

Birth weight and intellectual abilities among term-born children with neurodevelopmental disorders: Evidence from a clinically referred sample

Christine Dahl

`Christine.dahl@medisin.uio.no`

University of Oslo

Eden N. Barak

University of Oslo

Knut K. Kolskår

Oslo University Hospital

Dag Alnaes

University of Oslo

Terje Naerland

University of Oslo

Lars T. Westlye

University of Oslo

Ole A. Andreassen

University of Oslo

Research Article

Keywords: Birth weight, MoBa, Neurodevelopmental disorders, Autism, ADHD, Intellectual disability, Cognitive development, Term birth

Posted Date: August 6th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10340791/v1>

License: © ⓘ This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Additional Declarations: No competing interests reported.

Abstract

Background: Low birth weight has consistently been linked to poorer cognitive outcomes, yet less is known about whether variation in birth weight within the normal range (2500–4500 g) relates to intellectual functioning in children and adolescents referred for clinical evaluation of neurodevelopmental disorders (NDDs).

Objective: To examine whether birth weight within the normal range is associated with intellectual functioning and diagnostic status in a clinical sample of term-born children and adolescents with confirmed or suspected NDDs, and to evaluate whether deviations from expected birth weight are associated with diagnostic outcomes.

Methods: Clinical data were obtained from participants in the Norwegian BUPgen registry with available birth weight, full-scale IQ (FSIQ), and diagnostic data. After restricting the sample to individuals born at term (37–41 weeks of gestation) with birth weight within the normal range (2500–4500 g), 248 individuals with ASD, 68 with ADHD, 7 with ID, and 26 without registered NDD diagnoses were included. Birth weight was obtained through linkage to the Medical Birth Registry of Norway. Standardized birth weight was derived using a population-based reference sample of term-born participants without NDDs from the Norwegian Mother, Father and Child Cohort Study (MoBa; $n = 91,953$). Linear regression models were used to examine associations between standardized birth weight and FSIQ within BUPgen. Linear models including MoBa were used to compare standardized birth weight across groups.

Results: Standardized birth weight was not significantly associated with FSIQ ($\beta=0.92$, $SE=0.74$, $p=.213$). No evidence of non-linear effects ($p=.625$) or interactions with sex ($p=.602$) was observed. Mean FSIQ in the clinical cohort was also approximately 9 IQ points below the normative population mean, with significantly lower scores than the population norm observed in the total sample as well as within the ASD, ADHD, and ID groups.

Linear models showed a small but significant overall effect of group on standardized birth weight ($F(4, 83883) = 3.41$, $p = .009$). Post hoc comparisons indicated that BUPgen participants without registered NDD had lower standardized birth weight than MoBa ($p=.020$) and ASD ($p=.035$), whereas no significant differences were observed between the ASD, ADHD, ID, and MoBa groups.

Conclusions: Variation in standardized birth weight within the normal range was not associated with FSIQ and did not distinguish ASD, ADHD, and ID groups from the population-based MoBa sample. Although participants without registered NDD showed lower standardized birth weight, this finding should be interpreted cautiously due to the small and heterogeneous nature of this subgroup. Overall, variation in fetal growth within the normal range, as indexed by standardized birth weight, may have limited relevance for cognitive functioning and NDD diagnostic classification in clinically referred populations.

Introduction

Several studies have documented a robust relationship between low birth weight (< 2500 g) and reduced performance on standardized intelligence tests, suggesting that restricted fetal growth may have long-lasting consequences for neurocognitive development (Peyre et al., 2026; Shenkin et al., 2004). Low birth weight is frequently the result of prematurity, intrauterine growth restriction, or perinatal complications, all of which interfere with critical processes of brain maturation such as synaptogenesis, myelination, and cortical organization. These disruptions have been linked not only to lower intelligence quotient (IQ) but also to learning disabilities, poorer academic achievement, and neurodevelopmental disorders (NDDs) across childhood and adolescence (Guellec et al., 2011; Volpe, 2009)

Although the adverse effects of low birth weight are well established, less is known about whether more subtle variation in birth weight in term-born infants within the normal weight range (2500–4500 g) (World Health Organization, 2025) also contributes to differences in cognitive outcomes, especially in individuals with NDD. Birth weight serves as a widely used proxy marker of intrauterine growth and fetal health, and even within the normal spectrum, differences in placental function, maternal nutrition, or pregnancy complications have been suggested to influence brain development in ways that affect later intellectual functioning (Walhovd et al., 2024). This raises the possibility that birth weight exerts a continuous influence on cognitive ability, rather than conferring risk only at extreme levels. To delineate the underlying etiology in complex neurodevelopmental disorders, studying the association between birth weight and later cognitive performance in clinically referred populations is of particular importance.

NDDs comprise a group of early-onset conditions arising from atypical development, and among the most common and clinically important are Autism Spectrum Disorder (ASD), Attention Deficit-Hyperactivity Disorder (ADHD) and Intellectual Disability (ID). According to the DSM-5 they are characterized by impairments in cognition, communication, social interaction, and adaptive functioning, with wide genetic and clinical variability, as well as high comorbidity and symptom overlap (Morris-Rosendahl & Crocq, 2020; Parenti et al., 2020; Zhao et al., 2022). NDDs affect approximately 10% of children worldwide (National Institute for Health and Care Excellence, 2019) and represent a major source of long-term disability. Prevalence estimates vary substantially across studies depending on methodological and demographic factors. Recent meta-analyses and large-scale studies have estimated prevalence rates of approximately 5.9% for ADHD, 1% for ASD and 1% for ID (Bölte et al., 2023). Epidemiological studies have linked fetal growth and birth weight to increased risk of language delays, cognitive impairment, and behavioral dysregulation, which often persist into school age and adolescence (Leonard et al., 2006), as well as NDDs, including ADHD and ASD (Cortese et al., 2021; Heinonen et al., 2015; Larroque et al., 2008; Raznahan et al., 2012; Walhovd et al., 2012). Numerous studies have established an association between low birth weight and neurocognitive development across the lifespan. Literature has shown a close relationship between birth weight and brain development (Catena et al., 2019), suggesting that birth weight may serve as a proxy for underlying brain maturation. In example, low birth weight has been linked to reduced total brain volume, disrupted white matter integrity, and delayed cortical maturation (Isaacs et al., 2000).

Relevant for investigation of the association between birth weight and cognitive development in the normal weight range is the cascade of brain maturation happening close to birth. Approximately 50% of the increase in cortical volume occurs between the 34th and 40th gestational weeks (Yu et al., 2016). Indeed, the third trimester represents a critical period of brain development characterized by rapid synaptogenesis, myelination, and cortical folding, processes that are particularly vulnerable to reduced nutrient and oxygen supply in utero (Volpe, 2009).

In example, small for gestational age (SGA) is defined relative to gestational age and reflects birth weight below the 10th percentile. SGA infants often experience intrauterine growth restriction, which can compromise brain development due to chronic nutrient and oxygen deprivation during critical periods of gestation (Qing et al., 2025). Subclinical in utero inflammation, preeclampsia, primary and secondary placental dysfunction and intrauterine infection have also been shown to impair fetal growth (Cortese et al., 2021).

Longitudinal studies have demonstrated that SGA status is associated with lower IQ scores, reduced language skills, and poor academic performance extending into adolescence and adulthood (Guellec et al., 2011). Furthermore, functional imaging studies further suggest that individuals born SGA exhibit altered brain connectivity in key cognitive networks, including the default mode and frontoparietal networks, which may represent a brain correlate of the observed deficits in executive function, attention, and processing speed (Karolis et al., 2016; Smyser et al., 2010).

However, recent studies suggests that there may occur relevant fluctuations in cognitive outcome beyond the suggested thresholds associated with i.e. SGA. A recent cohort study evaluating cognitive outcome at 5 years of age found increasing cognitive abilities with increasing birth weight, plateauing just below the 70-percentile birth-weight marker (Eves et al., 2023). Accordingly, Burger and colleagues found positive associations between at-term birth-weight and school performance at 12 years of age, plateauing at the 80-centile (Burger et al., 2023). Taken together, these findings suggests that there may be unexplored clinical relevant information for clinical patient groups beyond the recognized birth-weight thresholds.

Several hypotheses propose how fetal birth weight may impact cognitive function. According to the brain reserve model (Katzman et al., 1988), individual differences in brain structure, such as brain size, neuronal count, or synaptic density, can provide a buffer against clinical symptoms of brain pathology. In this case, higher birth weight may be associated with higher brain growth before birth (Katzman et al., 1988; Walhovd et al., 2024). While the cognitive effects of low birth weight may be most pronounced in early childhood, subtle deficits may persist into adulthood, influencing real-life outcomes such as educational attainment and cognitive aging trajectories (Walhovd et al., 2024).

Prenatal adversity associated with fetal growth restriction may create a neurobiological vulnerability that may be amplified by postnatal stressors (Shenkin et al., 2004), and the prenatal environment may have long lasting effects on individual outcomes, especially cognition (Shenkin et al., 2004).

To support the importance of increased knowledge regarding birth markers as a proxy for later aberrant development, the World Health Organization (WHO, 2016) emphasizes early identification and intervention as critical to improving outcomes in children with NDDs. From a clinical perspective, testing the prognostic value of birth weight in this group may inform risk stratification, guide developmental monitoring, and highlight subgroups of children who may benefit from tailored interventions.

The aim of the present study is to investigate the relationship between birth weight, intellectual functioning and diagnostic status among children and adolescents referred for suspected NDDs, born at term (37–41 weeks) within normal BW-range (2500–4500 g). By using data from the Norwegian BUPGen research and biobank, we examine whether even relatively modest variations in fetal growth reassociated with differences in cognitive outcomes. To allow comparison with typically developing children, birth weight was standardized using data from term-born participants without NDDs from the Norwegian Mother, Father and Child Cohort Study (MoBa) as the reference group (Brandlistuen et al., 2025), enabling calculation of sex- and gestational age-adjusted birth weight Z-scores. Associations between birth weight and full-scale IQ (FSIQ) in the BUPGen sample were tested using linear models. Group differences in birth weight across all groups were examined using linear models with sex included as a covariate, testing the overall effect of NDD on birth weight, as well as differences between diagnostic groups. This approach allowed us to evaluate birth weight as both a continuous predictor of intellectual functioning and diagnostic outcomes in a real-life clinical study sample enriched for neurodevelopmental risk and phenotypes.

Methods

Participants

Clinical data were obtained from patients enrolled in BUPGen, a national research registry and biobank including patients referred to Norwegian specialized health services for clinical assessment of ASD and other NDDs. Since 2013, approximately 2400 participants have been included in the registry. From this cohort, 349 children and adolescents were included in the present analyses based on the availability of birth weight, full-scale IQ, and diagnostic classification data. In addition, participants were restricted to those born at term (37–41 weeks of gestation) and with birth weight within the range of 2500–4500 g. A total of 120 participants were excluded from the BUPGen sample due to birth weight outside of this range.

Among the included participants, 261 (74.8%) were male and 88 (25.2%) were female. Mean birth weight was 3586 g (SD = 462, range: 2600–4500), and mean age at cognitive assessment was 11.4 years (SD = 3.3, range: 4–18). Mean F IQ was 90.8 (SD = 15.4, range: 49–133). Mean standardized birth weight was -0.06 (SD = 1.12). ASD was the most common diagnosis ($n = 248$), followed by ADHD ($n = 68$) and ID ($n = 7$) (Table 1). A subset of participants had no registered ASD, ADHD, or ID diagnosis ($n = 26$). Among participants without ASD, ADHD, or ID the most common presentations were non-specific emotional and behavioral symptoms (34.6%) and anxiety, mood, obsessive–compulsive, or other emotional disorders

(30.8%). One participant had a developmental language disorder, one had an eating disorder, and six participants (23.1%) had no formal ICD-10 diagnosis assigned. Thus, the “no diagnosis” group represented a clinically referred population rather than unaffected controls. A subset of participants fulfilled diagnostic criteria for two neurodevelopmental disorders. Co-occurring ASD and ADHD was the most common combination (n = 61).

Descriptive Characteristics of the Sample (N = 349)

Table 1

Descriptive characteristics of the clinical sample overall and across diagnostic groups. Values are presented as mean (SD) unless otherwise specified. Standardized birth weight values (Z-scores) were calculated relative to the population-based MoBa sample of term-born participants without NDDs. Diagnostic categories were not mutually exclusive; some participants fulfilled criteria for more than one diagnosis.

Characteristic	Total sample	No diagnosis	ASD	ADHD	ID
Participants, n	349	26	248	68	7
Male, n (%)	261 (74.8)	17 (65.4)	194 (78.2)	47 (69.1)	3 (42.9)
Female, n (%)	88 (25.2)	9 (34.6)	54 (21.8)	21 (30.9)	4 (57.1)
Birth weight, mean (SD), g	3586 (462)	3381.5 (541.5)	3605 (459.7)	3622.7 (426.4)	3312.9 (360.2)
Age at assessment, mean (SD), years	11.4 (3.3)	13.0 (3.7)	11.2 (3.3)	10.9 (3.0)	13.7 (4.4)
FSIQ, mean (SD)	90.8 (15.4)	92.7 (18.1)	90.8 (14.8)	92.6 (14.9)	67.1 (10.7)
Standardized birth weight (Z-score), mean (SD)	-0.06 (1.12)	-0.50 (1.31)	-0.01 (1.13)	-0.01 (0.99)	-0.50 (0.69)

Population based birth weight data were obtained from The Norwegian Mother, Father and Child Cohort Study MoBa, restricted to term-born individuals without registered NDDs and with birth weight within the normal range (2500–4500 g, n = 90012, 50,2% females, 49,8% males). The Norwegian Mother, Father and Child Cohort Study (MoBa) is a population- based pregnancy cohort study conducted by the Norwegian Institute of Public Health. Participants were recruited from all over Norway from 1999–2008. Thewomen consented to participation in 41% of the pregnancies. The cohort

includes approximately 114.500 children, 95.200 mothers and 75.200 fathers.

MoBa is regulated by the Norwegian Health Registry Act.

Measures

Intelligence quotient (FSIQ)

IQ was assessed using age-appropriate Wechsler tests of intelligence, namely the Wechsler Intelligence Scale for Children (WISC) for participants above the age of 6, and The Wechsler Preschool and Primary Scale of Intelligence (WPPSI) for participants below the age of 6. FSIQ was derived from performance across multiple cognitive domains and used as a standardized measure of general intellectual ability. FSIQ is norm-referenced ($M = 100$, $SD = 15$) and reflects overall cognitive functioning relative to same-age peers (Wechsler, 2003, 2008; Wechsler, 2012).

WISC is a widely used, standardized measure of intellectual functioning designed for children aged 6 to 16 years. The test assesses a broad range of cognitive abilities, including verbal and nonverbal reasoning, working memory, processing speed, and provides an overall estimate of general intellectual ability relative to age-matched peers. FSIQ is calculated from a core set of subtests spanning five primary index domains: Verbal Comprehension, Visual–Spatial Reasoning, Fluid Reasoning, Working Memory, and Processing Speed. These domains collectively capture both verbal and nonverbal aspects of intellectual functioning (Wechsler, 2003, 2008; Wechsler, 2012).

WPPSI is a standardized measure of general intellectual functioning designed for children aged approximately 2 years and 6 months to 7 years and 7 months. FSIQ is derived from core subtests assessing verbal comprehension, visual–spatial ability, fluid reasoning, working memory, and processing speed (Wechsler, 2003, 2008; Wechsler, 2012).

Birth weight

Birth weight was defined as the infant's weight measured immediately after delivery. Information regarding birth weight was obtained through linkage to the Medical Birth Registry of Norway (MBRN), a national health registry containing information about all births in Norway. Normal birth weight was operationalized as 2500–4500 g, consistent with Norwegian population-based growth references (e.g., the Bergen Growth Study), in which this interval largely overlaps with the 10th–90th percentile range for term-born infants (Júlíusson et al., 2009).

Diagnostic classification

Diagnostic classification was based on clinical diagnoses recorded in the BUPgen registry and assigned according to the International Statistical Classification of Diseases and Related Health Problems, 10th Revision (World Health Organization, 2016). For the present study, analyses were restricted to the main diagnostic categories ASD, ADHD and ID. ID included ICD-10 codes F70 (mild), F71 (moderate) and F72 (severe). ASD included F84.0 (childhood autism), F84.1 (atypical autism), F84.5 (Asperger syndrome) and F84.9 (pervasive developmental disorder, unspecified). ADHD included F90.0 (disturbance of activity and attention) and F90.1 (hyperkinetic conduct disorder). In cases of comorbidity, a hierarchical classification approach was applied, whereby individuals were assigned to a primary diagnostic group

according to a predefined hierarchy (ASD > ADHD > ID). In addition, a binary variable indicating the presence of any NDD was defined.

Ethical Considerations

The study is approved by the Regional Ethical Committee (REK 2019/943; 2009/932; 2016/1756; 240277) and carried out in accordance with the World Medical Association's Helsinki Declaration. Written informed consent was obtained from parents or legal guardians on behalf of all participants prior to inclusion in the study.

Statistical analysis

To investigate the relationship between standardized birth weight, NDDs, and FSIQ within the normal birth weight range (2500–4500 g), a series of regression analyses were conducted using R version 4.5.3 using RStudio version 2026.03.11; Posit Team, 2023. Robust estimation was performed using the *estimatr* package in sensitivity analyses. All statistical tests were two-tailed, with statistical significance set at $p < .05$.

Birth weight was standardized using a population-based reference sample from MoBa, restricted to participants without registered NDDs who were born at term (37–41 weeks of gestation) and within the normal birth weight range (2500–4500 g, $n = 91,953$). A linear regression model was fitted in the reference sample with birth weight as the dependent variable and gestational age and sex as predictors. Standardized birth weight (Z-scores) in both samples was calculated as the difference between observed and predicted birth weight divided by the residual standard deviation of the reference model. Birth weight Z-scores therefore represent deviations from the expected birth weight for gestational age and sex among term-born children with birth weights within the normal range. To examine whether standardized birth weight was associated with intellectual functioning, multiple linear regression models were estimated with FSIQ as the dependent variable and standardized birth weight (Z-score) as the independent variable. Models were adjusted for sex, with males specified as the reference category. To explore potential non-linear associations, a quadratic term for standardized birth weight was included in the regression models. In addition, we tested the interaction effects between standardized birth weight and sex to evaluate whether the association differed between females and males. Estimated marginal means and post hoc pairwise comparisons between groups were examined using Tukey-adjusted contrasts. To assess whether FSIQ scores differed from the normative population mean, one-sample t-tests were conducted in the total clinical sample and within each clinical group, using an expected mean IQ of 100.

To examine whether standardized birth weight differed across clinical groups and relative to the population-based MoBa reference sample, linear regression models were estimated with standardized birth weight (Z-score) as the dependent variable and group (MoBa, ASD, ADHD, ID, and participants without registered NDD) included as a categorical predictor. Models were adjusted for sex, with males

specified as the reference category. The overall effect of group was evaluated using an F-test. Estimated marginal means were obtained from the fitted model, and pairwise comparisons between groups were conducted using Tukey-adjusted post hoc contrasts.

Sensitivity analysis

To assess the robustness of the findings, several sensitivity analyses were conducted. First, linear regression models were re-estimated using heteroskedasticity-consistent (HC2) standard errors. Second, influential observations were identified using Cook's distance (threshold $4/n$), and analyses were repeated after exclusion of influential cases. Third, analyses of IQ were repeated after exclusion of extreme values ($IQ < 40$ or > 130). Across all models, standardized birth weight (Z-score) was the primary exposure variable.

Results

Is birth weight associated with FSIQ?

Figure 1 shows the distribution of standardized birth weight (Z-score), and Fig. 2 illustrates the association between standardized birth weight and FSIQ across all BUPgen participants. Linear regression analyses revealed no significant association between standardized birth weight and FSIQ ($\beta = 0.81$, $SE = 0.77$, $p = .295$). Sex was also not significantly associated with FSIQ ($p = .963$), and the model explained a negligible proportion of variance in FSIQ ($R^2 = 0.007$).

To examine potential non-linear associations, a quadratic term for standardized birth weight was included in the regression model. The quadratic term was not statistically significant ($\beta = 0.63$, $SE = 0.61$, $p = .306$), indicating no evidence of a non-linear relationship between standardized birth weight and IQ. Likewise, there was no evidence of an interaction between standardized birth weight and sex ($\beta = -0.42$, $SE = 2.00$, $p = .834$), suggesting that the association did not differ between males and females.

One-sample t-tests demonstrated that mean FSIQ in the total clinical sample was significantly lower than the normative population mean of 100 (mean = 90.84, 95% CI [89.22, 92.46], $p < .001$). Mean FSIQ was also significantly below the normative mean within the ASD group (mean = 90.84, 95% CI [88.99, 92.70], $p < .001$), the ADHD group (mean = 92.56, 95% CI [88.96, 96.16], $p < .001$), and the ID group (mean = 67.14, 95% CI [57.20, 77.08], $p < .001$).

Finally, FSIQ differed significantly across diagnostic groups overall (model $p = .001$), primarily reflecting substantially lower FSIQ in the ID group compared with the ASD, ADHD, and no-diagnosis groups (Table 1). Post hoc comparisons showed that the ID group had significantly lower FSIQ than the ASD group (mean difference = -23.82 , $p < .001$), the ADHD group (mean difference = -25.41 , $p < .001$), and the group without registered diagnoses (mean difference = -25.91 , $p < .001$). No significant differences in FSIQ were observed between the ASD and ADHD groups ($p = .868$), between the ASD and no-diagnosis groups ($p = .909$), or between the ADHD and no-diagnosis groups ($p = .999$).

Standardized birth weight relative to population expectations and across clinical groups

Figure 3 shows the distribution of standardized birth weight (Z-score) across MoBa and BUPgen participants. Linear regression analyses including the population-based MoBa reference sample showed a significant overall effect of group on standardized birth weight ($F(4, 83883) = 3.41, p = .009$). Estimated marginal means adjusted for sex were 0.04 (95% CI [0.03, 0.04]) for the MoBa reference sample, -0.48 (95% CI [-0.81, -0.15]) for participants without registered diagnoses, 0.01 (95% CI [-0.19, 0.21]) for the ADHD group, 0.03 (95% CI [-0.08, 0.14]) for the ASD group, and -0.51 (95% CI [-1.14, 0.13]) for the ID group. Pairwise comparisons using Tukey-adjusted contrasts showed that participants without registered NDD had significantly lower standardized birth weight than the MoBa sample ($p = .020$) and the ASD group ($p = .035$). No other pairwise differences between groups were statistically significant.

Robustness of findings

Sensitivity analyses yielded results consistent with the primary findings. Robust regression models using HC2 standard errors produced similar estimates to the main analyses ($\beta = 0.92, SE = 0.75, p = .220$). Exclusion of influential observations based on Cook's distance resulted in comparable findings ($\beta = 1.14, SE = 0.72, p = .112$). Likewise, exclusion of extreme FSIQ values ($IQ < 40$ or > 130) yielded similar estimates ($\beta = 0.92, SE = 0.73, p = .211$), supporting the robustness of the null findings.

Discussion

The present study investigated whether variation in birth weight within the normal term range (2500–4500 g) was associated with intellectual functioning in a sample of children and adolescents referred for NDD evaluation. Across all analyses, we found no evidence of a meaningful association between standardized birth weight and full-scale IQ, and birth weight explained less than 1% of the variance in IQ. There was also no indication of non-linear effects or sex-specific associations, and results were consistent across diagnostic subgroups.

Notably, despite the absence of an association between standardized birth weight and FSIQ, the overall clinical cohort demonstrated substantially lower intellectual functioning than the normative population. Mean FSIQ across the entire sample was approximately 9 IQ points below the normative mean of 100, and mean FSIQ was also significantly below the population norm within the ASD, ADHD, and ID groups. These findings indicate that the cohort represents a clinically affected population with broadly reduced cognitive functioning, even though variation in birth weight within the normal range did not explain individual differences in intellectual ability. This further suggests that factors other than subtle variation in fetal growth, including genetic, neurobiological, and environmental influences, are likely to play a more prominent role in shaping cognitive outcomes among clinically referred children and adolescents.

Extending beyond cognitive outcomes, standardized birth weight did not differ significantly between the ASD, ADHD, and ID groups. Furthermore, neither the ASD, ADHD, nor ID groups differed significantly from the population-based MoBa reference sample. These findings suggest that, when excluding premature and low birth-weight infants, children and adolescents referred for clinical assessment of NDDs did not deviate systematically from population norms in birth weight after accounting for gestational age and sex. Taken together, the findings indicate that variation in birth weight within the normal term and weight has limited relevance for distinguishing between neurodevelopmental diagnostic categories in clinically referred populations.

Interestingly, participants without registered ASD, ADHD, or ID diagnoses showed significantly lower standardized birth weight compared with the MoBa reference sample and the ASD group. Although these participants did not meet criteria for the diagnostic categories examined in the present study, they had nevertheless been referred for clinical assessment, suggesting the presence of developmental, behavioral, cognitive, or psychiatric concerns. However, the subgroup was relatively small, and the finding should therefore be interpreted cautiously. One possible explanation is that these participants may nevertheless have presented with clinically relevant developmental concerns that prompted referral to specialized mental health services, despite not meeting criteria for ASD, ADHD, or ID at the time of assessment. Lower birth weight may therefore reflect broader neurodevelopmental vulnerability that is not fully captured by the diagnostic categories examined in the present study. The absence of formal ASD, ADHD, or ID diagnoses does not necessarily indicate typical neurodevelopment. In clinically referred populations, substantial functional difficulties may still be present even when categorical diagnostic criteria are not fulfilled.

It is also important to consider that the finding may partly reflect statistical instability related to the relatively small sample size of the no-NDD group. Confidence intervals were comparatively wide, and several participants in this subgroup had relatively low standardized birth weight values, contributing to increased variability within the group. Consequently, the observed difference may reflect sampling variability rather than a robust or clinically meaningful association. Replication in larger clinically characterized samples will therefore be important to determine whether this pattern reflects a genuine association between lower birth weight and broader developmental vulnerability outside traditional NDD categories.

The current findings differ somewhat from large-scale epidemiological studies reporting robust associations between low birth weight and neurodevelopmental risk (Cortese et al., 2021). However, much of the established literature has focused on infants born preterm or with clinically significant growth restriction (e.g., < 2500 g or SGA) (Peyre et al., 2026; Shenkin et al., 2004). Longitudinal studies have consistently shown that SGA is associated with lower IQ, poorer language abilities, and reduced academic achievement extending into adolescence and adulthood (Guellec et al., 2011). More recently, a large longitudinal study combining four birth cohorts demonstrated that the association between birth weight standardized for gestational age and IQ is non-linear, with cognitive performance increasing up to approximately the 69th birth weight percentile before reaching a plateau, without evidence that higher

birth weight became detrimental to cognitive functioning (Eves et al., 2023). The same study further showed that gestational age was independently associated with IQ, with the steepest gains occurring before 32 weeks' gestation and more gradual increases up to 41 weeks (Eves et al., 2023). Together, these findings suggest that both optimal fetal growth and longer gestation contribute to cognitive development, although the cognitive benefit of increasing birth weight appears to plateau within the normal range. By restricting the present study to term-born children with birth weights between 2500 and 4500 g, we deliberately excluded participants at the extremes of gestational age and fetal growth, where previous studies have reported the strongest associations with cognitive outcomes. Consequently, our findings are consistent with the notion that the birth weight–IQ relationship may primarily be driven by the lower end of the birth weight distribution, whereas relatively small differences in birth weight within the normal term range have limited influence on intellectual functioning. The absence of both linear and non-linear associations in the present study further suggests that subtle variation in fetal growth among term-born children may contribute little to cognitive variability within clinically referred populations.

The absence of consistent differences between diagnostic categories highlights the heterogeneity of neurodevelopmental conditions. NDDs arise from complex interactions between genetic, prenatal, and environmental factors. Birth weight, while a useful proxy marker of prenatal conditions, may not capture the multifactorial pathways that contribute to diagnostic status in clinically ascertained cohorts. Similarly, cognitive outcomes are influenced by complex and heterogeneous neurodevelopmental mechanisms, including genetic variation, atypical brain connectivity, perinatal complications, and environmental influences. In this context, relatively subtle birth weight variations within the normal range represent a crude marker of early neurodevelopmental vulnerability and were insufficient to capture the multifactorial determinants of intellectual functioning.

From a clinical perspective, these findings suggest that birth weight within the normal term range (2500–4500 g) has limited standalone predictive value for intellectual functioning among children referred for NDD assessment. While extreme deviations in birth weight remain well-established indicators of neurodevelopmental vulnerability, variation within typical limits appears to contribute minimally to risk stratification in this study. Clinicians should therefore interpret birth weight alongside a broader constellation of developmental, genetic, neurological, and psychosocial factors. In a specialized clinical setting where baseline neurodevelopmental risk is already elevated, modest differences in birth weight may offer little additional explanatory value.

Our findings did not demonstrate a consistent association between standardized birth weight and specific neurodevelopmental diagnoses. Thus, while Cortese et al. (2021) identified a population-level gradient of risk across the birth weight distribution, our results suggest that within a diagnostically enriched clinical cohort, variation in birth weight within the normal range may have limited discriminatory value.

One potential explanation for this discrepancy lies in differences in study design and population structure. Cortese et al. (2021) analyzed a general population cohort, where variation in birth weight

likely captures meaningful differences in fetal growth and associated biological vulnerability across the full risk spectrum. In such settings, modest reductions in birth weight may contribute incrementally to the probability of neurodevelopmental impairment. In contrast, clinically referred samples are already enriched for genetic liability, neurobiological alterations, and developmental concerns. Within this higher-risk context, the relative contribution of subtle differences in fetal growth may be overshadowed by stronger etiological factors, reducing the impact of birth weight variation within normative limits.

Our findings can be further contextualized in relation to previous findings that fetal growth exerts measurable associations with total brain volume and white matter organization across the lifespan, even within the normal birth weight range (Walhovd et al., 2024). Taken together, these studies support a model in which birth weight indexes early neurobiological development along a continuum: lower birth weight reflects subtle alterations in brain maturation that may increase vulnerability at the population level. However, the population in the current study may be characterized by substantial genetic and neurodevelopmental heterogeneity which may not translate into detectable differences in cognitive performance within the normal birth weight range. Thus, while fetal growth appears to contribute to neurodevelopmental variability, its relative impact may depend on the broader etiological context in which development unfolds.

A key strength of this study is the use of standardized birth weight (Z-scores), allowing adjustment for gestational age and sex and enabling a more precise assessment of fetal growth relative to expected norms. The study also extends previous research by focusing on a heterogeneous clinical population, providing insights beyond population-based samples.

Several limitations of the present study should be acknowledged. First, intellectual functioning was assessed using different versions of the Wechsler intelligence scales, selected according to participants' age at assessment. While all Wechsler instruments are standardized and designed to measure general intellectual ability, the use of multiple test versions may introduce measurement variability across age groups. In addition, the clinical groups differed somewhat in mean age at assessment, which may have contributed additional heterogeneity in cognitive performance. In preschool-aged children and in clinical populations, cognitive profiles are often uneven, with marked variability across domains. Consequently, while FSIQ provides a global estimate of intellectual ability, interpretation should consider individual index scores and qualitative aspects of test performance, particularly in children with suspected or diagnosed NDDs. These factors may contribute to increased heterogeneity in FSIQ measurement and should be considered when interpreting the findings. Furthermore, potential effects of fetal growth on neurodevelopment may become more apparent later in development as cognitive, academic, and social demands increase. While no association between standardized birth weight and FSIQ was observed in the present study, subtle developmental effects may emerge more clearly in outcomes such as school achievement, adaptive functioning, educational attainment, or occupational functioning across adolescence and adulthood. Longitudinal studies following individuals across developmental stages may therefore provide a more sensitive framework for understanding the long-term implications of variation in birth weight within the normal range. The clinical groups also differed somewhat in mean age

at assessment, with participants in the ID and no-diagnosis groups being somewhat older on average than those in the ASD and ADHD groups. Although these differences were modest, age-related variation in cognitive and adaptive demands may nevertheless have influenced the expression and detection of neurodevelopmental difficulties.

Next, the sample was characterized by a marked overrepresentation of males, reflecting the higher prevalence of NDDs among boys (Bölte et al., 2023). Nevertheless, this sex imbalance may restrict the generalizability of the findings, particularly with respect to female cognitive and developmental trajectories. Future longitudinal studies would allow examination of whether subtle birth weight differences within the normal range influence developmental trajectories over time. Investigating interactions with socioeconomic factors, early intervention exposure, and genetic variants and risk profiles may further clarify under which conditions birth weight contributes to cognitive variability.

In conclusion, among term-born children and adolescents referred for neurodevelopmental assessment, standardized birth weight within the normal range was not associated with intellectual functioning and did not meaningfully distinguish between ASD, ADHD, and ID groups. Although the clinical cohort demonstrated substantially lower intellectual functioning than the normative population, with mean FSIQ approximately 9 IQ points below the population mean, variation in standardized birth weight within the normal range did not explain individual differences in cognitive performance. These findings suggest that factors other than subtle variation in fetal growth, including genetic, neurobiological, and environmental influences, are likely to play a more prominent role in shaping intellectual functioning among clinically referred children and adolescents.

Participants without registered ASD, ADHD, or ID diagnoses showed lower standardized birth weight than the MoBa reference sample and the ASD group; however, this finding should be interpreted cautiously given the small size and clinical heterogeneity of this subgroup. Overall, the findings suggest that variation in birth weight within the normal range has limited independent relevance for cognitive functioning and neurodevelopmental diagnostic classification in clinically referred populations. They further support the view that previously reported associations between birth weight and neurodevelopment are primarily driven by preterm birth, clinically significant fetal growth restriction, or the lower tail of the birth weight distribution, rather than by subtle variation within normative birth weight ranges.

Declarations

Declaration of competing interest

The authors declare that they have no competing interests.

Clinical trial number:

Not applicable

Funding

This study was funded by Stiftelsen K.G. Jebsen (SKGJ-MED-021), the Department of Psychology, University of Oslo, NordForsk through the funding to Leveraging Nordic Opportunities to Transform Mental Health Care through Precision Medicine (164218), the Research Council of Norway (273345, 288083, 300767, 323951, 324499, 335258), and the South-Eastern Norway Regional Health Authority (2020060, 2025037).

Author Contribution

C.D.: Writing – original draft, Formal analysis, Visualization, Data curation, Investigation, Methodology, Conceptualization. E.D.B.: Writing – review & editing, Methodology, Data curation, Conceptualization. K.K.: Writing – review & editing, Data curation. D.A.: Writing – review & editing, Methodology. T.N.: Resources, Writing – review & editing. L.T.W.: Writing – review & editing, Supervision, Methodology, Formal analysis, Conceptualization. O.A.A.: Resources, Writing – review & editing.

Acknowledgement

We are grateful for the participation by the individuals consenting to be part of the BUPgen registry. We also thank the families who participate in the Norwegian Mother, Father and Child Cohort Study (MoBa) for their contribution to this ongoing research. This study was funded by Stiftelsen K.G. Jebsen (SKGJ-MED-021), the Department of Psychology, University of Oslo, NordForsk through the funding to Leveraging Nordic Opportunities to Transform Mental Health Care through Precision Medicine (164218), the Research Council of Norway (273345, 288083, 300767, 323951, 324499, 335258), and the South-Eastern Norway Regional Health Authority (2020060, 2025037).

Data Availability

The data used in this study is not publicly available due to ethical and legal restrictions related to sensitive clinical and registry data. Access to the data may be granted upon reasonable request and with appropriate ethical and institutional approvals.

References

1. Brandlistuen RE, Kristjansson D, Alsaker E, Valen R, Birkeland E, Røyrvik EC, Page CM, Aamelfot M, Vangbæk S, Ask H, Havdahl A, Brantsæter AL, Rortveit G, Håberg SE, Magnus P. Cohort Profile

- Update: The Norwegian Mother, Father and Child Cohort (MoBa). *Int J Epidemiol*. 2025;54(5). <https://doi.org/10.1093/ije/dyaf139>.
2. Burger RJ, Gordijn SJ, Mol BW, Ganzevoort W, Ravelli ACJ. Birth-weight centile at term and school performance at 12 years of age: linked cohort study. *Ultrasound Obstet Gynecol*. 2023;61(4):458–65. <https://doi.org/https://doi.org/10.1002/uog.26160>.
 3. Bölte S, Neufeld J, Marschik PB, Williams ZJ, Gallagher L, Lai MC. Sex and gender in neurodevelopmental conditions. *Nat Rev Neurol*. 2023;19(3):136–59. <https://doi.org/10.1038/s41582-023-00774-6>.
 4. Catena A, Martínez-Zaldívar C, Diaz-Piedra C, Torres-Espínola FJ, Brandi P, Pérez-García M, Decsi T, Koletzko B, Campoy C. On the relationship between head circumference, brain size, prenatal long-chain PUFA/5-methyltetrahydrofolate supplementation and cognitive abilities during childhood. *Br J Nutr*. 2019;122(s1):S40–8. <https://doi.org/10.1017/S0007114516004281>.
 5. Cortese M, Moster D, Wilcox AJ. Term Birth Weight and Neurodevelopmental Outcomes. *Epidemiology*. 2021;32(4):583–90. <https://doi.org/10.1097/ede.0000000000001350>.
 6. Eves R, Wolke D, Spiegler J, Lemola S. Association of Birth Weight Centiles and Gestational Age With Cognitive Performance at Age 5 Years. *JAMA Netw Open*. 2023;6(8):e2331815. <https://doi.org/10.1001/jamanetworkopen.2023.31815>.
 7. Guellec I, Lapillonne A, Renolleau S, Charlaluk ML, Roze JC, Marret S, Vieux R, Monique K, Ancel PY. Neurologic outcomes at school age in very preterm infants born with severe or mild growth restriction. *Pediatrics*. 2011;127(4):e883–891. <https://doi.org/10.1542/peds.2010-2442>.
 8. Heinonen K, Eriksson JG, Lahti J, Kajantie E, Pesonen AK, Tuovinen S, Osmond C, Raikonen K. Late preterm birth and neurocognitive performance in late adulthood: a birth cohort study. *Pediatrics*. 2015;135(4):e818–825. <https://doi.org/10.1542/peds.2014-3556>.
 9. Katzman R, Terry R, DeTeresa R, Brown T, Davies P, Fuld P, Renbing X, Peck A. Clinical, pathological, and neurochemical changes in dementia: a subgroup with preserved mental status and numerous neocortical plaques. *Ann Neurol*. 1988;23(2):138–44. <https://doi.org/10.1002/ana.410230206>.
 10. Larroque B, Ancel PY, Marret S, Marchand L, André M, Arnaud C, Pierrat V, Rozé JC, Messer J, Thiriez G, Burguet A, Picaud JC, Bréart G, Kaminski M. Neurodevelopmental disabilities and special care of 5-year-old children born before 33 weeks of gestation (the EPIPAGE study): a longitudinal cohort study. *Lancet*. 2008;371(9615):813–20. [https://doi.org/10.1016/s0140-6736\(08\)60380-3](https://doi.org/10.1016/s0140-6736(08)60380-3).
 11. Leonard H, de Klerk N, Bourke J, Bower C. Maternal health in pregnancy and intellectual disability in the offspring: a population-based study. *Ann Epidemiol*. 2006;16(6):448–54. <https://doi.org/10.1016/j.annepidem.2005.05.002>.
 12. Morris-Rosendahl DJ, Crocq MA. Neurodevelopmental disorders-the history and future of a diagnostic concept. *Dialogues Clin Neurosci*. 2020;22(1):65–72. <https://doi.org/10.31887/DCNS.2020.22.1/macrocq>.
 13. National Institute for Health and Care Excellence. (2019). *Autism*. NICE. Retrieved 24.05.2026 from <https://www.nice.org.uk/guidance/conditions-and-diseases/mental-health-behavioural-and->

14. Parenti I, Rabaneda LG, Schoen H, Novarino G. Neurodevelopmental Disorders: From Genetics to Functional Pathways. *Trends Neurosci.* 2020;43(8):608–21.
<https://doi.org/10.1016/j.tins.2020.05.004>.
15. Peyre H, Rios P, Botton J, Herlemont P, Olié V, Miranda S, Zureik M, Weill A, Dray-Spira R. Shared and disorder-specific prenatal and perinatal risk factors for neurodevelopmental disorders: a nationwide cohort study. *Mol Psychiatry.* 2026. <https://doi.org/10.1038/s41380-026-03607-2>.
16. Raznahan A, Greenstein D, Lee NR, Clasen LS, Giedd JN. Prenatal growth in humans and postnatal brain maturation into late adolescence. *Proc Natl Acad Sci U S A.* 2012;109(28):11366–71.
<https://doi.org/10.1073/pnas.1203350109>.
17. Shenkin SD, Starr JM, Deary IJ. Birth weight and cognitive ability in childhood: a systematic review. *Psychol Bull.* 2004;130(6):989–1013. <https://doi.org/10.1037/0033-2909.130.6.989>.
18. Volpe JJ. Brain injury in premature infants: a complex amalgam of destructive and developmental disturbances. *Lancet Neurol.* 2009;8(1):110–24. [https://doi.org/10.1016/s1474-4422\(08\)70294-1](https://doi.org/10.1016/s1474-4422(08)70294-1).
19. Walhovd KB, Fjell AM, Brown TT, Kuperman JM, Chung Y, Hagler DJ Jr., Roddey JC, Erhart M, McCabe C, Akshoomoff N, Amaral DG, Bloss CS, Libiger O, Schork NJ, Darst BF, Casey BJ, Chang L, Ernst TM, Frazier J, Dale AM. Long-term influence of normal variation in neonatal characteristics on human brain development. *Proc Natl Acad Sci U S A.* 2012;109(49):20089–94.
<https://doi.org/10.1073/pnas.1208180109>.
20. Walhovd KB, Krogsrud SK, Amlie IK, Sørensen Ø, Wang Y, Bråthen ACS, Overbye K, Kransberg J, Mowinckel AM, Magnussen F, Herud M, Håberg AK, Fjell AM, Vidal-Pineiro D. (2024). Fetal influence on the human brain through the lifespan. *Elife*, 12. <https://doi.org/10.7554/eLife.86812>
21. Wechsler D. Wechsler intelligence scale for children. 4th ed. Psychological Corporation; 2003.
22. Wechsler D. Wechsler Adult Intelligence Scale–Fourth Edition (WAIS-IV). APA PsycTests. 2008.
<https://doi.org/10.1037/t15169-000>.
23. Wechsler D. Wechsler preschool and primary scale of intelligence. 4th ed. The Psychological Corporation; 2012.
24. World Health Organization. International statistical classification of diseases and related health problems. 10th ed. World Health Organization; 2016.
25. World Health Organization. (2025). *Global nutrition targets 2030: low birth weight brief*.
<https://www.who.int/publications/i/item/B09402>
26. Zhao Y, Yang L, Gong G, Cao Q, Liu J. Identify aberrant white matter microstructure in ASD, ADHD and other neurodevelopmental disorders: A meta-analysis of diffusion tensor imaging studies. *Prog Neuropsychopharmacol Biol Psychiatry.* 2022;113:110477.
<https://doi.org/10.1016/j.pnpbp.2021.110477>.

Figures

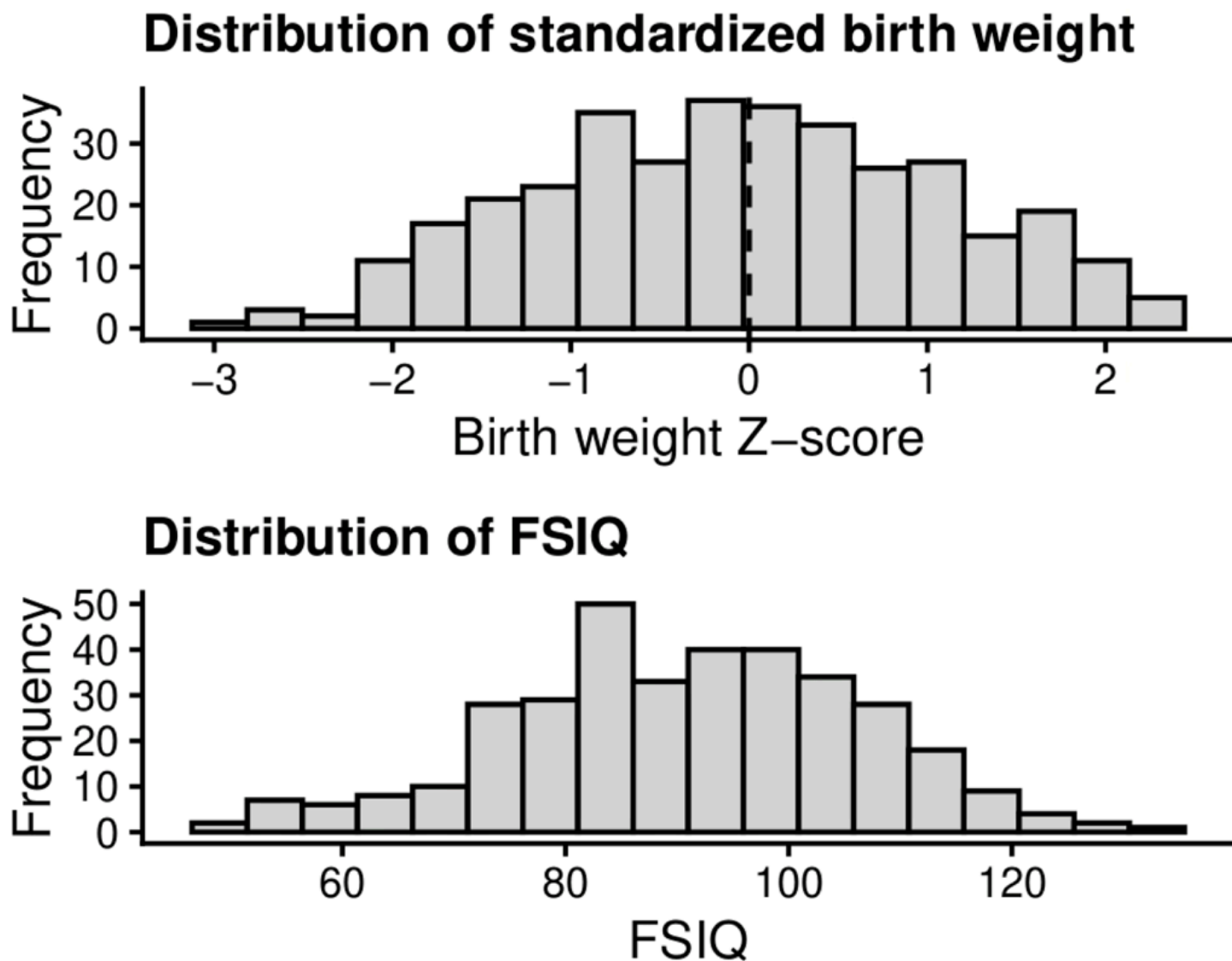


Figure 1

Distribution of standardized birth weight (Z-score) and FSIQ in the BUPgen sample (n=349).

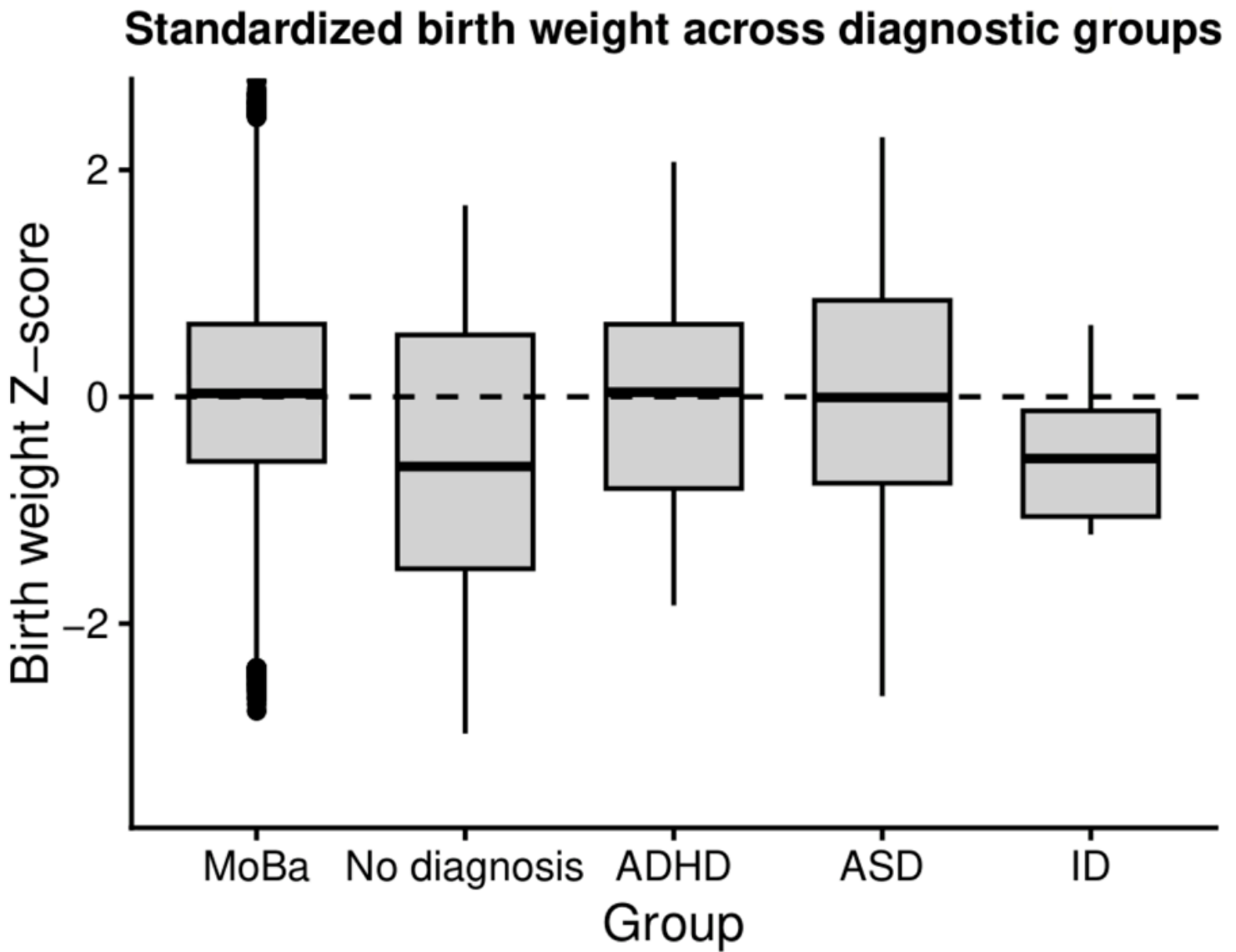


Figure 2

Distribution of standardized birth weight (Z-score) across MoBa and BUPgen participants without registered NDD ($n = 26$), ADHD ($n = 68$), ASD ($n = 248$), and ID ($n = 7$). Boxes represent interquartile ranges, horizontal lines within boxes indicate medians, and whiskers represent the range of the data. The dashed horizontal line indicates the expected population mean ($Z = 0$).

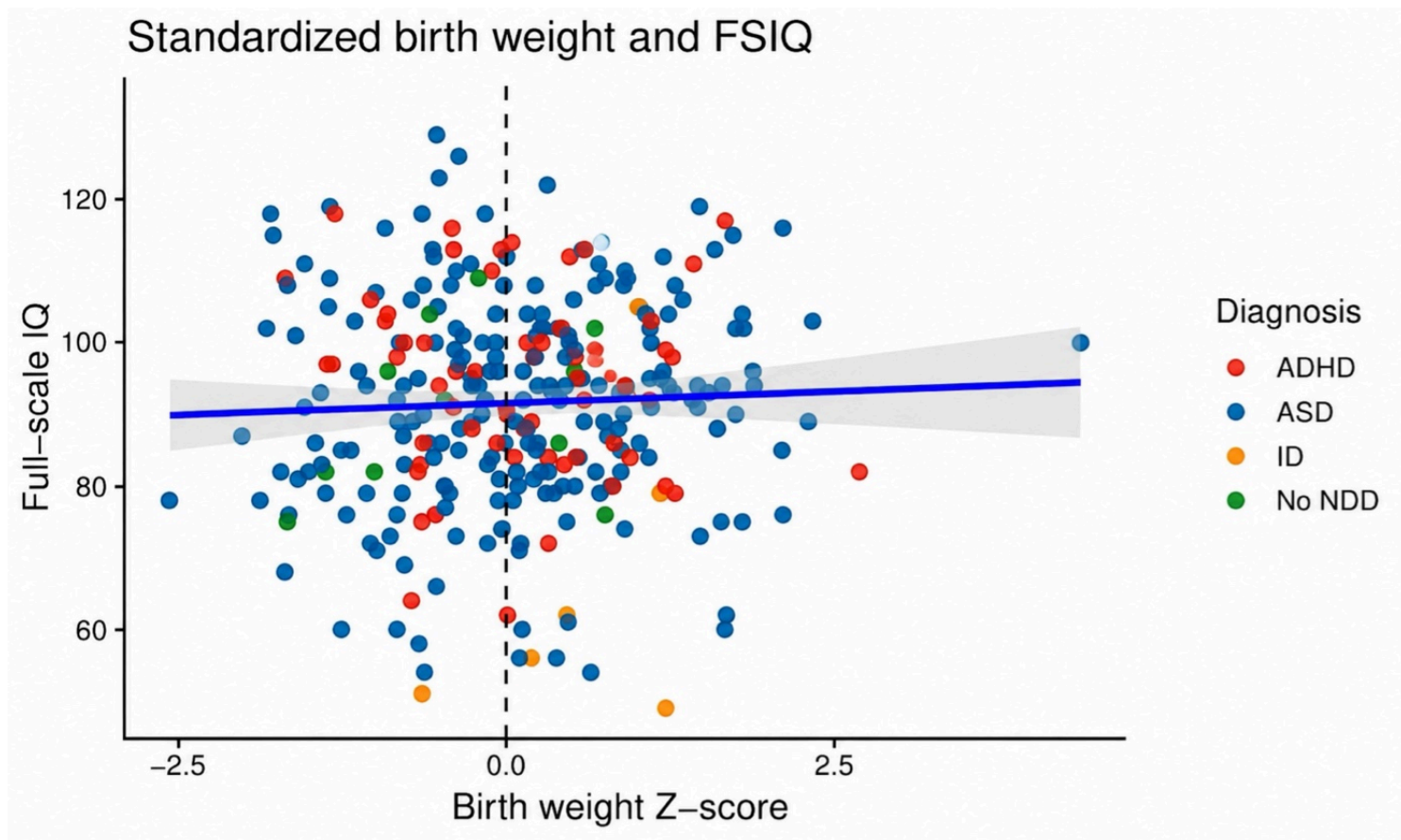


Figure 3

Scatterplot showing the association between standardized birth weight (Z-score) and full-scale IQ (FSIQ) in the BUPgen cohort. Each point represents one participant and is colour-coded according to diagnostic group (ASD, ADHD, ID, or no registered neurodevelopmental disorder [NDD]). The solid blue line represents the fitted linear regression, with the shaded area indicating the 95% confidence interval. The vertical dashed line denotes the expected birth weight for gestational age and sex (Z-score = 0).