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Childhood Linear Growth and Early Morbidity as Predictors of Adolescent Cognitive Ability in Malawi: A Prospective Observational Study

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Keywords: adolescent | cognitive ability | eye tracking | linear growth | morbidity

ABSTRACT

Aim: Growth faltering and loss of development potential are common in low- and middle-income countries. We aimed to study whether linear growth before and after 2 years, height-for-age Z-score (HAZ) from 1 month until 13 years and morbidity during the first 3 years predict adolescent cognitive ability.

Methods: Cognitive assessment was done between 2018 and 2019 using Raven's coloured progressive matrices ($N = 997$), a measure of inductive reasoning and eye-tracking measures of saccadic speed ($N = 760$) and saccadic control ($N = 618$) among children whose mothers originally participated in a randomised clinical trial in rural Malawi. Linear regression was used to predict cognitive ability. The primary model was adjusted for age and sex, and the covariate-adjusted model for other prespecified variables.

Results: Saccadic control was predicted by a change in HAZ between 2 and 13 years in the adjusted model (coef −0.03, $p = 0.04$). Raven's score was predicted by change in HAZ between 1 month and 2 years (coef. 0.47, $p < 0.05$), and HAZ at 2, 5 and 13 years (coefs. 0.27–0.38, $p < 0.05$). Morbidity did not predict adolescent cognition.

Conclusion: Linear growth before 2 years of age and single HAZ measurements from 2 years onwards associated with later cognitive ability measured with Raven's test, but not consistently with eye-tracking assessment.

Abbreviations: AZI, azithromycin; CPM, Raven's coloured progressive matrices; HAD, height-for-age deficit; HAZ, height-for-age Z-score; IQ, intelligence quotient; LAIS, Lungwena Antenatal Intervention Study; LAZ, length-for-age Z-score; LMIC, low- and middle-income countries; NS, nonscheduled; PCCR, Pupil Centre Corneal Reflection; PE, percentage of error; pSRTm, mean prosaccadic reaction time; SD, standard deviation; SES, socioeconomic status; SP, sulfadoxine–pyrimethamine; WHO, World Health Organisation.

Summary

- Early linear growth and later cognitive ability share common determinants.
- We evaluated if childhood growth in length and morbidity associated with adolescent cognitive performance measured with Raven's coloured progressive matrices and eye-tracking-based measures of pro- and antisaccades.
- Raven's result was predicted by linear growth before the age of 2 and height at 2, 5 and 13 years, and antisaccadic performance by linear growth after age 2 in the covariate-adjusted model.

1 | Introduction

Early childhood, especially the first 2 years of life, is an important period for cognitive development and human capital formation [1]. There are common determinants between poor growth in length (stunting, height-for-age Z-score (HAZ) < −2SD) and weak cognitive ability in later life, such as poverty, poor nutrition, repeated infections and compromised sanitation facilities [2]. Better cognitive ability of young individuals results in higher academic achievements and earnings in adulthood, leading to productive societies [3]. However, far from the sustainable development goal target, an estimated 250 million children under 5 years of age are not attaining their developmental potential [4].

It is well known that early growth in length during the first 1000 days of life is a good predictor of later cognitive ability [5–7]. The association between linear growth after age 2 and later cognitive performance is more complex. According to a recent meta-analysis, linear growth after 2 years had very little or no association with neurodevelopmental outcomes [8] but some studies suggest opposite results [9, 10]. Repeated infections in early life may also impact cognitive development in low- and middle-income countries (LMIC) through direct harm to the central nervous system, infection-related anaemia, or by reducing children's energy to play [11].

Developmental assessment should be cross-culturally acceptable, reliable, valid, standardised and easy to administer [12]. Raven's coloured progressive matrices test (CPM) measures fluid intelligence and nonverbal reasoning and has been used in LMIC [13, 14]. The test can be administered without highly skilled personnel [13, 14]. Eye-tracking technology is a novel and objective test method used in different settings, and age and participant groups [14]. The administration of the test is computer-based and standardised. Eye-tracking–based measures of processing speed (saccadic reaction time) are associated with intelligence [15] and saccadic control has been related to pruning of certain brain areas [16]. It is important to evaluate the utility of eye-tracking as a cognitive outcome also in the rural LMIC adolescent population.

In this study, we wanted to determine if cognitive ability measured with the CPM test and eye-tracking assessed gaze speed and control could be predicted by childhood growth and early morbidity in the rural, Malawian 13-year-old adolescent population. We hypothesised that faster linear growth before the age of 2 years, the period crucial for brain development and between 2 and 13 years, HAZ at five different time points and less early morbidity predict better cognitive performance in adolescence.

2 | Methods

2.1 | Study Design

The data were collected from approximately 13-year-old adolescents, offspring from a pregnancy cohort recruited between December 2003 and October 2006, 'Lungwena Antenatal Intervention Study' (LAIS). The data collection at 13 years was planned as a separate project. In this randomised clinical trial, eligible women received antenatal intermittent preventive treatment intervention against malaria and reproductive tract infections in three groups. The control group received sulfadoxine–pyrimethamine (SP) twice during pregnancy, one intervention group monthly SP and placebo twice during pregnancy (monthly SP) and the second intervention group monthly SP and azithromycin (AZI) twice (AZI-SP) [17]. Anthropometric outcomes of the offspring between birth and 13 years were measured regularly, and developmental assessment was done at 5 and 13 years. Morbidity data were collected until 3 years of age (Figure 1).

The provision of AZI-SP intervention was associated with a lower prevalence of preterm birth and higher weight at birth [17]. The children in the AZI-SP group performed better at 5 years on Griffith's mental developmental scales, which is a standard, multidomain test [18], had lower cumulative incidence of stunting until 13 years, but no differences in cognitive performance at 13 years, compared to the control group children [19]. Faster prosaccadic reaction time was a very weak correlate of CPM score, but the antisaccadic task after covariate adjustment was not. Schooling was a potential moderator of the association between the tasks, as the participants with more school years had a stronger association between CPM and eye-tracking tasks [20].

For the current study, 13-year-old participants were recruited between January 2018 and March 2019. The first visit was made to the homes of all the children not known to have died. If the participant had moved, their new location was attempted to be found. A second visit was arranged at a study clinic to perform growth measurements, conduct an interview and implement developmental assessments.

2.2 | Outcomes

2.2.1 | Eye-tracking–based assessment of pro- and antisaccades

Eye tracking is based on a Pupil Centre Corneal Reflection (PCCR) technique, in which gaze position is estimated by comparing the reflection of a near-infrared light source from the cornea to an image of the centre of the pupil. The eye-tracking camera used in the current study (Tobii x2-60) tracked the participant's point of gaze at 60 Hz temporal and 0.4° spatial

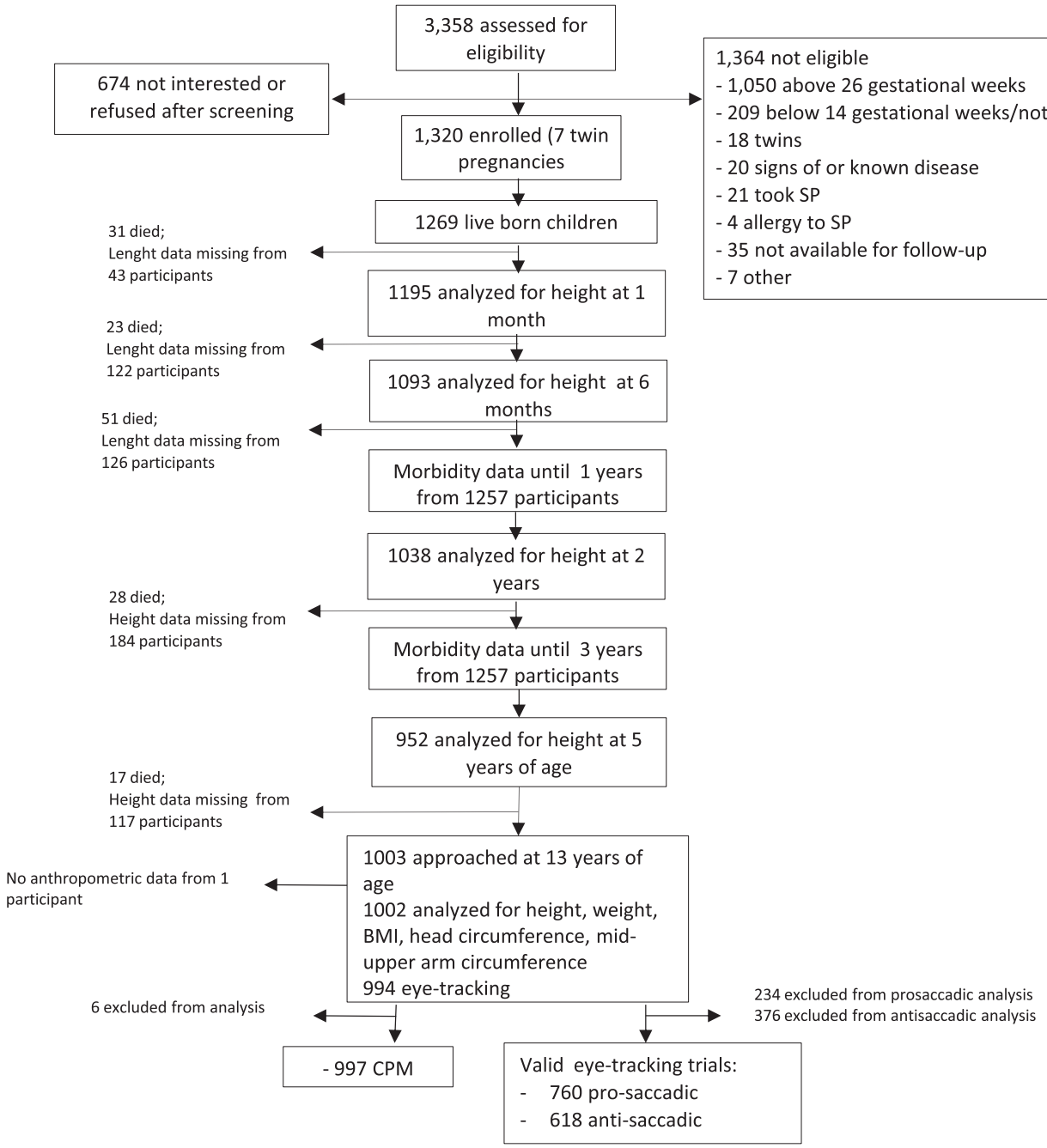

FIGURE 1 | Participant flow in CONSORT recommended format (Lancet 2001: 357: 1193).

accuracy, based on manufacturer specifications. A laptop computer was connected to the eye-tracker and a monitor to allow for data storing and stimulus presentation.

Participants performed two tasks. The prosaccadic task was used to test the subject's ability to generate reflexive, visually triggered saccades. Participants were instructed to look from a central fixation point towards an eccentric visual target on the left or right side of the screen. The mean of prosaccadic reaction times ($_pSRT_m$) was calculated for each participant. $_pSRT_m$ and other measures of processing speed have been shown to be associated with better cognitive ability in different age groups [15], and functional development of the subcortical and cortical areas relevant for this aspect of cognition starts early in childhood [21]. As most of the brain myelination takes place during the first 2 years of life, saccadic latencies as a measure of processing speed could provide a good indicator of early neurocognitive development [21]. In the antisaccade task, participants were instructed to suppress the reflexive saccade to the stimulus and look to the side opposite the target. The main outcome, percentage of erroneous eye movements towards the target (PE), was calculated for each participant as N error/N valid trials, where N valid trials is N errors + *N* correct saccades + *N* no saccades. Antisaccade performance relies on brain areas that continue to develop until early and late adolescence and thus could indicate the relation between later growth and cognitive development [22].

Prosaccade task. The child was instructed to maintain a stable position and look at the pictures on the screen. Each trial in the task started with a white fixation cross in the centre of the screen. After the child looked at the fixation, it remained on the screen for 500 ms, followed by a saccade target on the side of the screen (size 0.25 × 0.45 of the screen size). The target remained on the screen for 1000 ms after the prosaccade.

Antisaccade task. In the antisaccade task, the child was instructed to look at the fixation cross in the centre, and when the picture on the left or right appeared, to look away from it in the opposite direction. Trials were started with the central fixation stimulus presentation, followed by a lateral stimulus that remained on the screen for a total of 2 s on each trial (giving this period to make the correct antisaccade). Otherwise, the timing parameters were like those in the prosaccade task.

Both tests consisted of a total of 64 trials (4 blocks of 16 trials). The setting, equipment and the details of the calibration and pro- and antisaccadic procedures are described in the earlier work [20]. A minimum of 10 practice trials, repeated if needed, were presented for each child before both pro- and antisaccadic tests were started. The practice trial targets were yellow rectangular shapes with an instruction 'Look!' in the middle. The data collector monitored the assessments throughout the testing via an online visualisation of the child's gaze and made adjustments to the child's position as needed. During the actual test, the targets were pictures of human faces taken from the MUCT face database (www.milbo.org/muct).

2.2.2 | Raven's coloured progressive matrices

Raven's coloured progressive matrices (CPM) measure nonverbal intelligence and can be performed with illiterate participants [23]. CPM comprises 3 series of 12 pictures (36 tasks, result 0–36) that depict 2 × 2 matrices of geometric shapes with one missing piece, which was to be picked from an array of 6 alternatives by telling or pointing to the correct answers.

2.3 | Anthropometric measures

Child's length/height was measured at 1, 3, 6, 9, 12, 18 and 24 months and 5 and approximately 13 years. Before 2 years of age, the child was measured with a length board (Kiddimetre; Raven Equipment Ltd., Dunmow, United Kingdom), and data were considered missing if the actual date of measurement was off by more than 4 weeks from the target date. At 2 years or after that age, height was measured with a Harpenden stadiometer (increments of 1 mm) (Holtain Ltd., Crymych, United Kingdom). Data were considered missing if the measurement was off by more than 8 weeks from the target date. The anthropometric measurements were completed in triplicate. The mean of the first two readings was used if the reading did not differ by more than 0.5 cm. Otherwise, the pair of measurements with the smaller difference was used.

World Health Organisation (WHO) references were used to calculate age-and sex-specific height and length–/height-for-age Z-score. WHO references were also used to calculate height-for-age deficit (HAD, difference between measured height and the WHO standard height). As SD becomes bigger with age, HAZ is increasing despite HAD getting bigger [24, 25].

2.4 | Morbidity

Child morbidity was measured as the incidence of non-scheduled (NS) visits to a health centre made by the participant during the first 3 years of life, not including normal under-5 clinic visits. The information was recorded in real-time when the visit was done with structured data collection forms by the clinician doing the examination [26].

We calculated the incidence of NS visits by dividing the number of NS visits by the number of months in the study.

2.5 | Socioeconomic status (SES)

The SES score was created with principal component analysis by combining information on the building materials of the house, the main source of water, the sanitary facility and ownership of household items.

2.6 | Statistical Analysis

Variables indicating change in HAZ from 1 month to 2 years and from 2 to 13 years were created to evaluate the growth velocity during different age periods (later minus earlier measurement point).

To test the first hypothesis of linear growth before and after the age of 2 years as a predictor of adolescent cognitive abilities, we created regression models separately for all three primary outcomes. The testing procedure was adopted from an earlier study performed in the Philippines: [9]

1. The primary model included HAZ at 1 month, change in HAZ from 1 month to 2 years, interaction between these two, and change in HAZ from 2 to 13 years as independent variables, and was further adjusted for age (range 10.9 to 14.6 years) and sex.
2. The covariate-adjusted model was further adjusted for participants' SES, maternal education, gestational weeks at birth and intervention during pregnancy.

Interaction testing was done to evaluate the effect of initial HAZ and subsequent linear growth before 2 years on the results.

To test the second hypothesis of higher HAZ being a predictor of adolescent cognition, we created regression models for all the primary outcomes as dependent variables and HAZ at different time points. For the third hypothesis of early morbidity as a predictor of adolescent cognition, similar regression models were created to test if NS visits predict cognitive ability in adolescence. The primary model and the covariate-adjusted model were adjusted for similar covariates as described above.

In all the analyses, null hypotheses (i.e., no association) were rejected at an alpha of 0.05. We included in the analysis data from all the participants who had results from developmental outcome measures at 13 years, and the other data for growth and all the covariates used in the analysis. All the end points were considered exploratory.

2.7 | Ethical considerations

The participants and their guardians signed, or thumb printed the consent forms. The protocol was approved by the College of Medicine Research and Ethics Committee, Malawi, and the Ethical Committee of Pirkanmaa Hospital District, Finland. The trial was registered at www.clinicaltrials.gov (identifier NCT00131235). Both the original trial and the follow-up were performed according to Good Clinical Practice and the ethical standards of the Declaration of Helsinki.

3 | Results

There were 1269 children born alive to the mothers who originally participated in the LAIS trial. Length/height information was obtained for 1195, 1093, 1038, 952 and 1003 participants at 1 and 6 months, 2, 5 and 13 years respectively. The CPM test was completed by 997 participants and valid eye-tracking pro- and antisaccadic task results were attained from 760 and 618 participants at 13 years. Morbidity data were obtained for 1098–1257 participants (Figure 1). The characteristics of the participants at 13 years and the relevant characteristics of the mothers are presented in Table 1.

Mean (SD) HAZ of the participants was −1.3 (1.1) and −1.4 (1.2) at 1 and 6 months, −2.1 (1.0) at 2 years and −1.7 (0.9) and −1.7 (0.9 and 1.0) at 5 and 13 years of age (Table S1). HAD increased until 5 years, being mean (SD) −7.8 cm (4.4) and at 13 years, it was −7.4 cm (8.1) (Figure S1).

The mean (SD) incidence of NS visits was 0.14 (0.17) visits per month during the first 3 years of life. The visits were more frequent during the first year, mean (SD) 0.25 (0.25), compared to the later periods (Figure S2).

Children's linear growth between the time points was not associated with the mean pSRTm (Table S2). A change in HAZ between 1 month and 2 years was not associated with PE but there was a weak association between the change in HAZ between 2 and 13 years and PE in the covariate-adjusted model (primary and covariate-adjusted models coefs −0.03, (−0.06 to 0.03), $p = 0.08$ and −0.03, 95% CI (−0.07 to −0.001), $p = 0.04$, Table S3).

Length gain between 1 and 24 months predicted CPM performance (primary and covariate-adjusted models: Coef. 0.47, (0.12–0.82), $p = 0.009$, Coef. 0.45, (0.09–0.80), $p = 0.01$) and HAZ at 1 month predicted CPM performance in both models (primary and covariate-adjusted models: Coef. 0.40 (0.10–0.70), $p = 0.009$, Coef. 0.43 (0.11–0.75), $p = 0.009$). There was no interaction between initial HAZ and subsequent change in HAZ. Linear growth between 2 and 13 years did not predict CPM score (Table 2).

TABLE 1 | Characteristics of the participants at 13 years and their mothers.

Participant characteristics at 13 years	**1003**		
Mean (SD) age	12.8 (0.9)		
Proportion of boys (%)	50.3		
Mean (SD) anthropometric data		Boys	Girls
Height	142.8 (8.3)	140.8 (8.0)	144.8 (8.1)
Height-for-age Z-score	−1.7 (1.0)	−1.9 (0.9)	−1.4 (1.0)
Weight	33.9 (6.6)	32.3 (5.4)	35.6 (7.2)
Head circumference	51.6 (1.5)	52.0 (1.5)	51.3 (1.5)
Mid upper arm circumference	19.9 (2.1)	19.1 (1.7)	20.7 (2.3)
Mean (SD) years of school completed, $N = 996$	2.8 (1.8)		
Literate participants (%) ($N = 996$)	64.3		
Mean (SD) socioeconomic Z-score, $N = 990$, range from −1.3 to 4.2	0 (1)		
Maternal characteristic at trial enrolment			
Mean (SD) maternal age	24.9 (6.4)		
Mean gestational weeks (SD)	38.8 (1.8)		
Mean (SD) years of school completed	2.2 (2.7)		
Literate participants (%)	29.3		

HAZ at 1, 6 and 24 months, 5 and 13 years did not predict pSRTm and PE. The single measured HAZ at 1 and 6 months did not predict adolescent CPM performance. Higher HAZ at 24 months predicted better CPM result (coefs in primary and covariate-adjusted models 0.35 (0.12–0.59), $p = 0.003$ and 0.36 (0.12–0.60), $p = 0.003$). Similar results were observed at ages 5 and 13 years (coef. Varying from 0.27 to 0.38, $p < 0.05$) (Table 3).

Morbidity before 3 years of age measured with NS visit incidence per month did not predict pSRTm, PE or CPM score (Table 4).

4 | Discussion

We aimed to study if linear growth before and after the age of 2 years, height-for-age Z-score at different time points and early

TABLE 2 | Association of the child's first measured length and linear growth between two age intervals with and Raven's coloured progressive matrices score in early adolescence.

	Primary model[a] ($N = 892$)			Covariate-adjusted model[b] ($N = 883$)		
	Coef. (95% CI)	*p*	Adjusted R-squared/RMSE	Coef. (95% CI)	*p*	Adjusted R-squared/RMSE
HAZ_1	0.40 (0.10–0.70)	0.009	0.04/3.7	0.43 (0.11–0.75)	0.009	0.05/3.6
Change in HAZ 1–24 months	0.47 (0.12–0.82)	0.009		0.45 (0.09–0.80)	0.01	
Interaction: HAZ_1 and Change in HAZ 1–24 months	0.02 (−0.08–0.12)	0.64		0.03 (−0.07–0.13)	0.52	
Change in HAZ 24 months to 13 years	0.13 (−0.18–0.44)	0.42		0.13 (−0.19–0.44)	0.43	

Abbreviations: HAZ, height-for-age z-score; RMSE, root mean square error.
[a]Basic model adjusted for age and sex.
[b]Causal model adjusted for child age, sex, socioeconomic status, maternal education, gestational age at birth and maternal intervention during pregnancy.

TABLE 3 | Association of height-for-age z-score measured at five different time points during childhood and cognitive performance measured with prosaccadic reaction time (pSRTm), antisaccadic percentage of errors (PE) and Raven's coloured progressive matrices (CPM) in early adolescence.

		pSRTm		PE		CPM	
		Coef. (95% CI)	*P*	Coef. (95% CI)	*P*	Coef. (95% CI)	*P*
HAZ1	Primary model[a]	−0.63 (−1.86–0.60)	0.31	0.007 (−0.02–0.03)	0.54	0.09 (−0.12–0.30)	0.42
	Adjusted model[b]	−0.69 (−2.10–0.71)	0.33	0.002 (−0.02–0.03)	0.88	0.13 (−0.11–0.37)	0.28
HAZ6	Primary model[a]	−0.04 (−1.23–1.15)	0.95	0.02 (−0.005–0.04)	0.14	0.16 (−0.05–0.36)	0.14
	Adjusted model[b]	0.01 (−1.23–1.25)	0.99	0.01 (−0.009–0.04)	0.23	0.19 (−0.03–0.40)	0.09
HAZ24	Primary model[a]	−0.33 (−1.69–1.03)	0.64	−0.004 (−0.03–0.02)	0.71	0.35 (0.12–0.59)	0.003
	Adjusted model[b]	−0.28 (−1.67–1.10)	0.69	−0.006 (−0.03–0.02)	0.66	0.36 (0.12–0.60)	0.003
HAZ60	Primary model[a]	0.10 (−1.42–1.62)	0.90	−0.002 (−0.03–0.02)	0.87	0.38 (0.11–0.65)	0.006
	Adjusted model[b]	0.39 (−1.14–1.92)	0.62	−0.005 (−0.03–0.02)	0.71	0.36 (0.09–0.64)	0.009
HAZ144	Primary model[a]	−0.39 (−1.79–1.0)	0.58	−0.01 (−0.04–0.01)	0.30	0.27 (0.02–0.51)	0.03
	Adjusted model[b]	−0.16 (−1.58–1.26)	0.82	−0.02 (−0.04–0.008)	0.18	0.28 (0.03–0.53)	0.03

Note: N varying in primary model: pSRTm 661–755, PE 539–613, CPM 882–996, *Note: N* varying in covariate adjusted model: pSRTm 657–747, PE 535–607, CPM 874–984.
[a]Primary model adjusted for age and sex.
[b]Covariate adjusted model adjusted for child age, sex, socioeconomic status, maternal education, gestational age at birth and maternal intervention during pregnancy.

morbidity predict adolescent performance in CPM test and eye-tracking measured gaze speed and control. Linear growth before the age of 2 years, but not between 2 and 13 years, was associated with adolescent cognition, as measured with CPM. HAZ at 2, 5 and 13 years predicted CPM performance. We found no evidence for an association between childhood growth and eye-tracking measured speed and control, apart from a weak association between linear growth after age 2 and antisaccadic task performance when covariates were controlled for. Early life morbidity did not predict any measures of cognitive performance in adolescence.

There are some limitations in our study. First, some aspects of the early environment that may be important determinants of child development, such as playing materials during childhood, safety information, or breast-feeding duration were not covered in our study [27]. Socioeconomic status, which was included as a covariate in the analysis, can be used as a proxy for home environment indirectly covering some of the missing aspects [7]. Second, the exact purpose of NS health care visits was not recorded. However, the data were collected from over 1250 participants and can give insight into morbidity [26]. Also, imprecision in growth measurement is possible and

TABLE 4 | Association of morbidity before 3 and 1 year of age with cognitive performance measured with prosaccadic reaction time (pSRTm), antisaccadic percentage of errors (PE) and Raven's coloured progressive matrices (CPM) in early adolescence.

		pSRTm		PE		CPM	
		Coef. (95% CI)	*P*	Coef. (95% CI)	*P*	Coef. (95% CI)	*P*
Morbidity before 3 years of age	Primary model[a]	−0.19 (−12.7–12.4)	0.98	0.11 (−0.13–0.34)	0.37	−1.0 (−3.1–1.0)	0.34
	Adjusted model[b]	1.31 (−11.5–14.1)	0.84	0.10 (−0.13–0.34)	0.40	−1.77 (−3.8–0.3)	0.09
Morbidity during the third year	Primary model[a]	1.05 (−5.2–7.3)	0.74	0.11 (−0.13–0.34)	0.37	−0.54 (−4.2–3.0)	0.77
	Adjusted model[b]	1.85 (−4.4–8.1)	0.56	−0.06 (−0.16–0.05)	0.27	−1.18 (−4.8–2.4)	0.52
Morbidity during the second year	Primary model[a]	−3.3 (−13.7–7.2)	0.54	0.06 (−0.16–0.28)	0.59	−0.92 (−2.6–0.8)	0.29
	Adjusted model[b]	−1.94 (−12.4–8.6)	0.72	0.08 (−0.14–0.30)	0.50	−1.08 (−2.8–0.6)	0.22
Morbidity during the first year	Primary model[a]	1.92 (−4.3–8.2)	0.55	0.03 (−0.08–0.14)	0.62	−0.10 (−1.2–0.97)	0.86
	Adjusted model[b]	2.23 (−4.0–8.5)	0.49	0.02 (−0.09–0.13)	0.71	−0.30 (−1.37–0.77)	0.58

Note: N varying in models: pSRTm 728–754, PE 598–613, CPM 958–995.
[a]Primary model adjusted for age and sex.
[b]Covariate-adjusted model adjusted for child age, sex, socioeconomic status, maternal education, gestational age at birth and maternal intervention during pregnancy.

was tried to control with regular quality checks and triplicate measurements.

Early growth and stunting are linked to later cognitive performance in low-income setting [4, 6]. Faster growth between 6 months and 2 years, no matter of initial infant size, resulted in better cognitive performance at 11 years in a study performed in Philippines where linear growth faltering is common [9]. Our results were similar with this finding except for that forementioned study also observed that length gain between 2 and 11 years predicted later cognitive performance. Contrary to our results, in a Malawian study, faster length gain between 2 and 15 years predicted better performance in CPM test, partly mediated by schooling [13]. In a study using large dataset from USA, Belarus and Philippines, foetal, infant and mid-childhood (6.5 to 8.5 years) linear growth were consistently associated with mid-childhood intelligence quotient (IQ) but with modest magnitude. In the cohorts from Belarus and Philippines, the association of foetal and early growth with mid-childhood IQ mediated through later growth periods possibly indicating that in the US cohort postnatal growth was unconstrained compared to two other settings [28]. According to a recent a meta-analysis result, however, change in HAZ after age two in LMIC setting had a weak or no association with childhood neurodevelopment and our study partly supports these findings [8].

Higher HAZ at 2 years was positively associated with Full-Scale IQ at 6–7 years in Vietnamese study, in line with our results [10]. Early life stature (HAZ) at 1–2 years was associated with adult IQ in a large multicentre study, but the association attenuated when child IQ and schooling were controlled for [3]. The Young Lives study conducted in Ethiopia, India, Peru and Vietnam showed an association between HAZ at 8 and 15 years and educational attainment at 15 years, and HAZ at 12 and 15 years with cognitive performance in math and verbal skills at 15 years. Persistent stunting was associated with poorer educational attainment and cognitive performance. In these studies, continuous HAZ rather than specific cut-offs (HAZ < −2 SD) have been linked to later development, likewise in our study, where HAZ at 2, 5 and 13 years predicted adolescent CPM result [29].

To our knowledge, there is no data linking childhood growth and morbidity with adolescent eye-tracking test performance. Eye-tracking–based assessment could be a culturally independent marker of adolescent cognitive abilities. Early maturation and myelination of cortical visual and attention-related brain areas is believed to be important for the development of saccadic eye movements and improved saccadic latency [30]. However, we did not detect an association between linear growth before 24 months or early morbidity and adolescent pSRTm. Whereas saccadic eye movements emerge early, the ability to control eye movements may depend on brain mechanisms (e.g., the prefrontal cortex) that follow a more prolonged developmental trajectory, continuing into adulthood [22, 30]. We observed an association of faster linear growth between 2 and 13 years and better gaze control in adolescence in the covariate-adjusted model. This finding is biologically plausible but still needs further investigation.

There are data of the association between early infections and later cognitive development [11, 31]. For example, early life enteric infections can lead to compromised later neurodevelopment through malnutrition (including poorer iron absorption and myelin synthesis), microbiota dysbiosis and possible subclinical systemic inflammation affecting gut-liver-brain axis [32]. We did not observe association between early morbidity with adolescent cognitive performance which might be partly explained by the fact that our morbidity data did not include the accurate visit purpose.

5 | Conclusion

This study adds evidence of the shared determinants between growth before 2 years starting in utero and later cognitive ability. The first 1000 days in human life is an important period to promote healthy growth and development. Furthermore, lower HAZ from 2 years onwards could be an indicator of being at risk for cognitive adversity and offer opportunity for interventions.

Author Contributions

Karoliina Videman: formal analysis, data curation, writing – original draft, writing – review and editing. **Jukka M. Leppänen:** conceptualization, supervision, writing – review and editing, formal analysis, methodology. **Lotta Hallamaa:** writing – review and editing, formal analysis. **Kenneth Maleta:** writing – review and editing. **Per Ashorn:** conceptualization, funding acquisition, methodology, supervision, writing – review and editing. **Charles Mangani:** investigation, methodology, writing – review and editing. **Ulla Ashorn:** conceptualization, funding acquisition, methodology, supervision, writing – review and editing.

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Conflicts of Interest

The authors declare no conflicts of interest.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Appendix S1:** Supporting Information.