Original Article

Nutritional complexity in children with ADHD related morbidities in China: A cross-sectional study

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Background and Objectives: To assess the general and nutritional health of children with attention deficit/hyperactivity disorder (ADHD). **Methods and Study Design:** The National Multicenter Sleep Research Database for 23791 school-age children in grades 1-6 from 9 cities in China was accessed. Children with a specialist diagnosis of ADHD or not (non-ADHD) in 2005 were studied. National anthropometric growth standards for children aged 2-18 years classified children as underweight, wasted, stunted (short stature presumed nutritional), or overweight/obesity. Independent variables were preterm birth, sleep quality and prior disease and ADHD was the dependent variable. Binary logistic regression models were developed along with interaction analyses for associated disorder or disease on overweight/obesity, and stunted. **Results:** Some 18731 records were analyzed for 808 children with ADHD. The comparative prevalences for ADHD with non-ADHD children were stunted 9.8% vs 5.9% (p<0.001) and overweight/ obesity (32.6% vs 29.6%, p=0.002) respectively. ADHD boys were more often underweight (7.5% vs 5.3%, p=0.027), but not in girls. ADHD likelihood Odds Ratios, ORs (with 95%CI) were for premature birth 1.838, (1.393-2.423), allergic diseases 1.915 (1.526-2.399), otitis media 1.54 (1.118-2.146), tonsillar or adenoid hypertrophy1.662 (1.348-2.050), gastroesophageal reflux 3.008(1.792-1.792-5.049), and sleep disorder 2.201(1.847-2.623) were ADHD risk factors. Only poor sleep quality and ADHD exhibited an interaction for stunted with OR=0.409 (0.233-0.719). **Conclusions:** Compromised and complex nutritional health in ADHD children challenges clinical nutrition with a range of health problems, albeit coherent with the needed nutritional emphasis in the 'first 1000 days'.

Key Words: attention deficit disorder with hyperactivity, stunted, underweight, overweight/obesity, school-age children, first 1000 days

INTRODUCTION

Attention deficit/hyperactivity disorder (ADHD) is the most common neurodevelopmental disorder affecting the health of children and adolescents. The incidence of ADHD in China is 5.7%.[1] ADHD is a chronic disorder as 60-80% of ADHD cases can persist through adolescence, and 50% are affected in adulthood, with 71.9% of ADHD patients having concomitant disease.[2] The direct or potentially synergistic effects of ADHD, with comorbidities and pharmacotherapy, increase proneness to further health problems. These include nutritional disorders of growth, energy regulation attributable to physical activity, eating disorders,[3] sleep disorders or poor diet,[4,5] and immunoinflammatory disorder such as recurrent infection and allergic disease.[6-9] In children with ADHD who have reduced height and growth rates this may have been due to the long-term use of stimulants.[7] To understand, recognise and more effectively manage the health status of school-age children with ADHD in China, their physical growth and disease status the national multicenter children's sleep quality survey database. Our general hypothesis is that growth and body composition contribute to the health and wellbeing of children with ADHD, implicitly, to its mitigation and management.

METHODS

Participants and study design

Cluster sampling was conducted from November to December 2005 in 32 provinces (cities and autonomous regions). Nine cities including Shanghai, Guangzhou, Xi'an, Wuhan, Chengdu, Harbin, Hohhot, Urumqi and Shi he zi, were randomly selected for the questionnaire survey.

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Cluster-stratification was applied by city, district, school, grade, and class. Finally, 39 districts and 55 primary schools were selected (Figure 1). The study was approved by the Ethics Committee of Xinhua Hospital Affiliated with Shanghai Jiaotong University (No. XHEC-D-2017-047).

Questionnaires

Study objectives were explained to the principals and teachers of the selected schools. After permission was granted, children were asked to take the proposal, questionnaire and explanatory files to their parents. Participation depended on parental agreement and an assurance that information provided would be treated with anonymity. Eligible students were aged 5-12 years (grades 1 to 6) and studying in the selected schools. The survey was in three parts. The first comprised key sociodemographic information including gender, age, and perinatal factors (delivery mode, gestational weeks, and feeding patterns). The second documented family environmental factors (education level of the caregivers, family income, marital status of parents, and geographic location) The third explored disease history (ADHD, nasal obstruction, asthma, otitis media, tonsillar or adenoid hypertrophy, gastroesophageal reflux, food allergy, allergic rhinitis, and obesity). Each questionnaire had an informed consent form attached for agreement.

Measurement of physical growth

Physical measurements were made by school doctors and community health centers. Weight was measured using an electronic pediatric scale to the nearest 0.1 kg, and length was measured using a pediatric ruler to the nearest 0.1 cm. Growth and body compositional status was classified by the criteria for 2- to 18-year-old children in China in 2005, according to sex and age. Children below the 3rd percentile for weight were defined as underweight, below the 3rd percentile value of height as stunted, below the 3rd percentile value of body mass index (BMI) as wasted, above the 85th percentile value of BMI as overweight, and above the 97th percentile of BMI as obese.[10]

ADHD diagnosis

ADHD presence was based firstly on the parents' answers to the question: "has the child been diagnosed with ADHD by a specialist (developmental behavioral pediatrician or psychiatrist, DSM-IV diagnostic standard)? (yes or no)." Then, children were divided into an ADHD group and a non-ADHD group according to whether ADHD was diagnosed by a specialist. Behavioral–developmental pediatricians at behavioral pediatric clinics made ADHD diagnoses using the Diagnostic and Statistical Manual of Mental Disorders–Fourth Edition (DSM-IV; American Psychiatric Association, 2000) diagnostic criteria.

Age groups

The age range was from 6 years 0 days to 12 years 11-30 days, and each age group divided further into the following groups. Six years 0 days to 6 years 11-30 days as the 6-year-old group, and this pattern followed through to the 12-year-old group, group, resulting in 6 groups: the 6-, 7-, 8-, 9-, 10-, 11- and 12-year-old groups. This grouping avoided interference by children under 6 years old and over 13 years old (possible because of the early school and follow-up school children populations).

Statistical analysis

SPSS 21.0 (IBM, Armonk, NY, USA) was used for statistical analysis. Categorical data were described as frequencies and analyzed by chi-square. Continuous data are shown as mean ± standard deviation and analyzed with Student's t test or analysis of variance with Tukey's post hoc test, as appropriate. The factors with statistical significance on univariable analysis were included as independent factors in the multivariable binary logistic regression model along with ADHD as the dependent factor. Using preterm birth, sleep quality and prior disease as independent variables and diagnosed ADHD as the dependent variable, binary logistic regression models were established. Difference was regarded as statistically significant with $p<0.05$.

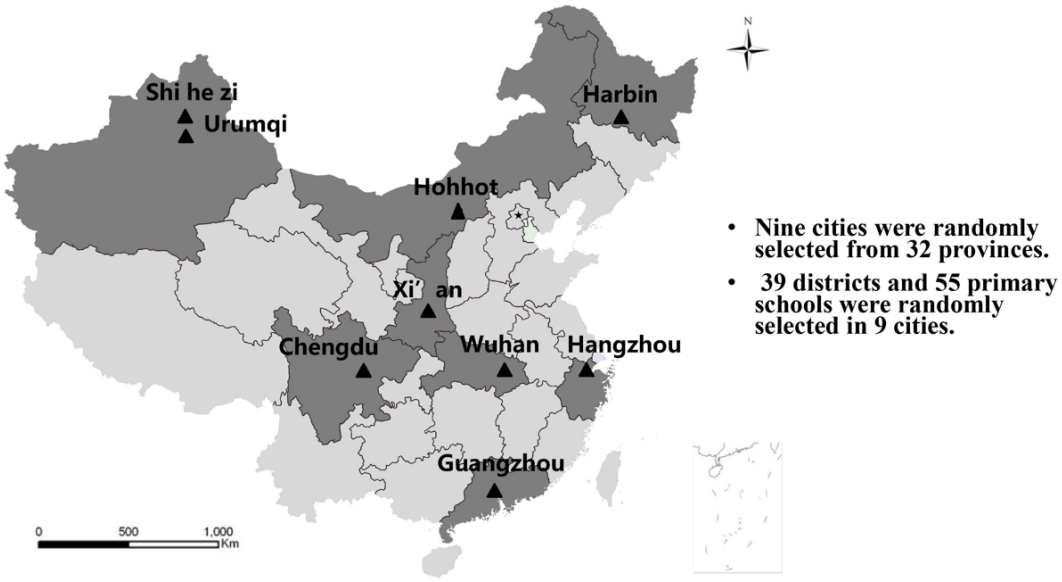

Figure 1. Geographical distribution of the nine cities (shaded for corresponding province) in China and number of selected schools.

RESULTS

Malnutrition of school-age ADHD children and non-ADHD children

In the survey, 23,791 children recruited from six grades of the chosen schools, 22018 (92.5%) returned completed questionnaires, and 970 (4.4%) children with ADHD. Among them, 18731 [9252 boys (49.4%) and 9479 girls (50.6%)] students within 6-12 years completed physical measurements, and 808 (4.3%) children with ADHD [532 boys (6.6%) and 276 girls (3.4%)], met the inclusion criteria.

Overweight, obesity, wasted, underweight and stunted prevalences were 15.9%, 11.9%, 4.8%, 2.5% and 5.2%, respectively. As shown in Table 1, the proportion of ADHD children who were overweight and obese was significantly higher than for non-ADHD children (32.6% vs 29.6%, χ^2=9.904, p=0.002). Those wasted among ADHD children were more prevalent than among non-ADHD children, but there was no significant differences between these groups (7.1% vs 5.5%, χ^2=3.461, p=0.063). The prevalence of stunted was significantly higher among ADHD children than among non-ADHD children (9.8% vs 5.9%, χ^2=20.353, p<0.001). There were no significant differences for underweight between the two groups (3.0% vs 2.9%, χ^2=0.009, p=0.924).

Sex difference in malnutrition among school-age ADHD children and non-ADHD children

The prevalence of stunted, underweight, being wasted and overweight/obesity in boys with ADHD were 9.6%, 3.4%, 7.5% and 35.2%, respectively.

As shown in Table 1, the stunted prevalence was higher in boys with ADHD than in boys without ADHD (9.6% vs 5.7%, χ^2=13.389, p<0.001), and being wasted was also more common in boys with ADHD (7.5% vs 5.3%, χ^2=4.877, p=0.027). For underweight and overweight/obesity, there were no significant differences between groups (underweight, 3.4% vs 2.9%, χ^2=0.389, p=0.533; overweight/obesity, 35.2% vs 35.4%, χ^2=0.018, p=0.894).

In girls, only being of shorter stature was higher with ADHD than in non-ADHD girls (10.1% vs 6.1%, χ^2=7.644, p=0.006). There were no significant differences in prevalence for underweight, being wasted or overweight/obesity between groups (Table 1, Figure 2).

Nutritional status of ADHD and non-ADHD children by age

As shown in Table 2 (Figure 2), except for the 7-year-old age group, the prevalence of being wasted was higher in ADHD than in non-ADHD students (10.53% vs 4.83%, χ^2=7.450, p=0.006). There was no statistically significant differences in prevalence for underweight, being wasted or overweight/obesity in the other age groups of ADHD and non-ADHD children. While the prevalence of overweight/obesity in ADHD children is significantly different in the total population, it is not statistically significant by age group.

In ADHD children stunted prevalence was higher than that in non-ADHD children in the 7-year-old group (10.26% vs 5.15%, χ^2=6.079, p=0.014), in the 8-year-old group (8.88% vs 5.03% χ^2=4.383, p=0.036), in the 9-year-old group (10.73% vs 5.96%, χ2=7.224, p=0.007) and in the 10-year-old group (8.82% vs 5.02%, χ^2=4.427, p=0.035). There was no significant difference in the prevalence of stunted in the 11- to 12-year-old groups between ADHD and non-ADHD children.

Multivariate logistic regression analysis of risk factors for ADHD

As shown in Table 3, a multivariable analysis was deployed to investigate risk factors for ADHD among school-age children. Adjustments were made for individual factors (gender, age), perinatal factors (delivery mode, feeding mode), and family environment factors (according to the education level of the guardian, per capita income of the family, marital status of parents and geographical distribution). Preterm birth (OR=1.838, 95% CI: 1.393-2.423), allergic diseases (allergic rhinitis, food allergy, asthma and repeated nasal congestion) (OR=1.915, 95% CI: 1.526-2.399), otitis media (OR=1.549, 95% CI: 1.118-2.416), tonsil or adenoid hypertrophy (OR =1.662, 95% CI: 1.348-2.050), gastroesophageal reflux (OR=3.008, 95% CI: 1.792-5.049), and poor sleep quality (OR=2.201, 95% CI: 1.847-2.623)
were risk factors for ADHD.

Table 1. Stunted, underweight, wasted, overweight/obesity prevalences for ADHD and non-ADHD school age children (2005 growth references for Chinese children aged 2-18 years)

	Total		Boys		Girls	
	ADHD (n=808)	Non-ADHD (n=17923)	ADHD (n=532)	Non-ADHD (n=8720)	ADHD (n=276)	Non-ADHD (n=9203)
Stunted, n (%)	79 (9.8)	1058 (5.9)	51 (9.6)	499 (5.7)	28 (10.1)	559 (6.1)
χ^2	20.353		13.389		7.644	
p	<0.001***		<0.001***		0.006**	
Underweight, n (%)	24 (3.0)	522 (2.9)	18 (3.4)	254 (2.9)	6 (2.2)	268 (2.9)
χ^2	0.009		0.389		0.520	
p	0.924		0.533		0.471	
Wasted, n (%)	57 (7.1)	989 (5.5)	40 (7.5)	461 (5.3)	17 (6.2)	528 (5.7)
χ^2	3.461		4.877		0.088	
p	0.063		0.027*		0.767	
overweight/obesity, n (%)	264 (32.6)	4947 (29.6)	187 (35.2)	3090 (35.4)	77 (27.9)	2233 (24.3)
χ^2	9.904		0.018		1.921	
p	0.002*		0.894		0.166	

n: Number;.* p<0.05; *** p<0.01; *** p<0.001

Table 2. Stunted, underweight, wasted, overweight/obesity prevalences for ADHD and non-ADHD school-age children by age group (2005 growth reference for Chinese children aged 2-18 years)

Age (year-old)	Number		Stunted (%)		Underweight (%)		Wasted (%)		Overweight/obesity (%)	
	ADHD	Non-ADHD	ADHD	Non-ADHD	ADHD	Non-ADHD	ADHD	Non-ADHD	ADHD	Non-ADHD
6~	70	1961	8.82	4.01	1.41	1.53	7.35	4.65	35.29	30.87
χ^2			3.338		0.007		1.066		0.600	
p			0.068		0.935		0.302		0.438	
7~	115	3136	10.26	5.15	0.00	1.82	10.53	4.83	35.96	33.23
χ^2			6.079		2.128		7.450		0.371	
p			0.014		0.145		0.006		0.542	
8~	165	3217	8.88	5.03	4.62	3.11	8.33	5.96	27.38	29.76
χ^2			4.383		1.223		1.569		0.431	
p			0.036		0.269		0.210		0.511	
9~	174	3115	10.73	5.69	2.79	2.31	3.98	5.55	31.82	27.22
χ^2			7.224		0.172		0.801		1.767	
p			0.007		0.678		0.371		0.184	
10~	130	3023	8.82	5.02	1.52	2.25	4.55	5.38	28.03	26.82
χ^2			4.427		0.314		0.173		0.094	
p			0.035		0.575		0.677		0.760	
11~	94	2375	7.37	6.50	1.05	4.29	8.42	6.19	25.26	21.43
χ^2			0.117		2.403		0.769		0.795	
p			0.732		0.121		0.380		0.373	
12~	38	1097	10.00	9.09	11.90	5.47	10.00	6.14	20.00	18.70
χ^2			0.008		3.113		0.977		0.043	
p			0.928		0.078		0.323		0.836	

Table 3. Likelihood of health disorder in school age children with ADHD by multivariate logistic regression analysis

Factor	ADHD n (%)	Non-ADHD n (%)	β (SE)	OR†	95% CI
preterm birth	70 (8.7)	985 (5.5)	0.608 (0.141)	1.838	1.393-2.423
allergic diseases	211 (26.1)	2724 (15.2)	0.650 (0.115)	1.915	1.526-2.399
otitis media	54 (6.7)	681 (3.8)	0.437 (0.166)	1.549	1.118-2.146
tonsil or adenoid hypertrophy	162 (20.0)	1989 (11.1)	0.517 (0.100)	1.662	1.348-2.050
gastroesophageal reflux	35 (4.3)	108 (0.6)	1.101 (0.264)	3.008	1.792-5.049
poor sleep quality	555 (68.7)	8442 (47.1)	0.789 (0.089)	2.201	1.847-2.623

n: Number.
†Adjusted for age and gender, perinatal factors (mode of delivery and feeding) and family environmental factors (geographical distribution, economic status, caregiver culture, parental marital relationship).

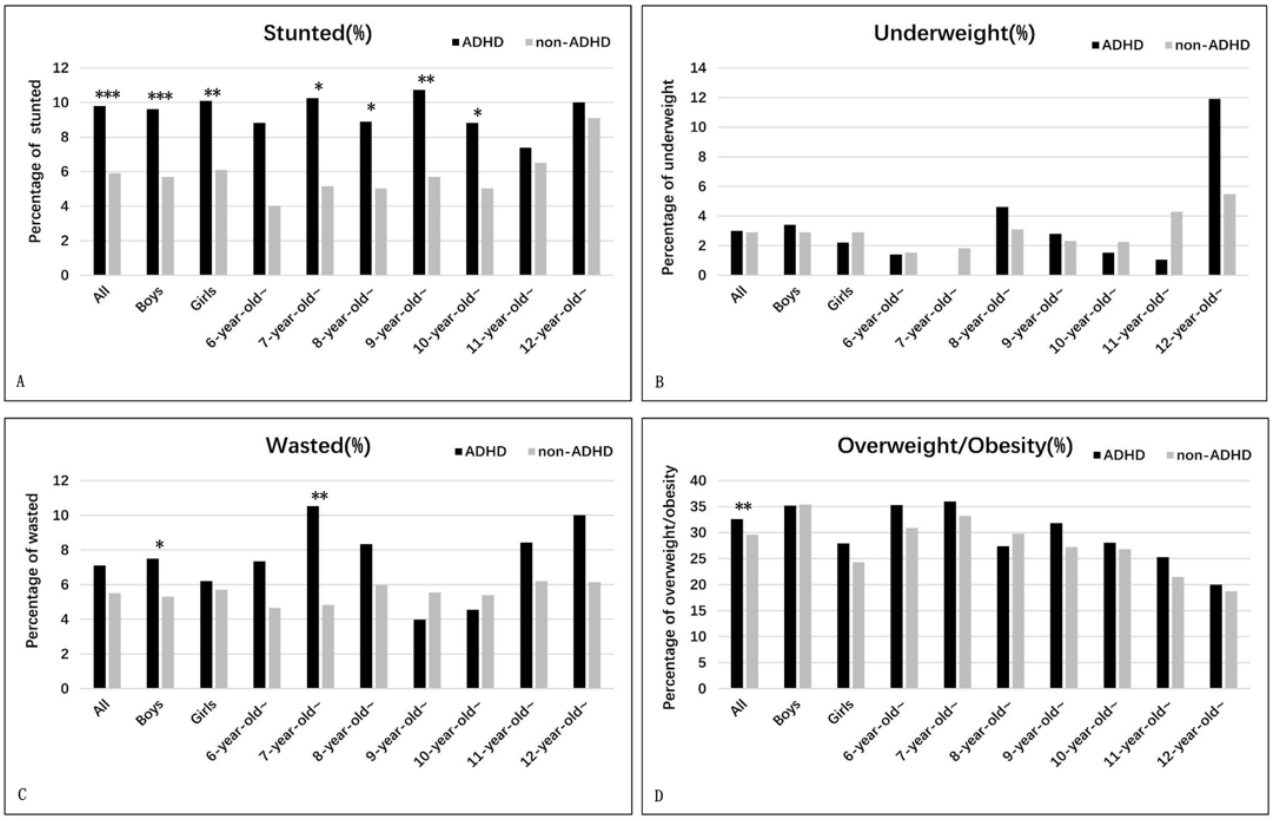

Figure 2. The percentage of (A) stunted, (B) underweight, (C) wasted and (D) overweight/obesity from 2 to 12-year-old school age children with or without ADHD. $^{*}p<0.05$, $^{**}p<0.01$, $^{***}p<0.001$.

As shown in Table 4 and Figure 3, interaction analyses were undertaken to investigate the effects of ADHD and associated disorder or diseases on overweight/obesity and stunted. Only poor sleep quality and ADHD exhibited significant interaction (OR=0.409, 95% CI: 0.233-0.719).

DISCUSSION

ADHD is recognized a major psychological disorder among children and adolescents. It affects growth and development as confirmed in the present investigation, but the mechanisms and broader consequences are unclear. In China, the focus of guidelines for diagnosis and management is behavioural and pharmacotherapy,[2,9] with little attention to the potential of nutritional management.

We found that ADHD children were more commonly of both shorter stature and overweight/obese, representing disordered growth and body composition. This phenomenon is seen increasingly with economic disparity and development, and referred to as the' double burden of disease', an undoubted oversimplification of its pathogenesis.[12-14] The proportion stunted was high in the 7- to 9-year-old age group, the main age range for the diagnosis and prescription of medication for ADHD children. Risk factors for and co-morbidities with ADHD were found to include preterm birth, allergic disease, otitis media, tonsillar or adenoid hypertrophy, gastroesophageal reflux and poor sleep quality. The nutritional status of school-age children with ADHD in China is in a state of polarization, which suggests not only should be paid attention to drug and behavior therapy, but nutrition management should also be included in the development of ADHD diagnosis and treatment guidelines. The finding of an

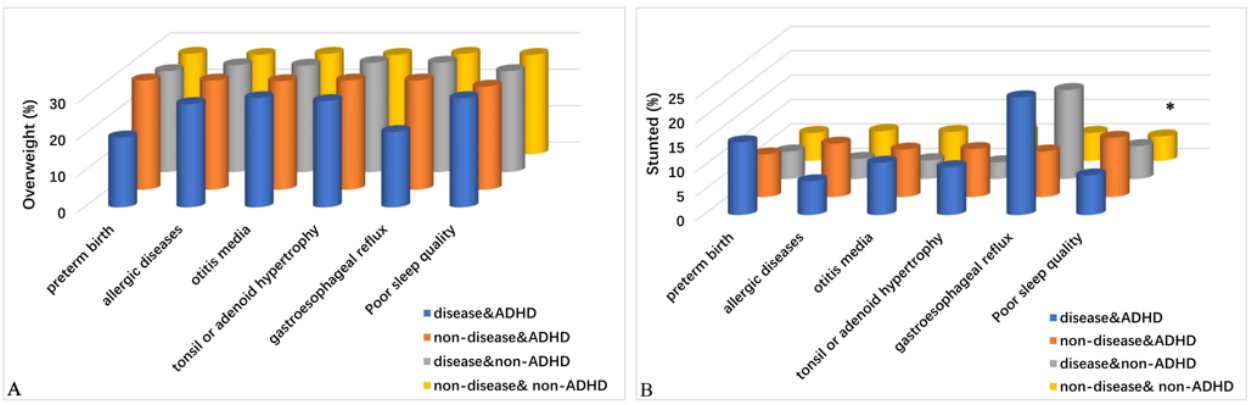

Figure 3. (A) Overweight/obesity and (B) stunted prevalence with or without ADHD & associated disorder or disease. $^{*}p<0.05$.

Table 4. Interactions of ADHD with health status for overweight/obesity or stunted by binary logistic regression analysis

Factor	Overweight/obesity n (%)					Stunted n (%)				
	ADHD	Non-ADHD	*p*	OR[†]	95% CI	ADHD	Non-ADHD	*p*	OR[‡]	95% CI
Preterm birth										
Yes	14 (19.2)	258 (27.7)	0.055	0.539	0.286-1.014	11 (14.9)	54 (5.6)	0.143	1.848	0.812-4.202
No	216 (30.7)	4593 (27.6)				62 (8.7)	967 (5.7)			
Allergic diseases										
Yes	61 (28.4)	810 (29.3)	0.509	0.875	0.589-1.300	15 (6.9)	116 (4.1)	0.553	0.801	0.385-1.666
No	177 (30.5)	4139 (27.3)				64 (10.9)	941 (6.1)			
Otitis media										
Yes	18 (32.1)	202 (29.2)	0.660	0.861	0.441-1.680	6 (10.7)	27 (3.8)	0.275	0.533	0.173-1.647
No	221 (29.8)	4748 (27.6)				73 (9.7)	1031 (5.9)			
Tonsil or adenoid hypertrophy										
Yes	47 (29.2)	620 (30.3)	0.325	1.246	0.804-1.929	16 (9.8)	70 (3.4)	0.167	0.591	0.281-1.246
No	192 (30.2)	4330 (27.3)				63 (9.8)	988 (6.1)			
Gastroesophageal reflux										
Yes	6 (20.7)	35 (32.4)	0.166	2.185	0.723-6.602	7 (24.1)	20 (18.2)	0.410	1.719	0.474-6.233
No	233 (30.3)	4914 (27.6)				72 (9.3)	1037 (5.7)			
Poor sleep quality										
Yes	150 (30.1)	2161 (27.7)	0.206	1.146	0.787-1.669	41 (8.1)	533 (6.7)	0.002	0.409	0.233-0.719
No	60 (28.3)	2391 (27.3)				27 (12.1)	441 (5.0)			

[†]OR: Interaction analysis by binary logistic regression for overweight/obesity as the dependent factor.
[‡]OR: Interaction analysis by binary logistic regression for stunted as the dependent factor.

association of ADHD with premature birth indicates that affected children were likely to have been born with a low birth weight and its attendant intrauterine nutritional determinants and long-term consequences as with intrauterine growth retardation (IUGR).[15,16] This situation is coherent with the substantial evidence that the first 1000 days from conception require attention to their nutritional optimisation for healthful human development.[17,18] Long-term clinical nutrition management in children with ADHD commences pre-conception with maternal health and continues through pregnancy to infancy and childhood. Mitigation in childhood will take account of several potential exacerbators: (1) proneness to abnormal eating behavior (2) nutrition- drug interactions and compliance; and (3) a spectrum of nutritional disorders and interactive complexity.

The probable association of ADHD with abnormal eating behavior, partly manifest in disordered growth (shorter stature) and body composition (fatness and its distribution) has been anticipated in earlier studies in China that of Yang et al conducted in 2007 in the Guangxi area is consistent with the present study.[19] In 2010,[20] Zhejiang reported that the overweight/obesity prevalence among 158 children with ADHD was 29.1%, higher than the 14.6% among children in the Family Planning Commission's investigation of student physique and health and where the basis of comorbidity is considered to be principally abnormal dietary pattern behavior. ADHD sufferers have diminished alertness and an increased prevalence of sleep disorders, with the greater likelihood of energy dense food choice, notably that of fast food, rather than healthful alternatives and those prepared by their own families.[21] Moreover, the Decreased alertness results in a delay in food satisfaction, disordered eating, such as not eating breakfast,[21] overeating, more snacking , sedentary behaviour and excessive screen time.[22-24] ADHD can also be associated with 'stress overeating',[25] a further contributor to overfatness in ADHD.

That ADHD may be familial, although not necessarily genetic, is of interest on account of shared and transmitted socioeconomic circumstances and behaviours, even with epigenetic expression.[26-28] In 2017,[29] Chen et al conducted a family aggregation study on ADHD prevalence, education, comorbidities (including drug abuse, anxiety, depression) and the associated overweight/obesity prevalences among Swedish servicemen and their offspring, finding that overfatness and ADHD have a shared familial risk whose specification is unclear. Insofar as genomic explanations are concerned, the overweight obesity and ADHD comorbidity may depend on such as the rs805013 fragment in FTO (obesity gene) as a mediator of the pathogenesis of ADHD.[30] The DRD4 gene is also involved in the pathogenesis of ADHD and increases the risk of obesity.[31] Low-birth-weight (IUGR) is not only a risk factor for ADHD but also for obesity.[32] Thus, it can be inferred that ADHD and obesity have a common genetic predisposition towards a phenotype in fetal programming.[33] In regard to growth retardation,[11] children with ADHD and its pharmacotherapy have relative prepubertal shortness, but this may not be apparent until adulthood, or not at all given the possibility of catch-up growth, delayed maturity and the resolution of underlying ADHD.[34] Indeed, it is known that height related emotional and learning differentials can disappear dependent on family circumstances and educational opportunity.[35] These observations can temper the implications of the findings in the present study of associations between ADHD and stature. Most importantly are the several co-existent morbidities, any one or several of which could have interrupted or stalled growth temporarily or permanently. To this must be added the contribution of socioeconomic factors, care and nutrition to neurodevelopment which exceeds those for linear growth, but are of functional consequence.[35,36] While short stature and associated morbidities may prompt nutritional assessment and intervention, its accelerated velocity and maximization ought not be the primary objective Whether linear growth and body fatness are interactive with the ADHD comorbidities for ADHD expression and severity is of pathogenetic relevance. Without food and nutrient intake data in this population, the question of food and nutrient therapies is mute, but the safest nutrition approach will be that of dietary pattern with attention to quality by way of biodiversity.[37,38] Specific nutrient interventions without evidence of deficiency would not satisfy a risk-benefit analysis.[39]

Drug therapy has been the first choice for children over 6 years old with ADHD. Central stimulants are used for children with ADHD. Their side effects are mainly manifest in early with loss of appetite, weight loss, reduced growth velocity and disordered sleep, which are mostly clinically manageable and tolerated.[40] However, the negative media reports and the stigma surrounding the social attitude towards the use of these drugs make it difficult for parents to accept medical treatment,[41] especially for children with ADHD who are stunted and wasted, so that parents decline the drugs and discontinue their use at an early stage.[42-44] A longitudinal community study in Australia followed up children with ADHD and found that the stimulant use was only 13.6% among children younger than ten-years-old, but increased to 25.6% thereafter.[45] Children with ADHD experience emaciation and growth retardation before the age of 10. But medication usage is planned long term when the nutritional problems of weight loss and growth retardation become increasingly problematic for clinicians and parents.[46] The European guidelines for the management of adverse drug reactions (ADRs) associated with ADHD suggest that body weight and height should be monitored every six months during drug treatment. If the growth rate is impaired, food therapy with energy dense and nutritious snacks and meals, rather than stopping the drug, is recommended. The nutrition management of children with ADHD is regulated in some jurisdictions.[47] As shown in Table 3, the risk of ADHD is increased in children with premature birth, allergic diseases, otitis media, tonsil or adenoid hypertrophy, gastroesophageal reflux, and poor sleep quality, consistent with other reports.[6,33] Not only are patients with the identified comorbidities prone to nutritional problems, but some evidence points to food or nutrient deficiency as contributory to ADHD itself.[48] Thus, dietary management is likely to need individualisation and professional clinical nutrition input with diet planning and supplementation as appropriate.[49] Recourse has been sometimes found justifiable for polyunsaturated fatty acid and vitamin D

supplements and particular attention to diets replete with zinc, iron and magnesium, apparently conducive to improvement in clinical symptomatology and overall nutritional status in children with ADHD.[50] The nutritional management of children with ADHD is demanding for clinicians, parents and child.

Limitations

Hierarchical cluster sampling for participants in this study of ADHD accessed more than 20,000 school-age children in different schools in 9 major cities in China. While the sample size was large with good overall representativeness, it does not necessarily reflect the overall distribution of ADHD and the associated nutritional problems, especially as they relate to similar if less well-defined health problems. Diagnosis ultimately depended on parental recall and communication which is inevitably imperfect Secondly, that problems were nutritional depends on the presumption that premature birth, height and body composition reflect, respectively, maternal diet and intrauterine nutrition in the first case, and the child's diet and nutritional biology in the second and third cases. Thirdly, this study is cross-sectional study so that associations may suggest, but not assure that any are those of cause and effect.

Conclusions

The nutritional status of school-age children with ADHD in China and elsewhere is complex, but most evident by an increased prevalence of short stature which may, in part, be attributable to dietary factors; and in overweight and obesity, a reflection of energy dysregulation. This is accompanied by an increased history of premature birth, immunoinflammatory and allergic disease, pharyngo-respiratory disease, gastroesophageal reflux, and poor sleep quality. Separately and together, these problems are likely to exacerbate any nutritional disorder and create a spiral of ill-health. The association with premature birth points to the critical importance of policies which address the first 1000 days or life from conception as crucial in the nutritional optimisation of human development. The prospects for better health in children at risk of and affected by ADHD may be advanced by greater regard to their nutritional biology and care.

AUTHOR DISCLOSURES

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