

Exploring Speech Delay in a Premature Five-Year-Old Child: An Integrative Psycholinguistic Case Analysis

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Abstract

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Premature birth is a well-established biological risk factor for neurodevelopmental delay; however, limited research integrates neurological vulnerability with psycholinguistic processing mechanisms at preschool age. This study explores how prematurity contributes to speech delay in a five-year-old child through an integrative psycholinguistic framework. Using a qualitative instrumental case study design, data were collected through semi-structured parental interviews, clinical observation records, and speech-language therapy documentation. Thematic analysis revealed five dimensions of psycholinguistic impairment: lexical limitation, delayed lexical retrieval, phonological encoding weakness, sentence formulation deficit, and environmental reinforcement gap. Findings indicate that disrupted white matter development and cognitive processing inefficiencies manifest as restricted vocabulary, delayed lexical retrieval, reduced sentence complexity, and minimal communicative responsiveness. Importantly, environmental inconsistency in parental language stimulation intensifies these deficits, suggesting a dual-risk interaction between biological and environmental factors. This study proposes a conceptual framework linking prematurity, psycholinguistic processing limitations, and observable speech delay behaviours. The findings extend existing literature by providing a theoretically integrated explanation of language delay at a critical developmental milestone—age five—when school readiness becomes linguistically demanding in the Indonesian educational context. Implications emphasize early detection, structured intervention, and consistent caregiver-mediated language enrichment.

Kata Kunci:

*prematuritas,
keterlambatan bicara,
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Abstrak

Kelahiran prematur merupakan faktor risiko biologis yang telah terbukti berkontribusi pada keterlambatan neurodevelopmental; namun, penelitian yang mengintegrasikan kerentanan neurologis dengan mekanisme pemrosesan psikolinguistik pada usia prasekolah masih sangat terbatas. Penelitian ini mengeksplorasi bagaimana prematuritas berkontribusi terhadap keterlambatan bicara pada seorang anak berusia lima tahun melalui kerangka psikolinguistik integratif. Menggunakan desain studi kasus instrumental kualitatif, data dikumpulkan melalui wawancara semi-terstruktur dengan orang tua, catatan observasi klinis, dan dokumentasi terapi wicara-bahasa. Analisis tematik mengungkap lima dimensi gangguan psikolinguistik: keterbatasan leksikal, keterlambatan pemulihan leksikal, kelemahan pengkodean fonologis, defisit formulasi kalimat, dan kesenjangan penguatan lingkungan. Temuan menunjukkan bahwa gangguan perkembangan materi putih dan inefisiensi pemrosesan kognitif bermanifestasi sebagai kosakata terbatas, keterlambatan pengambilan kata, kompleksitas kalimat yang rendah, dan respons komunikatif yang minimal. Penting untuk dicatat bahwa inkonsistensi lingkungan dalam stimulasi bahasa orang tua memperburuk defisit ini, mengindikasikan interaksi risiko ganda antara faktor biologis dan lingkungan. Implikasi penelitian ini menekankan pentingnya deteksi dini, intervensi terstruktur, dan pengayaan bahasa yang konsisten oleh pengasuh.

INTRODUCTION

Premature birth remains a global health concern, with increasing survival rates attributable to advances in neonatal care. However, improved survival has been accompanied by heightened neurodevelopmental vulnerability, particularly in language development. While extensive quantitative research confirms that preterm children are at increased risk for expressive and receptive language delays (van Noort-van der Spek et al., 2012), less attention has been paid to the underlying psycholinguistic mechanisms that translate neurological immaturity into observable speech delay behaviours. Understanding these mechanisms is essential not only for

researchers but also for speech-language practitioners, early childhood educators, and parents who must navigate intervention decisions based on what they see and hear in the child—not what neuroimaging reveals.

Language production requires efficient integration of conceptual planning, lexical selection, phonological encoding, working memory storage, and articulatory execution. Premature birth disrupts late-gestational cortical maturation, particularly white matter connectivity that is critical for language networks (Dubner et al., 2020). Yet, the field lacks integrative models connecting these biological disruptions to cognitive-linguistic processing deficits at preschool age.

In the Indonesian educational context, age five marks a critical transition into Taman Kanak-Kanak Besar (TK B) and the preparatory stage for Sekolah Dasar (SD). At this juncture, school readiness is heavily dependent on verbal communication competence: children are expected to follow multi-step oral instructions, engage in narrative recounting, and participate in structured classroom dialogue. A child with unresolved speech delay at age five thus faces compounded risks—both linguistic and social-academic. A qualitative case study approach was selected because it permits the depth of individual analysis necessary to capture how biological vulnerability and environmental factors interact in ways that aggregate quantitative data obscure.

This study aims to: (1) explore speech delay in a five-year-old premature child through psycholinguistic theory; (2) integrate neurological evidence with cognitive processing models; and (3) propose a conceptual framework linking biological and environmental factors.

Prematurity as Neurobiological Risk

Meta-analytic evidence confirms persistent language deficits among preterm children (van Noort-van der Spek et al., 2012). Preterm-born children score significantly lower on both simple and complex language function tests compared with term-born peers, and group differences in complex language function increase significantly from ages three to twelve years. Longitudinal studies further show that expressive language gaps widen under increasing linguistic demand (Coughlan et

al., 2023), suggesting that cognitive-linguistic vulnerabilities become more pronounced as educational demands grow.

Neuroimaging research demonstrates reduced white matter integrity in the arcuate fasciculus and associated language pathways (Salvan et al., 2017; Dubner et al., 2020). Salvan et al. (2017) found that term-equivalent fractional anisotropy in the arcuate fasciculi of preterm neonates was significantly associated with individual differences in linguistic and cognitive abilities in early childhood, independent of the degree of prematurity. These structural differences correlate with slower language processing and reduced verbal fluency. However, these studies remain largely correlational and neurologically focused, leaving a gap in explanatory models that bridge neurobiology and observable speech behaviours.

Psycholinguistic Processing Models

2.2.1 Levelt's (1989) Speech Production Model

Levelt (1989) conceptualizes speech production as a sequential three-stage process: the Conceptualizer (pre-verbal message formation), the Formulator (lexical retrieval and phonological encoding), and the Articulator (motor speech output). Speech delay in premature children is hypothesized to arise primarily at the formulation stage, where lexical retrieval and phonological encoding occur. Neurological immaturity may reduce efficiency in lexical access and syntactic structuring, resulting in short utterances, sound substitutions, and delayed responses. This framework provides a tractable theoretical lens for interpreting the observable delays described in the present case.

2.2.2 Baddeley's (2000) Working Memory Model

Working memory plays a central role in language acquisition. Baddeley (2000) identifies the phonological loop as the component responsible for the temporary storage and rehearsal of verbal information, while the central executive regulates attentional allocation across cognitive tasks. In language learning, the phonological loop supports retention of new word forms long enough for them to be associated with meaning, while the central executive facilitates hierarchical syntactic planning.

Loe et al. (2014) demonstrated that executive function mediates the relationship between gestational age and functional outcomes in preschoolers, with preterm children showing measurable deficits on both parent-rated and performance-based executive function instruments. Reduced phonological loop capacity limits vocabulary expansion and sentence complexity. Thus, speech delay may reflect underlying cognitive storage constraints rather than purely articulatory problems—a distinction with significant implications for intervention design.

Environmental Moderation

Although biological risk is significant, the quality and quantity of parental language input has been shown to moderate outcomes in preterm children. Adams-Chapman et al. (2018) demonstrated that caregiver talk and medical risk together predict language outcomes, indicating that post-NICU environmental factors play a substantial role in shaping developmental trajectories. Early developmental intervention programmes further enhance expressive language development: the Cochrane systematic review by Spittle et al. (2015) found that early post-discharge intervention produces moderate benefits for cognitive and behavioural outcomes up to preschool age. However, few studies integrate biological vulnerability and inconsistent home reinforcement within a unified explanatory model—a gap the present study addresses.

Despite strong evidence linking prematurity to language delay, three gaps remain in the existing literature. First, there is a lack of integration between neurobiology and psycholinguistic theory: existing studies tend to operate either at the neuroimaging level or at the behavioural level, without constructing a unified explanatory model. Second, there is limited focus on age five as a specific linguistic transition milestone, despite its documented importance for school readiness—particularly in the Indonesian context where TK B and SD entry create concrete, observable communicative demands. Third, there is insufficient qualitative analysis of the biological–environmental interaction at the individual case level. This study addresses these gaps by proposing an integrative psycholinguistic framework grounded in case-based analysis.

METHOD

This study employed a qualitative instrumental case study design (Stake, 1995). An instrumental case study is one in which a specific case is examined primarily to provide insight into a broader issue or to refine a theoretical understanding—in this instance, the psycholinguistic mechanisms through which prematurity produces speech delay. This design was chosen because it enables theoretically-driven, in-depth interpretation of a complex biological-environmental interaction that aggregate quantitative methods cannot adequately capture at the individual level. The case itself serves as a vehicle for illuminating larger questions about how neurological vulnerability is expressed through observable communicative behaviour. Credibility of interpretations was strengthened through methodological triangulation across three distinct data sources.

The participant was a five-year-old boy (referred to as Arka, a pseudonym) born prematurely at 32 weeks of gestation, with a birth weight of 1,650 grams. He had been enrolled in speech-language therapy since the age of two years, attending weekly sessions at a private developmental clinic in Pekanbaru, Riau, Indonesia. At the time of data collection, Arka was preparing to enter TK B. Informed consent was obtained from his guardians in accordance with institutional ethical guidelines. All identifying information has been anonymized.

Three primary data sources were used: (1) semi-structured parental interviews (two sessions, approximately 45 minutes each) exploring Arka's developmental history, home language environment, and daily communicative routines; (2) structured clinical observation records from four therapy sessions (each 45 minutes), documenting communicative behaviours, response latency, utterance complexity, and phonological accuracy; and (3) speech-language therapy documentation, including cumulative progress notes, therapist assessment summaries, and session transcripts compiled over six months of intervention.

Data were analysed using the six-phase inductive thematic analysis procedure described by Braun and Clarke (2006): (1) familiarization with the data through repeated reading and annotation; (2) generation of initial codes from across all three data sources; (3) searching for themes by collating related codes; (4)

reviewing and refining candidate themes against the dataset; (5) defining and naming final themes; and (6) producing the written analysis. A total of 47 initial codes were generated, which were progressively consolidated into 12 candidate themes, and finally refined into 5 overarching themes. Each theme was then interpreted through the dual theoretical lens of Levelt's (1989) Speech Production Model and Baddeley's (2000) Working Memory Model to connect empirical observations to established psycholinguistic theory.

FINDINGS AND DISCUSSION

Findings

Thematic analysis of the combined data sources yielded five dominant themes, each corresponding to a dimension of psycholinguistic processing. Representative vignettes drawn from observational records and parental interview transcripts are presented to substantiate each theme.

5.1 Lexical Limitation

Arka's productive vocabulary was restricted to a small set of high-frequency words used repeatedly across communicative contexts. Novel lexical items were rarely attempted, and referential flexibility was minimal. Across four observed therapy sessions, Arka used fewer than 30 distinct word types to communicate across a wide range of picture-naming, play-based, and conversational tasks.

Observation Record, Session 2:

"When presented with a set of ten picture cards (animals, food, household objects), Arka successfully named only 'cat', 'rice', and 'car'. For the remaining seven items—including 'umbrella', 'flower', and 'spoon'—he either pointed silently, produced an unintelligible vocalization, or looked away. No spontaneous labelling attempts were observed for unfamiliar referents."

This pattern is consistent with restricted phonological loop capacity limiting the encoding and consolidation of new word forms into long-term lexical representations (Baddeley, 2000).

5.2 Delayed Lexical Retrieval

Observations revealed consistently slow response latency when Arka was addressed verbally. Prolonged pausing before attempted responses, combined with visible effortful word-searching behaviours, suggested inefficiency at the

formulation stage of Levelt's (1989) model—specifically in lexical selection from the mental lexicon.

Parental Interview, Session 1 (Mother):

"Kalau saya tanya 'mau makan apa?', dia diam dulu lama, bisa sampai 10 detik, baru bilang 'nasi'. Kadang saya pikir dia tidak dengar, tapi ternyata dia sedang mikir." [Translation: "When I ask 'what do you want to eat?', he stays quiet for a long time—up to 10 seconds—before saying 'rice'. Sometimes I think he did not hear me, but actually he is thinking."]

Observation Record, Session 3:

"During a structured naming task, mean response latency for Arka was 8.4 seconds, compared with a clinical reference range of 1–2 seconds for typically developing five-year-olds. On three occasions, the latency exceeded 15 seconds, after which Arka abandoned the response entirely."

5.3 Phonological Encoding Weakness

Speech samples showed incomplete articulation of target words, frequent sound substitutions, and phoneme omissions in consonant clusters. These features indicate difficulty at the phonological encoding substage, consistent with reduced white matter integrity in the arcuate fasciculus, which connects temporal speech-perception regions with frontal motor-speech areas (Salvan et al., 2017).

Therapy Documentation, Progress Note (Month 4):

"Arka consistently substitutes /r/ → /l/ and /s/ → /t/ in word-initial positions (e.g., 'loti' for 'roti' [bread]; 'tatu' for 'satu' [one]). Consonant clusters in word-final position are reduced to single consonants in approximately 80% of attempts. Articulation errors are phonologically systematic rather than random, suggesting encoding-level impairment rather than purely motor-articulatory difficulty."

5.4 Sentence Formulation Deficit

Multi-word combinations were rare. Most spontaneous utterances consisted of one or two words, with grammatical morphemes (articles, verb inflections, prepositions) systematically absent. This reduced syntactic complexity suggests impairment in central executive functioning required for hierarchical sentence planning (Levelt, 1989).

Observation Record, Session 4 (Play-based interaction):

"During a 20-minute free play session involving toy animals and blocks, Arka produced 34 communicative turns. Of these, 26 (76%) were single-word utterances (e.g., 'kucing' [cat], 'jatuh' [fall]). Six turns consisted of two-word combinations (e.g., 'kucing sini' [cat here]). No utterance exceeded two words in length. No verb-argument structures, prepositions, or grammatical inflections were observed."

Parental Interview, Session 2 (Father):

"Dia tidak pernah bilang kalimat panjang. Paling banter dua kata. Waktu minta minum, dia bilang 'mau susu', bukan 'Arka mau minum susu' seperti anak-anak lain seusianya." [Translation: "He never says a long sentence. Two words at most. When he wants a drink, he says 'want milk', not 'Arka wants to drink milk' like other children his age."]

5.5 Environmental Reinforcement Gap

Parental interviews and home-environment observations revealed inconsistent engagement in language-stimulating activities. Parents reported limited structured reading routines, infrequent conversational elaboration, and reduced responsiveness to Arka's communicative attempts. This environmental profile aligned with the dual-risk model: biological vulnerability was compounded by insufficient environmental scaffolding (Adams-Chapman et al., 2018).

Parental Interview, Session 1 (Mother):

"Jujur, kami tidak sering baca buku sama dia. Kami sibuk kerja, dan kalau dia diam saja, kami pikir tidak apa-apa. Baru belakangan ini kami tahu seharusnya kami lebih banyak bicara sama dia dari kecil." [Translation: "Honestly, we do not often read books with him. We are busy with work, and when he stays quiet, we think it is fine. Only recently did we learn that we should have talked with him more from an early age."]

Therapy Documentation, Therapist Note (Month 6):

"Home practice compliance remains low: parents report completing structured language activities on average 1–2 days per week, against the recommended daily frequency. Conversational turn-taking at home is described as minimal. Television and tablet use exceeds 3 hours per day, with no interactive verbal engagement reported during screen time."

Discussion

The findings converge to suggest that prematurity contributes to speech delay through a multi-layered cascade of disruptions: neurological immaturity affects white matter connectivity; this produces working memory constraints that

reduce phonological storage; executive dysfunction limits sentence planning; and auditory processing inefficiency delays lexical access—resulting in the observable speech delay documented across the five themes.

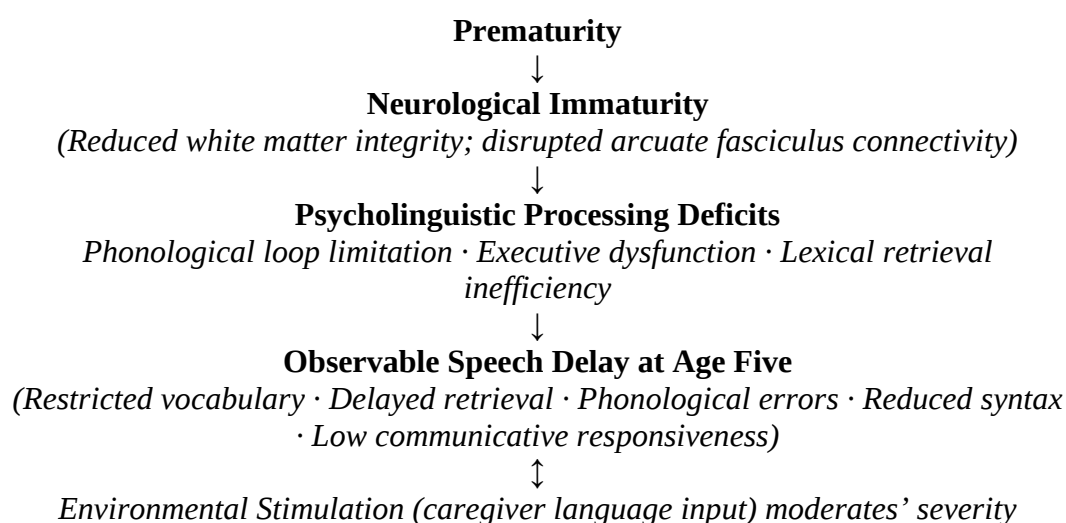
This cascade aligns theoretically with Levelt's (1989) formulation stage vulnerability: if lexical retrieval and phonological encoding are bottlenecks, output will be characterized by precisely the features observed—sparse vocabulary, long response latencies, phonological errors, and syntactically reduced utterances. The vignette evidence from Sessions 2 and 4 directly corroborates this: Arka's mean retrieval latency of 8.4 seconds and 76% single-word utterance rate are consistent with a formulator operating under capacity constraints. Similarly, Baddeley's (2000) phonological loop limitations explain why productive vocabulary remains constrained: insufficient rehearsal capacity means new word forms do not consolidate into long-term representations, as evidenced by Arka's failure to label seven of ten common pictured objects.

Crucially, the environmental reinforcement gap (Theme 5) exacerbates biological risk, supporting a dual-risk model consistent with Adams-Chapman et al. (2018). Parental language input quantity and quality are established moderators of language outcomes even in neurologically vulnerable populations, and their absence represents a tractable point of intervention. The finding that home practice compliance was only 1–2 days per week against a daily recommendation, combined with over three hours of passive screen time, suggests that intervention gains made in the clinical setting were not being transferred and consolidated in the home environment. Thus, speech delay in this case is not merely a medical outcome of preterm birth, but a cognitive-linguistic processing consequence shaped by the interaction of neurobiological vulnerability and environmental mediation.

The present findings extend existing literature in three ways. First, they provide a theoretically integrated explanation connecting neuroimaging evidence (white matter disruption) with psycholinguistic processing theory and observable behaviour—a synthesis largely absent from prior research. Second, they focus attention on age five as a critical juncture where cumulative deficits become educationally consequential (Twilhaar et al., 2018; van Noort-van der Spek et al.,

2012)—particularly salient in the Indonesian context where TK B marks the formal beginning of school-readiness assessment. Third, the instrumental case study design, supported by vignette evidence, illuminates the texture of individual experience in ways that complement large-scale quantitative research.

Based on the findings, the following integrative framework is proposed (Figure 1):



CONCLUSION

This study demonstrates that prematurity significantly influences speech delay through interconnected neurological and psycholinguistic processing pathways. Disrupted white matter development constrains phonological loop capacity and executive functioning, which in turn impairs lexical retrieval, phonological encoding, and syntactic formulation—the key substages identified in Levelt's (1989) Speech Production Model. Environmental stimulation, however, remains a critical moderating factor, and its absence compounds biological risk in a dual-risk interaction. Early detection of psycholinguistic processing deficits, combined with integrated cognitive-linguistic intervention and caregiver-mediated language enrichment, is essential for optimizing developmental outcomes for preterm children at school entry.

Several limitations of this study warrant acknowledgement. First, the use of a single case restricts generalizability: while the instrumental design is analytically valuable, conclusions cannot be extended to the broader preterm population without

further investigation. Second, no standardized cognitive assessment was administered within the scope of this study, limiting psychometric precision in characterizing the child's cognitive profile. Third, neurobiological mechanisms (white matter disruption, phonological loop capacity) were inferred from the literature rather than directly measured in this participant, meaning the causal chain remains inferential. Fourth, the study relies on parental self-report for aspects of the home language environment, which may be subject to social desirability bias. Fifth, the illustrative vignettes, while grounded in clinical and observational data, are presented as representative extracts and should be interpreted accordingly.

Future research should pursue longitudinal neurocognitive tracking of preterm children from birth through school entry to map the developmental trajectory of the psycholinguistic deficits identified here. Controlled comparisons between gestational age groups with standardized language and executive function instruments would strengthen causal inference. Experimental parent-mediated language intervention studies are needed to quantify the buffering effect of enriched home language environments on biologically-at-risk children—particularly in under-resourced Indonesian contexts where clinical access is limited. Finally, direct measurement of phonological loop capacity using digit span or nonword repetition tasks would permit empirical testing of the working memory pathway proposed in the conceptual framework.

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