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Original Article

Cognitive performance of 4 to 6-year-old children: a longitudinal study

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Abstract

Background Many children in low and middle-income countries (LMIC) have reduced cognitive abilities, but few studies have explored the influencing factors.

Objective To determine the determinants of children's low cognitive development at 4 to 6- year-old.

Methods This is a retrospective cohort study in year 2021. The data was obtained from *Bogor Longitudinal Study of Child Growth and Development* (BLSCGD) that started from year 2012. For this analysis, we considered 165 of children aged 4-6 years. The dependent variable in this analysis was cognitive development as measured by the *Indonesian Wechsler Preschool and Primary Scale of Intelligence* (WPPSI) with the indicators of full-scale IQ (FSIQ), verbal IQ (VIQ), and performance IQ (PIQ). The independent variables were determinants of children's cognitive development at 4-6 years, consisted of socio-demographic factors, determinants from early life (0-23 months), as well as determinants from current conditions. Multivariate analysis done with the logistic regression test.

Results Bivariate analysis revealed that paternal education level was significantly associated with FSIQ and inadequate calorie intake in infancy and poor child stimulation were significantly associated with VIQ. In the final model of multivariate analysis, low paternal education level retained a significant association with FSIQ. Psychosocial stimulation was significantly associated with VIQ and inadequate calorie intake during infancy was significantly associated with PIQ.

Conclusion Low paternal education level and inadequate psychosocial stimulation are risk factors for reduced child cognitive development in 4-6-year-olds. Inadequate calorie intake in infancy even though not statistically significant it has high OR values for low VIQ and PIQ, thus the results showed the importance of calory intake in infancy. **[Paediatr Indones. 2023;63:65-72; DOI: https://doi.org/10.14238/pi63.2.2023.65-72]**.

Keywords: *determinant factors; children; cognitive development; longitudinal study*

Every child has the right to optimal growth and development.[1] However, there are still many children in LMIC unable to reach their optimal potential.[2] Indonesia is one of 34 countries with a 60% or higher prevalence of children at risk of poor development.[3]

Children whose cognition does not develop optimally, tend to have poorer school performance as well as negative impacts later in life. As adults, they may attain only low-income levels, have high fertility rates, and continue the process of providing less than optimal care of their own children.[2] Several factors have been identified to influence child cognitive development, such as biological factors (e.g., birth weight, nutrition, and morbidity), socioeconomic factors (family income, level of education, and social class), environmental factors (e.g., home environment, providing appropriate toys for children, and access

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to health facilities), and psychosocial factors (e.g., parental mental health, child-parent interactions, cognitive stimulation, and opportunity to study).[2]

Several studies also mentioned that factors during early life could determine future child cognitive development. The importance of the first 1,000 days (fertilization to 24 months of age), especially the "critical and sensitive period" of brain development, have already been discussed and reviewed by many experts in neuroscience.[3] Brain development takes place immediately after conception and continues throughout childhood into adolescence and early adulthood. Environmental influences can alter both the structure and function of the developing brain.[4] Brain development is modified by the quality of the environment. Animal studies have shown that nutrition, iron deficiency, environmental toxins, stress, as well as poor stimulation and social interaction can affect brain structure and function.[5]

Despite the importance of current situation regarding children's cognitive abilities especially among children in LMIC, the potential risk factors of reduced child cognition still need to be explored. Thus, we aimed to determine potential associations between risk factors with children 4 to 6-year-old cognitive development.

Methods

This is a retrospective cohort study in the year 2021. The data was obtained from *Bogor Longitudinal Study of Child Growth and Development* (BLSCGD), Indonesia, conducted by *National Institute of Health Research and Development* (NIHRD, MoH) started from year 2012, and children were followed from birth. Bogor is a city that located near the capital city of Indonesia, Jakarta, and have characteristics of urban community. From total of 1054 children as the subjects of BLSCG in 2021, 540 were born between 2015-2017. From this number of subjects, after checking of the completeness of secondary data from the BLSCGD study, the data of children was available starting from the time of the pregnancy and follow up in monthly. As many as 71 exposed and 94 non exposed group are randomly selected from the list of subjects that met the inclusion and exclusion criteria (total 165 subjects). The inclusion criteria of the exposed group included: had severe linear growth failure and aged 4-6 years at the cognitive assessment, while exclusion criteria were having congenital defects nor congenital abnormalities. The inclusion criteria of the non-exposed group included: having normal linear growth and aged 4-6 years at the cognitive assessment, while exclusion criteria were having congenital defects nor congenital abnormalities.

Socio-demographic information was collected from the beginning of subject recruitment through interviews during home visits. The subjects were recruited since the mother was identified as having pregnancy. Subjects' birth weights were noted. Food intake was monitored monthly using 24-hr food recall.[6] At subjects' follow-up visits at the age of 4-6 years, we collected data on cognitive development, maternal mental health, current child psychosocial stimulation, and exposure to formal/non-formal education. All variables other than cognitive development were collected through home visit interviews, while cognitive development was evaluated at the BLSCGD basecamp.

The primary outcome of this analysis was cognitive development at 4-6 years as measured by the *Indonesian Wechsler Preschool and Primary Scale of Intelligence* (WPPSI).[7] The manual for the Indonesian version of the WPPSI was issued by the University of Indonesia Faculty of Psychology. The WPPSI test consisted of two scales, namely, verbal and performance. The final results were in the form of full-scale IQ, verbal IQ, and performance IQ. Verbal IQ (VIQ) was a measure of acquired knowledge, verbal reasoning, comprehension, and attention to verbal stimuli. It consisted of 5 subtests, which were information, vocabulary, arithmetic, similarity, and comprehension. Performance IQ (PIQ) was a measure of fluid reasoning, spatial processing, attentiveness to detail, and visual-motor integration. The scale consisted of five subtests (animal house, picture completion, mazes, geometric design, and block design). Full-scale IQ was a combination of verbal and performance IQs. IQ scores were categorized as normal (IQ $\geq$ 80) or low (< 80) cognitive ability.[8]

The potential predictors of reduced cognition were categorized into socio-demographic factors, exposures from early life (0-23 months), as well as exposures from current conditions. There were six variables under socio-demographic information: child gender, age of child at cognitive assessment, paternal and maternal education level and occupation. Education level was defined as the highest level completed and categorized into "Low" (completed up to junior high school) and "Middle and High" (completed above junior high school/high school or college). Paternal occupation was categorized into civil servant, private employee, entrepreneur, and laborer/other job. Maternal occupation was categorized into stay at home mother or working outside the home.

Exposures from early life consisted of birth weight and dietary intake [calorie, macronutrients (protein and fat), and micronutrients (Fe and zinc)]. Birth weight was measured within 24 hours of birth and categorized as low (< 2,500 g) and normal (≥ 2,500 g). Dietary intake was measured in the periods of infancy (0-11 months) and early childhood (12-23 months) and averaged. Adequate energy intake was defined as 70% of the *Recommended Daily Allowance* (RDA) and macronutrients as 80% of the RDA.[9] Adequate iron and zinc were defined as the *Estimated Average Requirement* (EAR) values mentioned in the *2018 Widyakarya Nasional Pangan dan Gizi* (WNPG).[10]

Exposures from current conditions included psychosocial stimulation, maternal mental health, and exposures to formal/informal education. Child psychosocial stimulation was obtained through interviews and observations using the *HOME Observation for Measurement of the Environment Inventory* (HOME Inventory)[11] for children aged 3-6 years. Scores were categorized into good (score > 80%), average (score 61%-80%), and poor (score ≤ 60%).[12] Maternal mental health was assessed using the *Self Rating Questionnaire* (SRQ-20), which consisted of 20 questions. Each of the 20 items was scored 0 or 1.13 A score of 1 indicated that the symptom was present during the previous month. If subjects answered "yes" to at least six questions, then they were considered to be experiencing stress or mental-emotional disorders.[13] Exposures to formal/informal education were categorized into received education ≥ 12 months, received education 1-11 months, and never received education.

Interviews were conducted by trained enumerators with backgrounds in health education, using pre-tested questionnaires. Cognitive tests were done by a certified psychologist and limited to only five children per day, as the examination time took 60 to 90 minutes per child. Interrater reliability test for cognitive assessment was performed before data collection; the intraclass correlation coefficients (ICC) showed that the scoring between raters was reliable (ICC > 0.95).

Descriptive, bivariate (Chi-square), and multivariate logistic regression analyses were used to assess the factors potentially influencing children's cognitive abilities. The statistical analysis was performed using *SPSS version 21*.

Parents provided written informed consent. This study was approved by the Ethics Commission of the Universitas Indonesia, Faculty of Medicine.

Results

The characteristics of subjects are described in **Table 1**. There were more female than male subjects. Around 15% of parents had low education level. More than half of fathers had occupations as laborers/other jobs, while most mothers were stay at home mothers.

Almost 10% of the infant subjects had low birth weight. During infancy, almost 20% had inadequate fat intake, 80% had inadequate iron intake, and almost half had inadequate zinc intake. In early childhood, protein, fat, iron, and zinc intake was not a problem in most subjects. During childhood, more than one-third of subjects had inadequate calorie intake, and more than 20% had inadequate fat and iron intake.

More than one-third of subjects had received poor psychosocial stimulation. For psychosocial factors, almost one-third of the children had never received formal/informal education. In addition, 6.1% of mothers had impaired maternal mental health.

Table 2 shows the analysis of subject characteristics and cognitive development (FIQ, VIQ, and PIQ). Low paternal education level had a significant association with FIQ (OR 2.7; 95%CI 1.0 to 7.1; P=0.043). Inadequate calorie intake in infancy and poor child stimulation were significantly associated with VIQ (OR 7.6; 95%CI 1.0 to 60.6; P=0.025 and OR 22.1; 95%CI 2.4 to 200.7; P=0.001, respectively).

We included all factors that had P values < 0.25 in the multivariate analysis. In the final model of multivariate analysis, low paternal education level retained a significant association with lower FSIQ (OR

Table 1. Characteristics of subjects

Characteristics	(N=165)
Socio-demographic factors	
Gender, n(%)	
Male	77 (46.7)
Female	88 (53.3)
Age at cognitive assessment, n (%)	
45-59 months	63 (38.2)
60-79 months	102 (61.8)
Parental education level, n (%)	
Low paternal	25 (15.2)
Low maternal	29 (17.6)
Paternal occupation, n (%)	
Civil servant	4 (2.4)
Private employee	38 (23.0)
Entrepreneur	26 (15.8)
Laborer/other job	97 (58.8)
Maternal occupation, n (%)	
Stay at home mother	131 (79.4)
Works outside the home	34 (20.6)
Exposures from early life	
Low birth weight (LBW)	16 (9.7)
Inadequate calorie intake in infancy	13 (9.0)
Inadequate calorie intake in early childhood	57 (38.0)
Inadequate protein intake in infancy	17 (11.8)
Inadequate protein intake in early childhood	19 (12.7)
Inadequate fat intake in infancy	28 (19.4)
Inadequate fat intake in early childhood	36 (24.2)
Inadequate iron intake in infancy	115 (79.9)
Inadequate iron intake in early childhood	30 (20.1)
Inadequate zinc intake in infancy	70 (49.0)
Inadequate zinc intake in early childhood	25 (16.7)
Exposures from current conditions	
Poor child stimulation	61 (37.0)
Received formal/informal education	
≥ 12 months	27 (16.4)
10-11 months	90 (54.5)
Never received education	48 (29.1)
Maternal mental health disorder	10 (6.1)

2.7; 95%CI 1.0-7.1; P=0.049) compared to children with middle/high paternal education level (**Table 3**).

Table 4 shows that, in the final model of multivariate analysis, poor child stimulation (OR 13.5; 95%CI 1.4 to 127.9; P=0.023) were the determinant factor significantly associated with low VIQ. Inadequate calorie intake in infancy even though not statistically significant, a high OR value (OR 6.3; 95% CI 0.8-50.8) indicated have a significant risk of low VIQ among children.

Discussion

Education is one of indicators that construct socio-economic status (SES). Parental low education has been confirmed as one of the factors contributed to child cognitive development. One study found that maternal education and vocabulary levels of mothers were strong predictors of the cognitive development of young children.[14] Furthermore, the *Aberdeen Cohort Study* also found an association between paternal SES and child cognitive abilities, with paternal social class found to contribute to child intelligence.[15]

UNICEF stated that the causes of malnutrition and low development among children are interlinked, whereas socioeconomics was the underlying factor.[16] This is also in line with Lancet that mention socio economic factor as one of the risk factors of child cognitive.[5] Low parental education can lead to poor care and less than adequate home stimulation.[5] Furthermore, several neuroscience studies have noted relationships between cognitive development and SES. Stress, nutrition, prenatal factors, parent-child interactions, and cognitive stimulation in the home environment are mediating effects of SES on both brain structure and cognitive function in both human and animal models.[17,18]

Several cognitive functions are influenced by SES: language, verbal math, attention, executive function, and memory. In language abilities, SES correlated with vocabulary and grammar. Verbal math correlated with SES in the math domain.[19]

One present study that examined the neural mechanism using an event-related brain potential (ERP) measure of selective auditory attention according to different SES background, comparing response on simultaneously presented narrative stories. It showed that SES correlated with attentional control, working memory, visual and novelty (N2) event-related brain potential (ERP) and ERP suppression response of unattended channel.[20]

We found that psychosocial stimulation was significantly associated with VIQ. Consistent with these findings, many studies have found that cognitive stimulation plays a significant role in children's cognitive ability and academic achievement.[21] For example, an intervention study in South Africa aimed to educate mothers about sensitive and responsive parenting. The intervention had a significant impact

Table 2. Bivariate analysis of potential determinant factors and cognitive development scales of children aged 4 to 6 years (Pearson's Chi-square)

Variables	Full-scale IQ		Verbal IQ		Performance IQ	
	OR (95% CI)	P value	OR (95% CI)	P value	OR (95%CI)	P value
Socio-demographic factors						
Female	0.9 (0.5 to 1.7)	0.833	0.9 (0.5 to 1.9)	0.983	1.5 (0.8 to 2.8)	0.193
Age 60-79 months at cognitive assessment	1.6 (0.8 to 2.9)	0.166	1.5 (0.8 to 2.9)	0.196	1.4 (0.7 to 2.6)	0.314
Low paternal education level	2.7 (1.0 to 7.1)	0.043*	1.5 (0.6 to 3.8)	0.416	1.8 (0.8 to 4.4)	0.155
Low maternal education level	2.2 (0.9 to 5.3)	0.075	1.2 (0.5 to 2.9)	0.609	2.2 (1.0 to 4.9)	0.062
Paternal occupation Laborer/other job	1.6 (0.2 to 12.0)	0.486	2.1 0.3-15.8	0.739	0.8 (0.1 to 5.6)	0.771
Maternal occupation Stay at home mother	1.4 (0.7 to 3.2)	0.345	1.2 (0.5 to 2.6)	0.701	1.4 (0.7 to 3.0)	0.357
Exposures from early life						
LBW	0.9 (0.3 to 2.7)	0.910	2.5 (0.7 to 9.3)	0.148	0.8 (0.3 to 2.2)	0.638
Inadequate calorie intake in infancy	2.9 (0.8 to 11.0)	0.104	7.6 (1.0 to 60.6)	0.025*	2.3 (0.7 to 7.4)	0.158
Inadequate calorie intake in early childhood	1.1 (0.5 to 2.0)	0.978	1.3 (0.6 to 2.5)	0.507	1.1 (0.5 to 1.9)	0.913
Inadequate protein intake in infancy	1.5 (0.5 to 4.4)	0.419	2.0 (0.6 to 6.4)	0.250	1.6 (0.6 to 4.3)	0.381
Inadequate protein intake in early childhood	1.1 (0.4 to 2.9)	0.859	1.3 (0.4 to 3.6)	0.622	0.8 (0.3 to 2.0)	0.583
Inadequate fat intake in infancy	1.6 (0.7 to 3.7)	0.300	2.4 (0.9 to 6.4)	0.072	1.7 (0.7 to 3.9)	0.211
Inadequate fat intake in early childhood	1.5 (0.7 to 3.3)	0.297	1.2 (0.5 to 2.6)	0.677	1.1 (0.5 to 2.3)	0.836
Inadequate iron intake in infancy	1.0 (0.4 to 2.3)	0.963	1.1 (0.5 to 2.6)	0.819	0.5 (0.2 to 1.2)	0.140
Inadequate iron intake in early childhood	1.0 (0.5 to 2.4)	0.906	0.6 (0.3 to 1.4)	0.215	1.1 (0.5 to 2.4)	0.896
Inadequate zinc intake in infancy	0.8 (0.4 to 1.6)	0.574	1.1 (0.5 to 2.1)	0.874	0.7 (0.4 to 1.4)	0.333
Inadequate zinc intake in early childhood	0.7 (0.3 to 1.6)	0.377	1.1 (0.4 to 2.5)	0.940	0.6 (0.2 to 1.4)	0.238
Exposures from current conditions						
Poor child stimulation	2.7 (0.6 to 13.4)	0.140	22.1 (2.4 to 200.7)	0.001**	1.1 (0.2 to 4.8)	0.996
Never received formal/informal education	2.3 (0.9 to 6.0)	0.238	2.2 (0.8 to 5.8)	0.289	1.1 (0.4 to 2.9)	0.965
Maternal mental health disorder	1.8 (0.4 to 7.1)	0.412	2.3 (0.5 to 11.0)	0.300	0.9 (0.2 to 3.2)	0.842

on mother-child relationships and predicted child development.[2] Another intervention study showed that cognitive stimulation significantly improved cognitive function with most effect sizes ranged from 0.5 SD to 1.0 SD.21 Furthermore, follow-up studies consistently report long term effects of early cognitive intervention on child cognition.[22,23]

Stimulation improved child cognitive and educational deficits in late adolescence among stunted children. Overall, stunted non-stimulated participants had significantly poorer scores than the non-stunted group on 11 of 12 cognitive and educational tests. Stunting in early childhood was associated with cognitive and educational deficits in late adolescence, which can be reduced by stimulation at a young age.[24] Moreover, cognitive stimulation, such as giving materials and experiences, is also well established in global policy practices. Lancet series on *Child Development in Developing Countries* identified factors with sufficient evidence to recommend implementing prevention strategies. They identified four key risk factors urgently requiring intervention: stunting, inadequate cognitive stimulation, iodine deficiency, and iron deficiency anemia.[21]

Table 3. Multivariate analysis of low paternal education and FSIQ

Factors	Estimated			
	Regression coefficient (β)	SE of (β)	P value	OR (95% CI)
Constant	0.172	0.170	0.311	1.2
Low paternal education level	0.981	0.498	0.049*	2.7 (1.0 to 7.1)

*P value < 0.05 was considered to be statistically significant

Table 4. Multivariate analysis of VIQ and poor child stimulation and inadequate calorie intake in infancy

Factors	Estimated			
	Regression coefficient (β)	SE of (β)	P value	OR (95% CI)
Constant	-1.609	1.095	0.142	0.2
Poor child stimulation	2.604	1.147	0.023*	13.5 (1.4 to 127.9)
Inadequate calorie intake in infancy	1.846	1.062	0.082	6.3 (0.8 to 50.8)

*significant P value < 0.05

Table 5. Multivariate analysis of PIQ and inadequate intake of calories, iron, and zinc

Factors	Estimated			
	Regression coefficient (β)	SE of (β)	P value	OR (95% CI)
Constant	0.208	0.373	0.578	1.2
Inadequate calorie intake in infancy	1.274	0.659	0.053	3.6 (1.0 to 13.0)
Inadequate iron intake in infancy	-0.624	0.433	0.536	0.5 (0.2 to 1.2)
Inadequate zinc intake in early childhood	-0.694	0.537	0.196	0.5 (0.2 to 1.4)

We found that inadequate calorie intake in infancy is a risk factor for children's low VIQ and PIQ among 4-to-6-year-olds as indicated by high OR values, although not statistically significant. These results demonstrate not only the large effect of inadequate calorie intake on cognitive development, but also its long-lasting impact up to 4-6 years of age.

A review stated that the plasticity of the brain, neurons or synapses is the human ability to perform complex mental activities. This depends on the brain's ability to adapt to the environment and change its functional and structural organization. In addition, to maintain such complex and dynamic brain capabilities and maintain this complex architectural function, an extraordinary supply of energy is required.[25]

While the energy requirements of the adult brain are large, energy requirements during early life are even higher and are important for supporting rapid brain development with a growth spurt that begins around the 5th month of pregnancy and continues after birth, increasing brain weight from its adult weight at birth. It becomes 80% by 2 years of age and reaches its maximum size around 7 years of age. Therefore, postnatal growth, especially in early childhood, is not the result of the addition of new neurons, but the development and maturation of neurons, but instead it is the development and the maturation of neurons that already present at birth including axon growth, dendritic arborization elaboration, synaptic formation/ elimination, and axon myelination.[25]

The energetic costs associated with brain function in estimation that neurons consume 75-80% of the energy produced, whereas the rest is used for glia-based processes. Two main reasons may explain why neurons have high energetic demands: first, the generation of action potentials along the axons and synaptic transmission from neuron to neuron are based on electrochemical and cellular processes, such as ion fluxes, neurotransmitter release and reuptake, and vesicle cycling, which are energetically costly. A signaling mechanism at the synapse has been suggested to be especially energy consuming; for example, it has been estimated that 80% of the energy in myelinated hippocampal axons is expended by postsynaptic potentials.[25]

In addition to its enormous demand of energy, the dramatic brain size expansion that happens during the first years of life requires specific nutrients, such as lipids, proteins, and micronutrients, which are not only the building blocks of brain structures but also support brain and cognitive functions during the rest of the lifespan.[25]

Another role of energy on brain function among adults has emerge from evidence that suggests excessive energy intake, together with a sedentary lifestyle places the brain at risk for the development of late life cognitive impairment and Alzheimer's disease. [26]

Conflict of interest

None declared.

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