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Original Article

Serum nitric oxide and acetylcholinesterase levels in children with functional dyspepsia and their relationship with nutritional status

Yuan Cheng, Qingfeng Fang, Biquan Chen

Department of Infectious Diseases, Anhui Provincial Children's Hospital, Hefei 230022, Anhui, China

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Abstract: Objective: To evaluate the serum levels of nitric oxide (NO) and acetylcholinesterase (AchE) in children with functional dyspepsia (FD) and their association with nutritional status. Methods: Clinical data from 165 children with FD attending Anhui Provincial Children's Hospital were retrospectively reviewed. Serum NO, AchE, pre-albumin (PA), transferrin (TRF), and retinol-binding protein (RBP) were measured. Correlations between serum NO, AchE, PA, TRF and RBP were analyzed. Risk factors for malnutrition in children with FD were identified. Results: Among the 165 children, 108 had normal nutrition, 18 had overnutrition, and 39 had malnutrition. The serum NO level in the malnutrition group was much higher than those in normal nutrition group and over nutrition group, while AchE, PA, TRF and RBP were much lower ($P < 0.05$ for all). In the malnutrition group, serum NO level was negatively correlated with PA (r = -0.825, $P = 0.019$), TFN (r = -0.781, $P = 0.007$), and RBP (r = -0.799, $P = 0.005$), whereas serum AchE was positively correlated with PA (r = 0.741, $P = 0.022$), TFN (r = 0.762, $P = 0.011$), and RBP (r = 0.783, $P = 0.030$). Age, *Helicobacter Pylori* (HP) infection, and living with parents were identified as influencing factors for malnutrition, with the area under the curves (AUCs) of 0.602, 0.768, 0.633, respectively. Conclusion: Serum NO and AchE levels are closely associated with nutritional status in children with FD. HP positivity shows predictive value for malnutrition and may serve as a potential target for clinical intervention.

Keywords: Functional dyspepsia, nitric oxide, acetylcholinesterase, nutritional status, risk factors, correlation

Introduction

Functional dyspepsia (FD) is a prevalent gastrointestinal disorder in children [1]. It has a significant impact on nutritional status, growth, and development in affected children. A large-scale population study in China reported an FD prevalence of 5.9%, accounting for 16% of all functional gastrointestinal disorders (FGIDs) based on the Rome IV criteria [2]. Current treatment is mainly clinical and includes acid suppressants, gastric mucosal protectants, prokinetic agents, digestive enzyme preparations, and other symptomatic treatments [3, 4]. However, their therapeutic efficacy is suboptimal due to the single-target mechanism of action, high recurrence rate, and adverse effects associated with long-term medication [5]. The etiology and pathophysiologic mechanisms of FD remain unclear and may involve a variety of factors.

According to the latest international and national consensus, FD is a condition that arises from aberrant gut - brain interactions [6], encompassing disturbances in gastrointestinal dynamics, gut microbiota, and central nervous system (CNS) regulation [7]. Recent evidence suggests that nitric oxide (NO) may play a regulatory role in gut microbiota [8]. NO is a biologically active molecule involved in many physiological and pathological processes. In the digestive tract, NO can relax smooth muscle, regulate gastrointestinal motility, and inhibit gastric acid secretion [9]. In children with FD, abnormal NO metabolism may contribute to impaired gastrointestinal motility and delayed gastric emptying, thereby aggravating dyspeptic symptoms [10]. Acetylcholinesterase (AchE), an enzyme that degrades acetylcholine, regulates contraction and relaxation of gastrointestinal smooth muscles [11]. Altered AChE activity is associated with disordered gastrointestinal motility and may affect digestive tract function [12]. Al-

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Table 1. Nutritional status of children ≥ 6 years (kg/m^2)

Gender	Age	Malnutrition	Normal nutrition	Overnutrition
Male	6	< 13.6	13.6-18.0	> 18.0
	7	< 13.9	13.9-18.8	> 18.8
	8	< 13.9	13.9-19.7	> 19.7
	9	< 13.9	13.9-19.7	> 19.7
	10	< 14.4	14.4-22.5	> 22.5
	11-12	< 15.1	15.1-22.5	> 22.5
Female	6	< 13.9	13.9-18.4	> 18.4
	7	< 13.9	13.9-18.4	> 18.4
	8	< 14.1	14.1-19.0	> 19.0
	9	< 14.4	14.4-20.4	> 20.4
	10	< 14.9	14.9-21.2	> 21.2
	11-12	< 15.5	15.5-22.8	> 22.8

though previous studies have examined the nutritional status and biochemical parameters in children with FD, this study is the first to systematically analyze the associations between serum NO, AchE, and nutritional biomarkers - including pre-albumin (PA), transferrin (TRF), and retinol-binding protein (RBP) - in different nutritional subgroups. Moreover, by incorporating *Helicobacter pylori* (HP) infection status and family environment factors, this study explores the multidimensional mechanisms underlying malnutrition in children with FD, providing novel insights and potential targets for clinical intervention.

Methods

Participant enrollment

Clinical data of 165 children with FD who attended the outpatient department of Anhui Provincial Children's Hospital between January 2020 and June 2024 were retrospectively analyzed. Based on the criteria for children's nutritional status [13], participants were divided into malnutrition, normal nutrition, and overnutrition groups. According to the literature [14], for children younger than 6 years, nutritional status was classified as follows: body mass index (BMI) < 15 kg/m^2 as malnutrition, BMI 15-18 kg/m^2 as normal nutrition, and BMI > 18 kg/m^2 as overnutrition; For children aged ≥ 6 years, the specific criteria are shown in **Table 1**. This study was approved by the Medical Ethics Committee of the Anhui Provincial Children's Hospital.

Inclusion criteria: Patients who met the Rome IV diagnostic criteria for FD [14], with at least one of the following symptoms: postprandial fullness, epigastric pain, early satiety, and epigastric burning. Episodes of postprandial fullness or early satiety occurring 3 d/week were included, provided they were not relieved by defecation or bowel movement; Episodes of epigastric pain and epigastric burning occurring ≥ 1 d/week, persisting for > 6 months, and present within the last 3 months were required. All patients underwent detailed history-review and physical examination to exclude organic diseases, gallbladder or sphincter of Oddi dysfunction, and other identifiable causes of abdominal pain. Eligible participants had not taken medications affecting gastrointestinal function and had complete medical records.

Exclusion criteria: Children with combined cardiac, hepatic, or renal dysfunction; severe infectious diseases; psychiatric disorders; organic gastrointestinal diseases; tumors or other immune system disorders; or respiratory diseases such as bronchial asthma and tuberculosis.

Methods

Peripheral venous blood was collected from all participants. Samples were centrifuged 2000 r/min at low temperature for 15 min. Serum NO levels were detected by nitrate reductase method using a commercial kit (Shanghai Yaji Biotechnology Co., Ltd., China). Serum AchE levels were detected by colorimetry using a kit from Wuhan Aimie Technology Co., Ltd., China. Nutritional biomarkers, including PA, TRF and RBP, were quantified using a fully automated biochemical analyzer (COBAS C702, Roche, Switzerland) using immunoturbidimetric assays, with reagents supplied by Tiangen Biochemical Technology Co., Ltd., China.

Observational indicators

Laboratory indicators: Serum NO (μmol/L) by nitrate reductase method (detection limit: 0.1 μmol/L, intra-assay CV < 5%); AchE (U/L) by colorimetry (linear range: 5-200 U/L); PA/TRF/RBP by immunoturbidimetry (Roche Cobas C702). Clinical data included: (1) Demographics: age, gender; (2) Lifestyle factors: sleep

Table 2. Details of symptoms in children with FD

Symptoms, n (%)	Present	Mild	Moderate	Severe
Epigastric pain	164 (99.4)	114 (69.5)	48 (29.3)	2 (1.2)
Bloating	45 (27.3)	42 (93.3)	3 (6.7)	0
Postprandial fullness	62 (37.6)	50 (80.6)	11 (17.7)	1 (1.6)
Early satiety	90 (54.5)	57 (63.3)	33 (36.7)	0
Nausea	58 (35.2)	55 (94.8)	2 (3.4)	1 (1.7)
Non-bilious vomiting	41 (24.8)	38 (92.7)	3 (7.3)	0
Belching	83 (50.3)	51 (61.4)	32 (38.6)	0
Epigastric burning	65 (39.4)	51 (78.5)	14 (21.5)	0

Abbreviation: FD, functional dyspepsia.

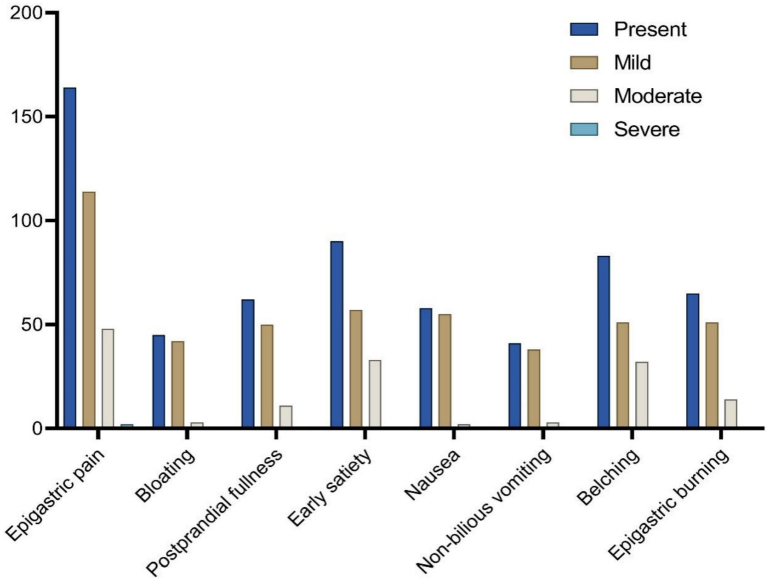

Figure 1. Details of symptoms in children with FD. Abbreviation: FD, functional dyspepsia.

duration (hours/night), breakfast frequency (≤ 50% or > 50% days/week); (3) Socioeconomic factors: family income (≤ 8000 or > 8000 yuan/month), parental cohabitation status.

Statistical analysis

All statistical analyses were performed using SPSS 26.0 software. Continuous variables with normal distribution were expressed as mean ± standard deviation (SD). One-way analysis of variance was used for comparisons among three groups, while independent sample t-tests were used for comparisons between two groups. Count data (n, %) were analyzed using chi-square test.

Pearson correlation analysis was used for correlation analysis. To identify risk factors for malnutrition in children with FD, malnutrition status (yes/no) was defined as the dependent variable. Variables with $P < 0.05$ in the univariate analyses were entered into a binary logistic regression model to calculate odds ratios (ORs) and 95% confidence intervals (95% CIs). The discriminatory ability of predictors was assessed using receiver operating characteristic (ROC) curve, with an area under the curve (AUC) > 0.7 considered clinically significant. All statistical tests were two-tailed, and $P < 0.05$ was considered statistically significant.

Results

Symptoms of FD

The symptoms of FD in children are summarized in **Table 2** and **Figure 1**. Most symptoms were of mild to moderate intensity. Epigastric pain (99.4%) and early satiety (54.5%) were the most frequently reported.

Comparison of serum NO, AchE, PA, TRF and RBP levels among three groups

Among the 165 children, 108 cases were classified as normal nutrition, 18 as over-nutrition, and 39 of malnutrition. As shown in **Table 3**, serum NO levels were significantly higher in the malnourished group than in the normal and overnutrition groups ($P < 0.05$). Conversely, AchE, PA, TRF, and RBP were significantly lower in the malnutrition group than in the other two groups ($P < 0.05$).

Correlation between serum NO, AchE and nutritional markers

As shown in **Table 4**, in the malnutrition group, serum NO levels were negatively correlated with PA ($r = -0.825$, $P = 0.019$), TFN ($r = -0.781$, $P = 0.007$), and RBP ($r = -0.799$, $P = 0.005$). In contrast, serum AchE was positively correlated with PA ($r = 0.741$, $P = 0.022$), TRF ($r = 0.762$, $P = 0.011$), and RBP ($r = 0.783$, $P = 0.030$).

Table 3. Comparison of serum NO, AchE, PA, TRF, and RBP among three groups of children with FD

	Normal nutrition	Overnutrition	Malnutrition	*F*	*P*
NO (μmoL/L)	46.32±9.17[a]	46.45±9.21[a]	182.62±25.41	128.012	< 0.001
AchE (U/L)	132.85±25.57[a]	133.07±26.83[a]	74.00±15.43	91.663	< 0.001
PA (mg/dL)	18.10±3.67[a]	18.20±3.82[a]	6.19±1.97	186.756	< 0.001
TRF (g/L)	2.51±0.47[a]	2.54±0.45[a]	0.84±0.20	236.494	< 0.001
RBP (μg/mL)	27.32±5.33[a]	27.18±5.41[a]	11.04±1.69	174.943	< 0.001

Abbreviation: NO, nitric oxide; AchE, Acetylcholinesterase; PA, pre-albumin; TRF, transferrin; RBP, retinol binding protein; FD, functional dyspepsia. Note: compared with malnutrition group, [a]$P < 0.05$.

Table 4. Correlation analysis between serum NO, AchE levels with nutrition-related factors

	PA		TFN		RBP	
	r	*P*	*r*	*P*	*r*	*P*
NO	-0.825	0.019	-0.781	0.007	-0.799	0.005
AchE	0.741	0.022	0.762	0.011	0.783	0.030

Abbreviation: NO, nitric oxide; AchE, Acetylcholinesterase; PA, pre-albumin; TRF, transferrin; RBP, retinol binding protein.

Comparison of general and clinical characteristics among groups

As shown in **Table 5**, significant differences were found between the malnutrition and normal nutrition groups in age (t = -2.215, P = 0.028), sleep duration (t = -3.059, P = 0.003), HP infection (χ^2 = 34.212, $P < 0.001$), parental cohabitation status (χ^2 = 8.466, $P < 0.001$), and monthly family income (χ^2 = 8.932, $P < 0.001$).

Logistic regression analysis of influence factors of malnutrition in FD children

Logistic regression analysis revealed that age [OR = 1.826, 95% CI: 1.129-2.953] and HP infection [OR = 9.969, 95% CI: 3.685-26.967] were risk factors for malnutrition in children with FD, while parental cohabitation [OR = 0.338, 95% CI: 0.133-0.860] was a protective factor (**Table 6**).

ROC analysis of predictive factors

ROC analysis demonstrated that AUC was 0.602 for age, 0.768 for HP infection, and 0.633 for parental cohabitation in predicting malnutrition in FD children. Among these, HP positivity had the highest predictive value, with a sensitivity of 79.5% and specificity of 74.1% (**Table 7** and **Figure 2**).

Discussion

In this study, children with FD and malnutrition exhibited significantly higher serum NO levels and lower AchE, PA, TRF, and RBP levels compared with those in the normal and overnourished groups. These findings suggest that malnutrition is prevalent among FD children, and is characterized by elevated NO and reduced AchE and nutritional biomarkers.

NO is a small, non-adrenergic, non-cholinergic neurotransmitter in the gastrointestinal system and serves as a key inhibitory mediator of gastrointestinal motility [15]. Elevated NO levels indicate dysfunction of the nitrergic neural pathways, leading to an imbalance between excitatory and inhibitory signals in the gut. This imbalance can cause motility disorders and exacerbate functional dyspepsia [16, 17]. Previous studies have reported that higher serum NO levels in FD patients are associated with greater impairment of neural pathways and more severe disturbances in gastrointestinal motility, which may contribute to malnutrition [18-20]. Acetylcholinesterase (AchE) hydrolyzes acetylcholine, and reduced AchE activity decreases acetylcholine breakdown, slowing gastrointestinal motility and delaying gastric emptying, thereby aggravating FD symptoms in kids [21]. Prealbumin (PA), synthesized in the liver, is a plasma transport protein that carries thyroid hormones and vitamin A. Reduced PA levels reflect decreased protein intake and serve as a sensitive biochemical marker for nutritional assessment in children [22]. Transferrin (TRF), also primarily synthesized in the liver, transports iron to red blood cells for hemoglobin synthesis. Poor nutritional status

Table 5. Comparison of general and clinical information between the malnutrition group and normal nutrition group

Factors	Malnutrition (n = 39)	Normal nutrition (n = 108)	t/x^2	*P*
Gender				
Male	21 (53.8)	64 (59.3)	0.344	0.627
Female	18 (46.2)	44 (40.7)		
Age (year)	7.85±1.09	8.30±1.09	-2.215	0.028
Breastfeeding				
Yes	33 (84.6)	83 (76.9)	1.038	0.270
No	6 (15.4)	25 (23.1)		
Breakfast frequency				
≤ 50%	11 (28.2)	18 (16.7)	2.409	0.234
> 50%	28 (71.8)	90 (83.3)		
Sleep time (h)	7.85±1.01	8.34±0.81	-3.059	0.003
Helicobacter Pylori positivity				
Yes	31 (17.95)	28 (80.56)	34.212	< 0.001
No	8 (82.05)	80 (19.44)		
Parental cohabitation				
Yes	16 (79.49)	73 (15.74)	8.466	< 0.001
No	23 (20.51)	35 (84.26)		
Parental divorce				
Yes	15 (61.54)	30 (30.56)	1.540	0.305
No	24 (38.46)	78 (69.44)		
Monthly family income (yuan)				
≤ 8000	19 (48.7)	25 (28.70)	8.932	< 0.001
> 8000	20 (51.3)	83 (71.30)		

Table 6. Logistic regression analysis of factors influencing the occurrence of malnutrition in children with FD

Influence factor	β	SE	Wald	*P*	OR	95% CI
Age	0.602	0.245	6.023	0.014	1.826	1.129-2.953
Sleep time	0.435	0.251	3.007	0.083	1.546	0.945-2.529
Helicobacter Pylori positivity	2.300	0.508	20.513	< 0.001	9.969	3.685-26.967
Parental cohabitation	-1.085	0.477	5.179	0.023	0.338	0.133-0.860
Monthly family income	-0.613	0.486	1.590	0.207	0.542	0.209-1.404

Abbreviation: FD, functional dyspepsia; SE, standard error; OR, odds ratio; CI, confidence interval.

Table 7. ROC analysis

Influence factor	AUC	Sensitivity	Specificity	95% CI
Age	0.602	45.4%	71.8%	0.500-0.704
Helicobacter Pylori positivity	0.768	79.5%	74.1%	0.680-0.856
Parental cohabitation	0.633	67.6%	59.0%	0.529-0.736

Abbreviation: ROC, receiver operating characteristic; AUC, area under the curve; CI, confidence interval.

impairs hepatic TRF synthesis, leading to lower serum TRF levels in malnourished individuals [23]. Retinol-binding protein (RBP) is a carrier of retinol and retinoic acid in serum and cells. RBP levels decrease rapidly in malnourished individuals and are frequently utilized as a specific marker of nutritional status [24]. Thus, children with FD and malnutrition exhibit elevated serum NO levels and decreased AchE, PA, TRF and RBP levels. Moreover, NO and AchE levels are closely correlated with these nutritional biomarkers, indi-

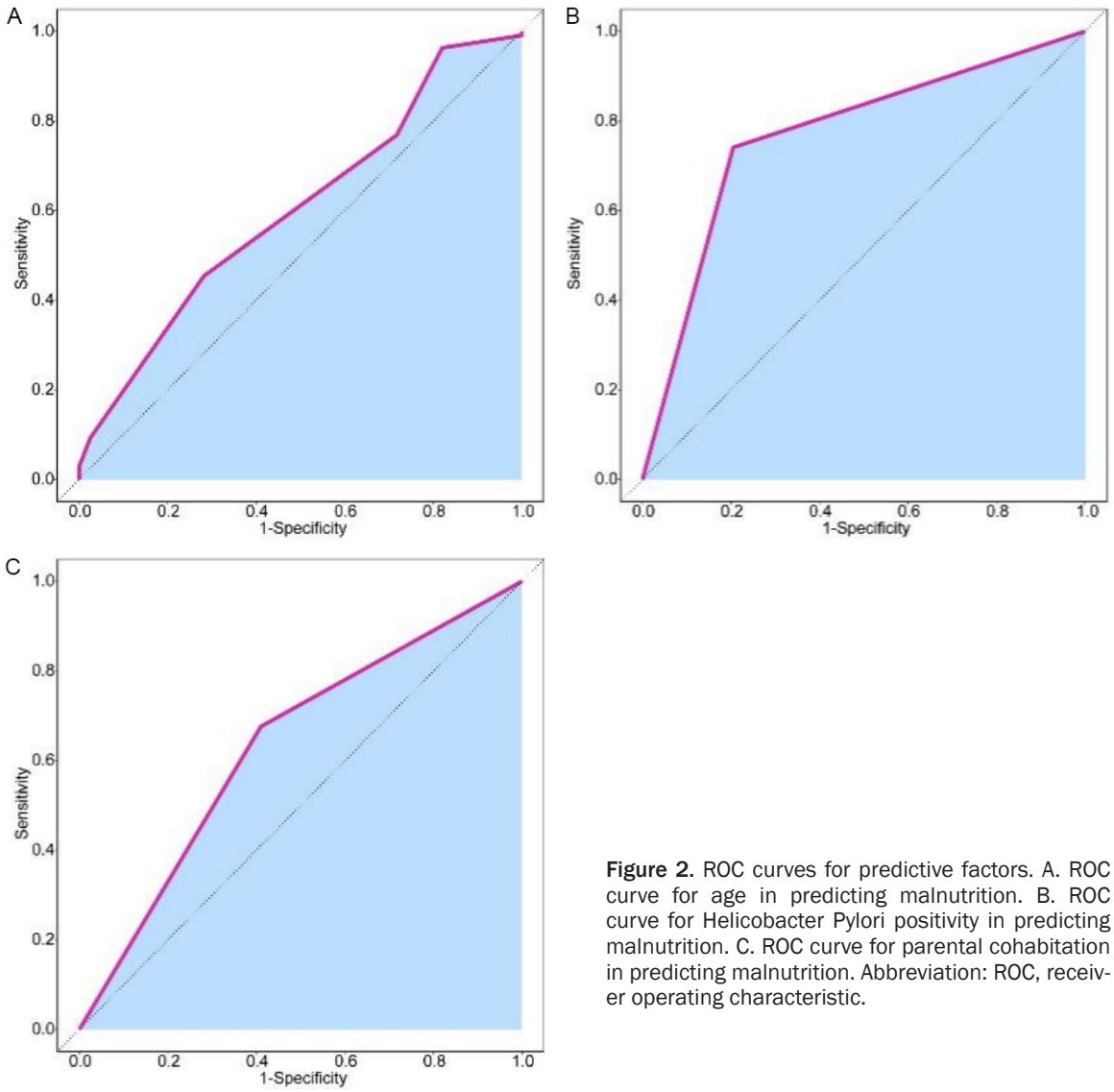

Figure 2. ROC curves for predictive factors. A. ROC curve for age in predicting malnutrition. B. ROC curve for Helicobacter Pylori positivity in predicting malnutrition. C. ROC curve for parental cohabitation in predicting malnutrition. Abbreviation: ROC, receiver operating characteristic.

cating their potential role in the pathophysiology of malnutrition in FD.

The study revealed that serum NO levels were negatively correlated with PA, TFN, and RBP, whereas serum AchE levels were positively correlated with these nutritional biomarkers. These findings indicate a close association between serum NO and AchE levels and nutritional status in children with FD. The etiology of FD among children is multifactorial. Li et al. reported that FD children often exhibit compromised immune function, which lowers their disease resistance, making them more vulnerable to infections [25]. Such immune compromise may disrupt metabolism and impair gastrointestinal, hepatic, and renal function, leading to elevated serum NO levels and decreased AchE, PA, TFN and RBP levels, which may hinder nutrient absorption and contribute to malnutrition. Therefore, monitoring and managing the levels of NO, AchE, PA, TFN, and RBP levels within the normal range is important for improving nutritional status in children with FD.

Furthermore, this study showed that age and HP positivity were risk factors for malnutrition in FD children, whereas living with parents was a protective factor. Notably, HP infection demonstrated high predictive value for malnutrition in this population. HP infection is a well-established risk factor for various gastrointestinal disorders, including FD, and our results reinforce its role in the development of malnutrition in affected children [26]. Previous studies have demonstrated that HP infection can cause gas-

tric inflammation, alter gastric acid secretion, and impair nutrient absorption, all of which contribute to malnutrition [27, 28]. In particular, HP infection may disrupt normal gastric motility and acid-base balance, exacerbating FD symptoms and further compromising nutritional status [29, 30]. The present finding that HP positivity is a strong predictor of malnutrition is supported by the aforementioned reports, suggesting that HP eradication should be considered as part of the management plan for children with FD who are at risk of malnutrition. In addition, parental cohabitation was identified as a significant protective factor. This protective effect may be related to enhanced family support, dietary supervision, and emotional stability, all of which are known to positively influence nutritional status in children with chronic gastrointestinal disorders. However, this association may be affected by unadjusted confounding factors, such as family economic level, parental involvement in dietary habits, and the wide confidence interval warrants cautious interpretation. Future research should investigate its independent effect and underlying mechanism through stratified or path analysis.

This study has several limitations. Measurement of NO and AchE levels in the gastric and duodenal mucosa requires biopsy, which is invasive and does not meet the ethical requirements of our hospital. Therefore, this study did not explore the relationship between mucosal NO and AchE levels and nutritional status in children with FD. Future studies, including animal experiments may help address this issue, providing deeper insights into the mechanisms of malnutrition in FD and guiding the development of targeted therapeutic strategies.

Disclosure of conflict of interest

None.

Address correspondence to: Biquan Chen, Department of Infectious Diseases, Anhui Provincial Children's Hospital, No. 39 Wangjiang East Road, Baohai District, Hefei 230022, Anhui, China. Tel: +86-0551-62237114; E-mail: chengyi579593@163.com

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