



RESEARCH ARTICLE

URL article: <http://jurnal.fkmumi.ac.id/index.php/woh/article/view/woh9315>

The Effect of Birth History on Speech Delay in Toddlers

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ABSTRACT

Speech delay is one of the most common developmental disorders among toddlers. Globally, speech and language delays affect approximately 5% to 12% of preschool-aged children, making communication disorders among the most prevalent neurodevelopmental concerns worldwide explicitly framed speech delay as a public health concern affecting long-term educational attainment and neurodevelopment affecting approximately 5–10% of preschool children in Indonesia, and it has the potential to negatively impact children's intellectual functioning and social interaction. The high prevalence indicated by more than ten toddlers receiving speech therapy daily in services in Tegal and Brebes highlights the need to identify underrecognized risk factors, such as birth history. While prior work focuses on environmental and parenting factors, birth history variables (perinatal factors) remain understudied locally. This study aimed to analyze the relationship between birth history factors (gestational age, mode of delivery, birth weight, and maternal age at childbirth) and the occurrence of speech delay in toddlers. This quantitative analytic study employed a case control design (1:1 ratio) involving a total of 110 participants (55 cases and 55 controls). The inclusion criteria for mothers who had children aged 2–6 years with speech delay, whereas the control group were mothers who had children aged 2–6 years without speech delay, selected through purposive sampling at RSIA Permata Insani Brebes (cases) and KB Tabiyatul Khasanah (controls). Data were collected from June to August 2025. Birth history data were collected through interviews and questionnaires, adapted from a previously validated and reliable instrument, and data were analyzed using the chi-square test and multivariate logistic regression with the enter method. The results showed a significant association between gestational age (<37weeks or >40weeks) and speech delay ($p=0.002$; $OR=3.586$), cesarean section delivery ($p=0.028$; $OR=2.260$), and abnormal birth weight (<2500g or >4000g) ($p=0.045$; $OR=2.286$). However, no significant association was found between maternal age at childbirth (<20years or >35years) and the occurrence of speech delay ($p=0.339$). In conclusion, Birth history factors may assist clinicians in identifying toddlers at higher risk for speech delay and prioritizing developmental surveillance.

Keywords : Birth History; Speech Delay; Toddlers

PUBLISHED BY :

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Universitas Muslim Indonesia

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Article history

Received 19 February 2026

Received in revised form 27 July 2026

Accepted 18 August 2026

Available online 1 September 2026

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INTRODUCTION

Speech and language development are crucial indicators of a child's intelligence and overall growth, as well as a primary asset for social interaction(1),(2). Speech delay, characterized by a child's failure to attain standardized developmental milestones (e.g., WHO, CDC, and AAP criteria) and restricted vocabulary acquisition, represents a major global neurodevelopmental concern(3),(4). Globally, speech and language delays affect 5%–12% of preschool-aged children, with regional prevalence rates reported at 5%–8% in the United States and 5%–10% in both Southeast Asia and Indonesia(5). Given its long-term adverse impacts on academic and social integration, epidemiological investigations are imperative to map underlying risk factors and establish timely public health interventions(6).

High clinical service utilization across speech and occupational therapy centers in the Tegal and Brebes regions where over ten toddlers receive daily therapy reflects a substantial local demand for speech delay interventions. However, timely intervention is frequently hindered by parental misconceptions that speech delays will self-correct over time. This lack of early recognition leads to missed therapeutic opportunities during the critical "window of opportunity" of peak neuroplasticity. Consequently, delayed identification significantly elevates the risk of secondary developmental deficits, including persistent communication impairments, reduced academic readiness, emotional-behavioral difficulties, and compromised social integration upon school entry.

Toddlers who experience speech and language delay may suffer from decreased intellectual function and cognitive ability, be unable to keep up with their peers, experience difficulties in communication and interaction due to abnormalities in the speech organs, and encounter challenges in comprehension and socialization(7). A child is considered to have speech delay when, at the age of 2–3 years, they are not yet able to speak fluently(8). Early speech delays compromise phonological awareness, vocabulary acquisition, and narrative comprehension, thereby impairing foundational literacy skills and school readiness. The inability to verbally express internal states and distress induces communicative frustration, manifesting as externalizing behaviors such as temper tantrums, aggression, anxiety, and social withdrawal. Furthermore, impaired cooperative play skills increase vulnerability to peer rejection and social isolation in early educational settings. Without timely intervention during peak neuroplasticity, these early communication deficits tend to persist into later childhood and adolescence.

Based on a review of several articles, numerous factors influence the occurrence of speech delay in children, including internal and external factors. Internal factors include physical disabilities, sex, and preterm birth. External factors include parenting style, maternal education, family socioeconomic status, lack of parent–child communication, and excessive use of gadgets(9). In this study, we focused on birth history factors, as these factors are often underrecognized as causes of speech delay. Most previous studies have emphasized external factors because they are more commonly encountered in children's daily lives. However, birth history is also known to increase the risk of speech delay, such as preterm birth, low birth weight (LBW), and birth-related injuries, including oxygen deprivation (hypoxia).

Complications or medical conditions during pregnancy and childbirth may cause brain damage or neurodevelopmental disturbances, which subsequently have a negative impact on children's speech and language abilities. According to Nestria Budiasih, speech delay is more frequently caused by prenatal and perinatal factors, such as prematurity and low birth weight. Children born prematurely have a higher risk of developing neurodevelopmental disorders(10). Other studies have found that these perinatal factors do not show a significant independent association after controlling for environmental variables. Methodological limitations in previous studies further complicate the issue; most research has employed cross-sectional designs—which are susceptible to temporal ambiguity bias—relied on limited sample sizes, or analyzed single birth-history variables in isolation without controlling for confounding factors. Empirical evidence regarding perinatal determinants in the Indonesian population remains very limited, as most local research has focused almost exclusively on parenting factors and screen time, resulting in a scarcity of perinatal data. To date, no study has simultaneously evaluated the four key factors gestational age, birth weight, mode of delivery, and maternal age at birth within a single case-control analysis model.

In this study, the authors examined the relationship between birth history and the occurrence of speech delay. The birth history variables analyzed included gestational age, mode of delivery, birth weight, and maternal age at childbirth as independent variables, with speech delay as the dependent variable. This study aimed to not only to determine the association between birth history and speech delay but also to quantify the magnitude of risk associated with each perinatal factor to support early developmental screeningdetermine the magnitude of the risk posed by birth history factors for speech delay in children. Furthermore, early identification of a child's birth history is expected to encourage parents to provide early intervention to prevent the occurrence of speech delay

METHOD

This study employed a quantitative analytic approach using a case-control design. The population consisted of all subjects (humans, laboratory data, etc.) who met the predetermined characteristics(11) The study population was divided into two groups: a case group, comprising parents of children aged 2–6 years who experienced speech delay, and a control group, comprising parents of children aged 2–6 years without speech delay. This study employed a quantitative analytic approach using a case-control design. The population consisted of all subjects (humans, laboratory data, etc.) who met the predetermined characteristics (9) The study population was divided into two groups: a case group, comprising parents of children aged 2–6 years who experienced speech delay, and a control group, comprising parents of children aged 2–6 years without speech delay. The sample size was determined using the formula for calculating sample sizes for paired or independent case-control studies. Sample determination was based on the following parameters: 1) Confidence Level: 95% ($\alpha=0.05$, $Z_{1-\alpha/2} = 1.96$); 2) Statistical Power ($1-\beta$): 80% ($Z_{1-\beta}=0.84$); 3) Estimated Odds Ratio (OR): a minimum OR of 2.5 (based on existing literature regarding the association between prematurity/low birth weight and speech delay); and 4) Proportion of Exposure in the Control Group (P_2): estimated at 15%–20% based

on data regarding complicated birth histories in the general population. Based on these parameter assumptions, the minimum required sample size was 48 subjects per group. To account for potential missing data or participant dropouts, the sample size was increased by approximately 15%, resulting in a total of 110 participants (55 in the case group and 55 in the control group). This study has several limitations that should be considered when interpreting the results. First, the use of different recruitment settings for the case group (hospitals/clinics) and the control group (early childhood education centers) could potentially introduce selection bias. The population reached in hospital settings may differ from the community- or education-based population in terms of health awareness, access to medical services, or socioeconomic status.

The inclusion criteria for the case group were mothers who had children aged 2–6 years diagnosed with speech delay, whereas the inclusion criteria for the control group were mothers who had children aged 2–6 years without speech delay. Children were classified as having speech delay based on a diagnosis by a physician or speech therapist, indicating failure to achieve age-appropriate speech and language developmental milestones. Case group participants were recruited from RSIA Permata Insani Brebes. Conversely, children were classified as having normal speech and language development if they had achieved age-appropriate speech and language milestones; control group participants were recruited from KB Tabiyatul Khasanah. Exclusion criteria for the case group included children with the following conditions: autism, cerebral palsy, hearing impairment, Down syndrome, intellectual disability, and congenital neurological disorders. Birth history data were obtained through interviews and questionnaires, and the birth history was verified using the Maternal and Child Health (MCH) handbook. Data were collected from June to August 2025. Birth history factors examined in this study, including gestational age at birth, mode of delivery, birth weight, and maternal age at childbirth, were obtained through interviews and questionnaire completion by the participants. Univariate analysis was used to describe participant characteristics. Associations between independent variables and speech delay were examined using the Chi-square test. Variables with $p \leq 0.25$ in the bivariate analysis were included in a multivariate logistic regression model using the enter method. Statistical significance was set at $p < 0.05$ with 95% confidence intervals. Ethical approval was obtained from the Health Research Ethics Committee of Poltekkes Kemenkes Semarang (No. 816/EA/F.XXIII.38/2025).

RESULTS

To identify birth history factors associated with speech To identify birth history factors associated with speech delay

Table 1 shows that respondents with a gestational age of <37 weeks or >40 weeks were more prevalent in the speech delay group (47.30%) compared to the non-speech delay group (20.00%). The Chi-square test indicated a significant association between gestational age and the occurrence of speech delay ($p = 0.045$). The Odds Ratio (OR) analysis showed that respondents with a gestational age of <37 weeks or >40 weeks had a 3.586 times higher risk of speech delay in their children compared to those with a gestational age of 37–40 weeks (95% CI: 1.53–8.36)

Table 1. Factors Related to Birth History

Variable	Speech delay		OR 95%CI	P value
	yes	No.		
Gestational Age				
<37 / >40 weeks	26 (47.30%)	11 (20.00%)	3.586	0.002
37-40 weeks	29 (52.70%)	44(80.00%)	(1,53 -8,36)	
Mode of Delivery				
SC	31 (56.40%)	20 (36.40%)	2.260	0.028
Normal	24 (43.60%)	35 (63.60%)	(1.051-4.859)	
Birth Weight				
<2500 dan >3500	20 (36.40%)	11 (20.00%)	2.286	0.045
2500-3500	35 (63.60%)	44 (80.00%)	(0.968-5.398)	
Maternal Age at Childbirth				
<20/>35 years	18(54.50%)	15(45.50%)	1.29(0.572-	0.339
20 – 35 years	37(48.10%)	40(51.90%)	2.940)	

Respondents with a history of cesarean section delivery were more common in the speech delay group (56.40%) than in the non–speech delay group (36.40%). The Chi-square test revealed a significant association between the mode of delivery and speech delay ($p = 0.028$). The OR analysis indicated that respondents with a history of cesarean section delivery had a 2.260 times higher risk of having children with speech delay compared to those who had normal vaginal delivery (95% CI: 1.051–4.859).

Respondents whose children had a birth weight of <2500 g or >4000 g were more frequently found in the speech delay group (36.40%) than in the non–speech delay group (20.00%). The Chi-square test showed a significant association between birth weight and the occurrence of speech delay ($p = 0.045$). The OR analysis demonstrated that respondents with a history of abnormal birth weight had a 2.286 times higher risk of speech delay compared to those with a birth weight of 2500–4000 g (95% CI: 0.968–5.398).

In contrast, respondents who were <20 years or >35 years of age at childbirth were more prevalent in the speech delay group (54.50%) than in the non–speech delay group (45.50%). However, the Chi-square test indicated no significant association between maternal age at childbirth and the occurrence of speech delay ($p = 0.339$). The OR analysis showed that maternal age <20 years or >35 years was associated with a 1.29 times higher risk of speech delay compared to maternal age of 20–35 years (95% CI: 0.572–2.940).

The influence of birth history factors on speech delay

Based on Table 2. Logistic regression analysis results indicate that birth history, specifically gestational age and delivery history, is significantly associated with the occurrence of speech delay in children. Conversely, birth weight did not show a significant association. Among these factors, gestational age and delivery history were identified as the most dominant factors influencing speech delay.

Table 2. Results of logistic regression test analysis

No	Variable	B	S.E.	Wald	Df	Sig.	Exp.(B)	CI 95%
1	Gestational Age	-4.409	0.456	9.544	1	0.002	0,244	0.100 - 0.366
2	Mode of Delivery	-0,964	0.423	5,199	1	0.023	0,381	0.166 - 0.873
3	Birth Weight	-0.856	0.467	3.440	1	0.064	0.420	0.168 - 1.050
4	Constant	1.152	0.366	9.904	1	0.002	3.165	

DISCUSSION

Gestational Age Factor

This study highlights a statistically significant association between gestational age and speech delay in children ($p = 0.002$). Odds Ratio analysis indicated that children with non-optimal gestational ages (<37 weeks or >40 weeks) had a 3.586 times higher risk of speech delay compared to those born at full term (37–40 weeks). Furthermore, the persistent significance of this association in multivariate regression analysis confirms gestational age as a strong independent risk factor for speech development. The results of this study align with research by Zambrana et al. (2021), which found that prematurity (gestational age <37 weeks) is causally linked to an increased risk of language delay up to the age of 5, even after controlling for genetic and family environment confounders ($RR = 4.51$ to 10.32 at 1.5 years of age)(12). Prematurity and low birth weight significantly increase the risk of speech and language delays in two-year-old toddlers, with early cognitive impairment serving as a major, persistent predictor. This finding is consistent with other studies reporting that lower gestational age is associated with a higher risk of developmental disorders(13).

A study by Duwandani and Iskandar (2022) reported a significant relationship between a history of preterm birth and speech delay in children. Preterm birth carries a risk of neurodevelopmental dysfunction according to its classification: late preterm (35–36 weeks), moderate preterm (32–34 weeks), early preterm (<32 weeks), and extremely preterm (<28 weeks). Even late preterm infants have a significantly higher risk of long-term neurological problems, which may result in attention deficits and difficulties in transmitting, receiving, and processing information(14). Previous research has shown that children born preterm perform more poorly in language abilities compared to full-term children. These language difficulties may persist into elementary school, a period when language development becomes more stable and adult-like(15).

The findings of this study further reinforce the concept that fetal brain maturation is strongly influenced by gestational age. The third trimester represents a critical period characterized by rapid growth of cerebral cortical volume, myelination, and the formation of neuronal connectivity essential for language function. In infants born before 37 weeks of gestation, these critical processes are disrupted, resulting in suboptimal development of neural networks associated with language production (Broca's area) and language comprehension (Wernicke's area)(11).

These findings are consistent with a study by Noorlinda et al. (2025) in Banjarmasin, which examined the correlation between a history of prematurity and speech delay in toddlers. The study concluded that there was a significant association between gestational age of less than 37 weeks (prematurity) and the occurrence of speech delay ($p = 0.000$). This conclusion is supported by the theory that preterm birth disrupts crucial neurobiological maturation processes and brain plasticity, thereby affecting speech and language development. Preterm infants exhibit reduced cortical thickness and lower white matter integrity, particularly in temporoparietal regions associated with phonological processing and language skills. Disruption of the arcuate fasciculus, the primary neural pathway connecting language areas, provides a biological explanation for the persistence of speech delay in preterm infants into school age. In addition, post-term infants (>40 weeks) are at increased risk of placental insufficiency, chronic hypoxia, and meconium aspiration, all of which may adversely affect central nervous system development(16).

In addition, a study by Hassa et al. (2023) demonstrated that birth history has a significant impact on child development, including the language domain. This longitudinal study of preterm infants found that biological factors such as gestational age and low birth weight, as well as the duration of neonatal intensive care unit (NICU) stay, were correlated with cognitive and motor development scores. Although these biological factors were not the sole predictors of language development, the study emphasized that perinatal risk factors, such as prematurity, interact with environmental factors (e.g., family income and stimulation), ultimately influencing language development at 12 months of age(17).

A scoping review by Al-Bluwi et al. (2025) on the epidemiology of Developmental Language Disorder (DLD) confirms that perinatal factors specifically preterm birth and low birth weight (LBW) are primary contributors to the risk of language delay in children. Prematurity potentially impairs neurological maturation and the development of brain language centers due to sub-optimal neurodevelopmental processes. Furthermore, the risk of language impairment is exacerbated by labor-related complications, such as forceps delivery and cesarean section, as well as postnatal conditions including cyanosis, collectively indicating that gestational complications and birth trauma significantly impact central nervous system maturation.(18).

Both prematurity and post-term birth can adversely affect central nervous system maturation. In preterm infants, cortical regions involved in language production and comprehension, such as Broca's and Wernicke's areas, may not yet be fully developed. In contrast, post-term infants (>40 weeks of gestation) are at increased risk of intrauterine hypoxia due to placental aging, which may impair cognitive and language abilities. Furthermore, preterm infants often experience mild hearing impairment, sensory regulation difficulties, and delays in fine motor development, all of which are critical components of language acquisition. Environmental factors also play a role; for example, reduced stimulation during intensive care (NICU stay) may limit infants' exposure to verbal input during critical periods of language development. The study emphasized the importance of early detection, with prematurity highlighted as a key variable to be considered (19),(20).

In contrast, another study focusing on late talkers with a history of low-risk preterm birth reported different findings. A study by Suttora et al. (2020) comparing low-risk preterm late talkers with full-term late talkers at 30 months of age found no significant differences in speech and language abilities between the two groups. These results support the view that prematurity-related gestational age, particularly in low-risk cases (without major brain injury or severe neonatal complications), may have a minimal or non-significant impact on the development of speech delay(21). Previous studies demonstrating the significant impact of gestational age on language impairment have often included populations of premature infants with medical complications (high-risk). The findings of Suttora et al. confirm that prematurity itself is not the sole determinant; rather, the presence of clinical or medical complications at birth plays a more dominant role in influencing neurodevelopment.

Mode of Delivery

This study demonstrated a statistically significant association between the mode of delivery and speech delay in children ($p = 0.028$). Odds ratio analysis revealed that children delivered via Caesarean section had a 2.26-fold higher risk of experiencing speech delay compared to those born vaginally. Furthermore, the persistent significance of this association in multivariate regression analysis reinforces mode of delivery as an independent risk factor for speech development outcomes. This finding suggests that mode of delivery may serve as an important perinatal indicator related to early language outcomes. However, previous studies have reported mixed findings. One study reported that infants delivered via vaginal delivery had a 1.5-fold higher failure rate in the first Otoacoustic Emission (OAE) screening compared to infants delivered by cesarean section, indicating that vaginal delivery does not necessarily confer a universal protective effect on early auditory outcomes(22).

Although the present study demonstrates a significant relationship between cesarean delivery and speech delay, theoretically, this association is multifactorial and not entirely attributable to the cesarean procedure itself. Several international studies emphasize that the medical indications leading to cesarean delivery play a more substantial role in influencing child development than the delivery method(22).

Theoretically, the association between cesarean section and speech delay is multifactorial and cannot be fully explained by the delivery procedure itself. Several studies have shown that the medical or obstetric conditions underlying the decision to perform a cesarean delivery may contribute to child neurodevelopmental outcomes(23),(24). In this context, it is important to consider the possibility of confounding by indication or indication bias, which is when the clinical condition that is the reason for the cesarean section is also independently associated with the risk of developmental disorders in children.

Emergency or intrapartum cesarean delivery due to fetal distress differs substantially from elective cesarean delivery, both in clinical context and in the antecedent perinatal conditions. Emergency cesarean delivery occurs more frequently in situations such as fetal distress, preeclampsia, dystocia, or failure to progress in labor, conditions that themselves may be associated with adverse neonatal and

neurodevelopmental outcomes(23),(24). Therefore, when emergency and elective cesarean deliveries are combined into one group, differences in clinical characteristics and indications may obscure the true relationship between mode of delivery and speech delay. In other words, the increased risk of speech delay found in the cesarean delivery group may partly reflect the underlying medical conditions underlying the decision to perform the operation, rather than solely the cesarean procedure itself.

This is supported by a large Swedish population cohort study that distinguished between planned and intrapartum cesarean deliveries. The study showed that the association between intrapartum cesarean delivery and neurodevelopmental impairment was significantly weaker after adjusting for indications such as fetal distress, dystocia, and failed induction. The authors concluded that some of the observed associations were likely related to confounding factors, including familial factors and the clinical indication for cesarean delivery(24). Furthermore, systematic reviews and meta-analyses emphasize that elective and emergency cesarean deliveries occur in different clinical contexts and should therefore be analyzed separately when evaluating associations with neurodevelopmental outcomes(23).

Physiologically, vaginal delivery promotes the release of key hormones such as oxytocin and catecholamines, which support neonatal neurological readiness and facilitate adaptation from the intrauterine to extrauterine environment. Infants delivered by cesarean section do not experience this hormonal surge, potentially leading to slower sensory regulation and social responsiveness, both of which are strongly associated with early language development.

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A study by Pratiwi et al. (2022) further supports this explanation, reporting that complicated deliveries, including forceps-assisted delivery and emergency cesarean section, which may result in neonatal asphyxia or birth stress, are important risk factors for speech and language delay. Cesarean delivery may also disrupt early skin-to-skin contact and delayed initiation of breastfeeding, thereby reducing early verbal and sensory interactions between mother and infant. These early interactions are

well established as foundational elements in pragmatic language development and early vocalization skills(12).

Infants at risk of developmental delay are often those whose birth conditions necessitate medical interventions. Thus, while the mode of delivery itself may not be the primary causal factor, it may function as a marker of underlying neurocognitive risk. A population-based study by Tseng et al. (2023) involving very low birth weight (VLBW) infants identified severe neurological complications such as intraventricular hemorrhage (IVH) and cystic periventricular leukomalacia (PVL) as the strongest predictors of language delay(13). These severe complications often necessitate emergency operative delivery, including cesarean section, indicating that the observed association between cesarean delivery and language delay reflects the presence of high-risk perinatal conditions rather than the delivery method itself. Cesarean delivery may therefore be associated with developmental delay, including language delay, not because of the procedure alone, but due to clinical conditions prompting its use, such as fetal distress, preeclampsia, or premature rupture of membranes. These conditions increase the risk of impaired cerebral oxygenation, potentially affecting language-related brain regions. Additionally, infants delivered via cesarean section do not undergo thoracic compression during vaginal birth, increasing the risk of pulmonary fluid retention, prolonged neonatal care, and reduced early mother–infant bonding. Early bonding and timely initiation of breastfeeding have been shown to support social and sensory development, both of which are closely linked to language acquisition. Nevertheless, several studies have reported no direct association between cesarean section and language delay, suggesting that mediating factors, such as quality of environmental stimulation, mother–infant bonding, and overall pregnancy and perinatal conditions, play a crucial role in determining language outcomes(27).

Birth Weight Factor

Bivariate analysis revealed a significant association between birth weight and speech delay ($p = 0.045$), with infants born with birth weights $<2,500$ g or $>3,500$ g demonstrating a higher risk compared to those with normal birth weights ($OR = 2.286$). After adjusting for gestational age and other confounding variables using multivariate logistic regression, the association between birth weight and speech delay lost its statistical significance. This indicates that birth weight does not make a significant independent contribution to the risk of speech delay. From both biological and methodological perspectives, the strong association between gestational age and birth weight introduces the potential for collinearity within the regression model; therefore, the correlation between variables must be examined before drawing conclusions regarding their independent effects on child development.

A review of multiple studies provides substantial evidence that preterm infants exhibit poorer performance on language-related developmental indicators, with expressive language being more severely affected than receptive language. Higher parental education and family income have been identified as protective factors for language development, whereas lower birth weight and higher degrees of prematurity are consistently reported as major risk factors. Studies comparing various levels of

prematurity and birth weight have shown that children born with low birth weight perform worse on standardized language assessments than their normal-weight counterparts(28)

Birth weight is an indicator of the quality of intrauterine growth that significantly impacts a child's neurological and language development. Low birth weight (LBW)—triggered by placental insufficiency, malnutrition, or pregnancy complications—can disrupt brain structure (such as reduced gray matter volume and delayed myelination), trigger hearing or sensory impairments, and hinder oral motor control. Conversely, macrosomia (>4,000 g) increases the risk of birth trauma, neonatal hypoglycemia, and perinatal asphyxia, which can potentially impair cortical development. Overall, these findings support the fetal programming hypothesis, wherein suboptimal intrauterine conditions exert long-term effects on neurological development and language acquisition.

Nevertheless, inconsistent findings have been reported in the literature. Tan et al. (2019), in a study involving children aged 1–2 years, found that low birth weight (<2,500 g) was not significantly associated with speech delay ($p = 0.296$) in bivariate analysis and was excluded from the final multivariate model. Their results emphasized the importance of environmental stimulation, identifying gross motor delay, exclusive breastfeeding for less than six months, and excessive media exposure as the strongest predictors of speech delay. These findings suggest that the impact of low birth weight on language development may be moderated by postnatal environmental factors. Low birth weight infants often experience impaired brain growth, particularly in myelination and synaptic formation, processes that are fundamental to language development. These risks are exacerbated in infants with intrauterine growth restriction (IUGR), where inadequate oxygen and nutrient supply during gestation disrupts neurodevelopment. Conversely, macrosomic infants are more prone to delivery complications such as birth trauma, neonatal hypoglycemia, and asphyxia, all of which contribute to long-term neurodevelopmental risk(29).

Supporting this mechanism, a cross-sectional study by Rosdiana, Kesoema, Rio, and Widanarti (2024) identified a significant association between speech delay and a history of perinatal asphyxia and neurological comorbidities, including cerebral palsy and autism/ADHD. These findings reinforce the notion that birth weight alone does not operate in isolation; rather, acute perinatal complications, particularly asphyxia—which may occur in both low birth weight and macrosomic infants—constitute a critical component of birth history that directly affects neurological and language development. The study also highlighted non-medical risk factors, such as noise exposure and insufficient communicative stimulation, as additional contributors.

Furthermore, Barbosa et al. (2025) reported that gestational diabetes mellitus (GDM) exposure in utero did not significantly predict receptive language impairment. This lack of association was attributed to effective clinical management of GDM, as evidenced by comparable birth weights between exposed and non-exposed groups. These findings suggest that birth weight contributes to speech delay primarily when prenatal conditions are severe or poorly managed, whereas well-controlled prenatal conditions may mitigate its adverse impact(30).

Although a number of studies suggest an association between low birth weight, prematurity, and language development, findings in the literature are not completely consistent. A systematic review reported that prematurity and low birth weight were generally associated with poorer language performance in children, but results between studies showed heterogeneity in the magnitude of the association(28). Contrary findings were reported by Pérez-Pereira, who in a longitudinal study of low-risk preterm children found no consistent differences in the prevalence of language delay by gestational age group(31). These differences in results indicate that the relationship between perinatal characteristics and language development is not uniform across populations.

Differences in study design are one possible explanation for this heterogeneity. Cross-sectional studies only describe the relationship between perinatal factors and language ability at a single point in time, and therefore cannot describe the trajectory of language development or distinguish transient delays from persistent impairments. In contrast, longitudinal studies can evaluate changes in language ability over time and identify predictive factors at different stages of development. In a longitudinal cohort, language ability at ages 2 and 4 was found to have a stronger predictive contribution to language ability at age 7 than most early life factors(32).

The age of the participants may also influence the magnitude of the observed associations. Language skills develop rapidly during the toddler and preschool years, so the manifestation of delays may vary by age at assessment. At younger ages, some children with biological vulnerabilities may still fall within a wide range of developmental variation, whereas at later ages differences in vocabulary, receptive language, expressive language, and complex language skills may become more pronounced(31),(32). Thus, studies using different age groups do not always produce the same estimates of associations.

In addition to biological characteristics and age, postnatal environmental stimulation is an important factor that may influence language development. Parental education, socioeconomic status, the quality of parent-child interactions, cognitive stimulation, and learning opportunities at home can act as both mediators and protective factors in language development(33). Therefore, children with a history of low birth weight or prematurity who grow up in an environment with adequate communication stimulation may show better language development than children with similar perinatal conditions but who receive more limited stimulation. These differences in environmental context have the potential to weaken or strengthen the relationship between perinatal factors and language delay.

Thus, conflicting findings in the literature cannot be explained solely by differences in study outcomes. This heterogeneity is likely a consequence of differences in study design, participant age at evaluation, clinical characteristics of the populations, and the quality of postnatal environmental stimulation. In this study, the loss of significance of the birthweight-birthweight association after adjustment for gestational age may indicate that some of the associations in the bivariate analysis reflect the influence of gestational age or other perinatal factors closely related to birthweight. Therefore, future studies should use longitudinal designs and consider gestational age, fetal growth, and the language

stimulation environment simultaneously to obtain a more accurate estimate of the independent contribution of birthweight to speech delay(28),(33)

Maternal Age at Childbirth Factor

The results of this study indicate no significant association between maternal age at childbirth and the occurrence of speech delay ($p = 0.339$). The odds ratio analysis showed that mothers who gave birth at <20 years or >35 years of age had a 1.29-fold increased risk of having children with speech delay compared to mothers aged 20–35 years. However, this increased risk was not statistically significant.

These findings are consistent with several previous studies suggesting that maternal age is not a direct causal factor of language delay, but rather serves as a proxy indicator for other risk conditions. Younger mothers (<20 years) may face challenges related to limited parenting experience, psychological readiness, or family support, which can influence early stimulation patterns. Conversely, advanced maternal age (>35 years) is associated with increased pregnancy-related risks such as hypertension, gestational diabetes, and delivery complications; however, not all of these conditions necessarily affect language development.

The absence of a significant association in this study may be explained by the influence of confounding factors, including maternal education level, home stimulation, and the quality of mother–child interaction, which may play a more dominant role in determining speech and language outcomes. This suggests that maternal age alone does not directly determine speech delay; instead, environmental factors, family communication patterns, and caregiving quality are likely to be more influential.

Although no statistically significant relationship was observed, maternal age remains theoretically relevant. Young maternal age (<20 years) is frequently linked to socioeconomic risk factors, such as lower educational attainment, limited knowledge of early childhood stimulation, and psychological instability, all of which may negatively affect parenting quality. When these factors are well controlled or not predominant in the study population, the independent effect of maternal age may fail to emerge as a significant variable.

Similarly, advanced maternal age (>35 years) is associated with an increased risk of pregnancy complications, including gestational diabetes, hypertension, and chromosomal abnormalities. Nevertheless, current evidence indicates that the quality of mother–child interaction, parental responsiveness, and the level of verbal stimulation in the home environment exert a far greater influence on language development than maternal age as a biological factor. It is also important to consider that the lack of significance in this study may be influenced by sample composition, relative homogeneity in socioeconomic characteristics, and the presence of additional confounding variables such as maternal education, home environment quality, child screen time duration, and early bonding experiences.

These findings are supported by a systematic review and meta-analysis by Novianti, which analyzed 63 cohort studies and 12 case-control studies and concluded that advanced maternal age (AMA; >35 years) significantly increases the risk of adverse pregnancy outcomes. Although the primary

focus of the review was stillbirth (OR = 1.75; 95% CI: 1.62–1.89), it also identified increased risks of conditions that may indirectly affect neurodevelopment and language outcomes, including fetal growth restriction (FGR), preterm birth (PTB), preeclampsia (PE), and neonatal intensive care unit (NICU) admission(34).

Maternal age at childbirth represents a fundamental biological factor that influences the risk of specific fetal pathologies, which may ultimately contribute to speech delay. This is supported by a population-based case-control study conducted by Luo et al. (2023) in China, which examined the association between maternal age and isolated birth defects. The study reported that advanced maternal age (>35 years) was significantly associated with an increased risk of cleft lip and/or palate (CL/P), while younger maternal age was associated with a higher risk of polydactyly.(35) Although these findings focus on congenital malformations, the increased risk of fetal pathology associated with maternal age logically aligns with a higher likelihood of perinatal complications, which may subsequently increase the risk of neurological impairment and developmental deficits, including speech delay.

However, the link between congenital anomalies and speech delay must be interpreted cautiously through specific mechanistic pathways to avoid overinterpretation. Structural anomalies such as cleft lip and/or palate (CL/P) impact language development primarily through direct anatomical and mechanical impediments to the articulatory apparatus, compromising intraoral pressure generation and phoneme precision. Furthermore, major orofacial anomalies are frequently accompanied by secondary perinatal complications—such as early feeding difficulties, recurrent otitis media leading to fluctuating conductive hearing loss, and psychosocial stress on early caregiving—which indirectly hinder language acquisition. Conversely, for isolated minor anomalies that do not involve the orofacial structure or the central nervous system (such as polydactyly associated with younger maternal age), there is no direct neurobiological or articulatory pathway leading to speech delay. Therefore, findings regarding congenital anomalies in maternal age studies should not be generalized as direct predictors of speech delay across all malformation types; rather, they serve as indicators of heightened fetal vulnerability and perinatal complexity that may indirectly raise the overall risk of neurodevelopmental deficits.

CONCLUSION AND RECOMMENDATION

This study demonstrates that specific birth history factors—specifically non-optimal gestational age (<37 or >40 weeks), cesarean delivery, and non-optimal birth weight (<2,500 or >3,500 grams)—are significantly associated with an increased risk of speech delay in children aged 2–6 years, whereas maternal age at delivery showed no statistically significant association. These findings highlight the importance of targeted early developmental screening and routine monitoring for children with high-risk birth histories alongside early language stimulation from parents and healthcare professionals; furthermore, future prospective cohort studies are recommended to confirm these temporal relationships

and clarify the complex interactions between biological vulnerabilities and postnatal environmental factors.

ACKNOWLEDGMENTS

The authors express their gratitude to Universitas Harkat Negeri for providing the financial support that facilitated the completion of this research. Special thanks are also extended to the parents of toddlers who were willing to participate in this study, as well as speech therapists and health workers who have been dedicated and whose valuable assistance has contributed significantly to the implementation and success of this study. The collaborative efforts of Harkat Negeri University and the commitment of health workers have played a key role in accelerating the progress of this study. Their support has been crucial in enabling the investigation of key aspects related to speech delays in children.

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