assess the physical activity of ADHD children in a further study.

On the other hand, the results also indicated that the intake of iron was higher in children with ADHD than in control subjects. Pollitt et al. indicated that a dietary iron deficiency–induced abnormal behaviors that were similar to ADHD, such as a short attention time [19,20]. Iron is a cofactor for the enzymes tyrosine hydroxylase and tryptophan hydroxylase, which are essential for the synthesis of the neurotransmitters dopamine, norepinephrine, and serotonin. Thus, an iron deficiency may modify the production

of these neurotransmitters and affect the central nervous system (CNS) [21]. However, dietary records and blood biochemical data (e.g., RBCs, hemoglobin, hematocrit, and iron levels) showed no inadequate iron intake or iron deficiency anemia in children with ADHD. Therefore, these results do not seem to explain the relationship between iron deficiency and ADHD.

Similar to iron intake, children with ADHD consumed more vitamin C than did control subjects. Gershoff reported that vitamin C was involved in the hydroxylation of tryptophan for the synthesis of the neurotransmitter serotonin in the brain [22]. Moreover, it was also indicated that vitamin C might prevent serotonin from being oxidized and thus increase the combination of serotonin with receptors in the cerebral cortex [23]. Spivak et al. reported that serotonin exerted a calming effect, including drowsiness and alleviation of aggressive behavior, and that children with ADHD had a low serum level of serotonin [24]. From these reports, it was suggested that a higher intake of vitamin C in children with ADHD might benefit the synthesis and antioxidation of serotinin and thereby diminish hyperactive symptoms. To confirm this theory, blood levels of vitamin C in children with ADHD must be measured in a further study.

The blood total protein level in children with ADHD was significantly decreased, although the dietary protein intake showed no variation. Of the children with ADHD in this study, 34% were on medication to improve their symptoms. Methylphenidate (Ritalin; Novartis Pharmaceuticals, USA) was a major medication among those given. Ritalin, which is a CNS stimulant, is also used in therapy for depression. Newcorn indicated that chronic administration of Ritalin might induce body weight loss and protein loss, finally leading to a decrease in blood total protein [25]. On the other hand, the blood total protein level also reflects the amino acid pattern in the body. Bornstein et al. suggested that the levels of phenylalanine, tryptophan, isoleucine, and tyrosine were lower in subjects with ADHD [26]. Reimherr et al. reported that supplementation with L-tyrosine temporarily improved the symptoms of ADHD [27].

Children with ADHD had higher oleic acid content and lower nervonic acid in the phospholipids in RBC membranes. Oleic acid is converted to nervonic acid by elongation of the carbon chain. Nervonic acid can accumulate in the sphingomyelin of RBC membranes and can yield information on cerebral maturation in premature infants [28]. Babin et al. indicated that the concentration of nervonic acid in sphingomyelin of RBCs significantly increased according the gestational age of infants at birth [28]. They also suggested that nervonic acid levels in sphingomyelin of RBCs might reflect the nervonic acid levels in sphingomyelin of the brain and could reflect brain maturity in infants [28]. Therefore, it may be considered that the conversion of oleic acid to nervonic acid is irregular in children with ADHD, and that the irregular metabolism of fatty acids might be related to the symptoms of ADHD.

On the other hand, subjects with ADHD had lower concentrations of linoleic acid, arachidonic acid, and docosahexsaenoic acid in the phospholipid isolated from the RBC membranes. Additionally, the total n-3 fatty acids of lipid from RBC membranes were also lower in ADHD subjects. Many studies also reported significantly lower concentrations of linoleic acid, arachidonic acid, and docosahexsaenoic acid in hyperactive children, but the actual cause is not clear [29,30]. In our study, the dietary factor was first excluded, because the dietary intake data showed no difference in polyunsaturated fatty acid (PUFA) intake between the two groups. Furthermore, Johanna et al. indicated that the fatty acid composition in RBC membranes reflects the dietary pattern before the 2 weeks to 3 months [31]. Thus, a long period of dietary records is necessary in future studies. Another possible reason to explain the lower concentrations of linoleic acid, arachidonic acid, and docosahexsaenoic acid is an inferior ability to convert 18-carbon fatty acids to longer forms in children with ADHD. Decreased concentrations of arachidonic acid and docosahexsaenoic acid could thus adversely affect behavior [32]. First, lower concentrations of substrate might decrease the production of eicosanoids, which act as mediators or modulators of nerve transmission in the CNS [33]. Second, because docosahexaenoic acid is the important PUFA in the cerebral cortex, especially in cell membranes that are the most fluid and metabolically active [33], decreasing concentrations of docosahexaenoic acid might negatively affect the function of the cerebral cortex in several ways, such as in the fluidity and transport processes of cell membranes [34]. The PUFA composition of membranes affects not only their biological and physical properties but also those of the membrane-bound proteins [35]. In contrast, some research has suggested that the lower levels of arachidonic and docosahexaenoic acids could be induced because of the increased metabolism of these two specific fatty acids to eicosanoids [36]. In children with ADHD, this might be related to allergies such as asthma and eczema [37].

Another interesting result was that the mean content of 18:3n-6 (γ-linolenic acid, GLA) in ADHD subjects was significantly higher in plasma. However, in the RBC membranes, there was no significant difference between the control and ADHD groups in regard to the mean content of 18:3n-6. In 2 previous studies, primrose oil (a concentrated source of GLA) failed to improve the behavior in children with ADHD [38,39]. Our results might explain why attempts in those studies failed to control the symptoms of ADHD. From our results, it is reasonable to conclude that the real cause of ADHD might be related to multiple physiological and environmental factors.

In conclusion, our results showed that children with ADHD in Taiwan consumed more iron and vitamin C and had higher blood iron levels than did the control children. In addition, children with ADHD in Taiwan showed a higher content of oleic acid and lower contents of nervonic acid, linolenic acid, arachidonic acid, and docosahexsaenoic acid in the phospholipids isolated from RBC membranes; how-

ever, these children had a higher level of α-linolenic acid in plasma. These results are similar to those of other studies that have discussed fatty acid metabolism in hyperactive children. Although the mechanism could not be identified, impaired fatty acid metabolism in children with ADHD was confirmed once again in our study.

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