

# Developmental Trajectories of Early Higher-Order Thinking Talk Differ for Typically Developing Children and Children With Unilateral Brain Injuries

Rebecca R. Frausel<sup>1</sup> , Elayne Vollman<sup>1</sup>, Antonia Muzard<sup>2</sup>, Lindsey E. Richland<sup>3</sup> , Susan Goldin-Meadow<sup>1</sup>, and Susan C. Levine<sup>1</sup>

**ABSTRACT**—The use of higher-order thinking talk (HOTT), where speakers identify relations between representations (e.g., comparison, causality, abstraction) is examined in the spontaneous language produced by 64 typically developing (TD) and 46 brain-injured children, observed from 14–58 months at home. HOTT is less frequent in lower-income children and children with brain injuries, but effects differed depending on HOTT complexity and type of brain injury. Controlling for income, children with larger and later-occurring cerebrovascular infarcts produce fewer *surface* (where relations are more perceptual) and *structure* (where relations are more abstract) HOTT utterances than TD children. In contrast, children with smaller and earlier occurring periventricular lesions produce HOTT at comparable rates to TD children. This suggests that examining HOTT development may be an important tool for understanding the impacts of brain injury in children. Theoretically, these data reveal that both neurological (size and timing of brain injury) and environmental (family income) factors contribute to these skills.

As children develop language, they also develop reasoning skills, particularly their ability to use language to reason about and express relationships. Take, for example, a parent who asks, “What happens in a tornado?” while playing with her three-year-old child, who responds, “A tornado is like a mean monster!” The child’s manipulation of ideas in language—drawing comparisons, making inferences, abstracting beyond the current environment, and recognizing relationships between representations—is defined here as *higher-order thinking talk* (HOTT). This language reflects the cognitive processes through which reasoners manipulate information and rearrange or extend knowledge in novel ways (Bruner, 1973; Frausel et al., 2020; Lewis & Smith, 1993). The ability to use higher-order thinking is thought to be critical for later success in many areas, including school, the job market, and creative innovation (Koenig, 2015; Lawrence & Snow, 2010; Schleppegrell, 2004).

However, not all children acquire higher-order thinking skills, or the ability to use them in language using relational talk, along the same trajectory. There is marked variability among typically developing (TD) children in the acquisition of these skills (Richland & Burchinal, 2013), as well as differences among children with specific language impairments (Leroy, Parris, & Maillart, 2012). Here, we focus on HOTT in a group of children with prenatal or perinatal unilateral brain injury (BI). Prior research has shown that these children can have deficits in cognitive functions, such as attention, complex linguistic skills, and reasoning (Avecilla-Ramírez et al., 2011;

<sup>1</sup>Department of Comparative Human Development, The University of Chicago

<sup>2</sup>Escuela de Psicología, Pontificia Universidad Católica de Chile

<sup>3</sup>School of Education, University of California, Irvine

Correspondence concerning this article should be addressed to Rebecca R. Frausel, who is now at the Susan Hirt Hagen Center for Community Outreach, Research, and Evaluation (CORE), Penn State Erie, The Behrend College, 4909 Jordan Road, Erie, PA 16563; e-mail: rrf5129@psu.edu

Demir, Levine, & Goldin-Meadow, 2010; Demir, Rowe, Heller, Goldin-Meadow, & Levine, 2015; Gutiérrez-Hernández, Harmony, & Carlier, 2018; Moreno-Aguirre, Santiago-Rodríguez, Harmony, Fernández-Bouzas, & Porras-Kattz, 2010; Reilly, Bates, & Marchman, 1998; Reilly, Wasserman, & Appelbaum, 2013).

In this paper, we ask whether the early developmental trajectories of HOTT differ between TD children and children with early BI. Additionally, we examine other factors, such as family income (an index of environmental variation) and characteristics of the BI (an index of biological variation), that have the potential to affect the developmental trajectories of HOTT (Demir et al., 2015; Rowe, Levine, Fisher, & Goldin-Meadow, 2009). This work has the potential to provide unique insights into how biological and environmental factors predict the growth of reasoning and higher-order thinking skills, and to provide information about the plasticity of the brain's response to an early injury.

### Defining HOTT

We conceptualize HOTT as the cognitive ability to linguistically construct an explicit reference to inference, comparison, abstraction, and hierarchy (Frausel et al., 2020). Although prelinguistic children can engage in relational reasoning (e.g., causal reasoning; Wang & Baillargeon, 2008), our focus in this paper is on children's ability to express relational reasoning in language. We focus on relational language expressed through HOTT because we take the viewpoint that language provides children with strategies and concepts to support their burgeoning reasoning skills (Gentner & Goldin-Meadow, 2003).

In naturalistic settings, spontaneous HOTT provides an important window onto how children engage in complex reasoning through everyday talk. In the conversation mentioned earlier, the child compares two representations when she says a "tornado" is like a "mean monster," where the abstract nature and impact of the storm is expressed in the new representation of a monster. This example is a spontaneous version of the relational reasoning often studied in more controlled psychological contexts (Gentner, 1983; Holyoak, Thagard, & Sutherland, 1995; Richland, Morrison, & Holyoak, 2006).

Research suggests that acquiring terms such as "like" enables children to make different kinds of relational statements, such as comparisons. Özçalışkan, Goldin-Meadow, Gentner, and Mylander (2009) examined the spontaneous comparisons made by 1- to 3-year-old TD children and 2- to 4-year-old deaf children being raised without access to a usable language model. To communicate, the deaf children used gesture systems called *homesigns* with their hearing family members (Feldman, Goldin-Meadow,

& Gleitman, 1978; Goldin-Meadow, 2005). Özçalışkan et al. (2009) found that the homesigners expressed relations in their gestures that highlighted similarities (e.g., point at cat + point at tiger), but their comparisons tended to be more global, involving objects from the same category that share multiple features. While TD children also produced these broad comparisons, they also produced more focused comparisons between objects from different categories that revolved around a single feature (e.g., pointing out the similarity in shape between a lollipop and a balloon). However, TD children only produced these focused comparisons after they acquired the word "like," around 30 months. The homesigners, who lacked an explicit word for similarity such as "like," did not produce specific comparisons during the ages examined. This suggests that language has the unique feature of supporting specific higher-order representations and may be instrumental for the development of sophisticated higher-order thinking skills. As such, we view children's spontaneous use of relational language through HOTT as a window onto their higher-order thinking skills.

While the cognitive mechanisms of spontaneous HOTT cannot be as clearly defined as in an experimental paradigm, there are distinctions in the overall level of similarity and structural alignment that can be identified in each HOTT utterance. These distinctions provide a means for examining the foundations of HOTT and its growth over time. In TD children, prior research (Frausel et al., 2020) has defined two separate trajectories of children's use of HOTT: *structure* HOTT and *surface* HOTT. *Structure* HOTT requires structural alignment—as in the "tornado: mean monster" example—drawing a complex, distal comparison. *Surface* HOTT, in contrast, displays more surface-level similarity (Frausel et al., 2020; Richland & Simms, 2015). For example, the child could have said, "A tornado is like a dark cloud" (a surface HOTT comparison). This utterance also displays the linguistic construction of a comparison, which links two representations and involves higher-order thinking. For a young child, even this connection is quite sophisticated. However, the higher-order thinking exemplified in the "tornado:dark cloud" example is less of a mental leap, and relies more on perceptual information, than the structure HOTT comparison exemplified by the "tornado:mean monster" example.

In TD children, we find that surface HOTT emerges earlier, and is more frequent, than structure HOTT, so is likely a first indicator of children's developing skills at integrating reasoning and language. However, in TD children, structure HOTT is a better predictor than surface HOTT of children's later reasoning skills (Frausel et al., 2020). Thus, both surface and structure HOTT provide insights into how children's reasoning develops in everyday spontaneous interactions.

### Language Development in Children With Brain Injuries

Children with prenatal or perinatal brain injuries show remarkable plasticity for language compared to adults with comparable injuries. This is related to the fact that the developing nervous system is reliant on input to construct its processing networks (Bates et al., 2001; Bates & Roe, 2001; Stiles, Reilly, Paul, & Moses, 2005). When lesions affect classic language areas, this input sensitivity enables the language functions of children with early lesions to utilize alternative neural pathways to support language abilities that are similar in terms of the level of functioning as the language abilities of TD children (Bates & Dick, 2002; Feldman, 2005). However, children with BI often display delays in their language development (Bates et al., 1997; Feldman, Holland, Kemp, & Janosky, 1992; Thal et al., 1991), as well as more marked differences for particularly challenging language skills, such as narrative and complex sentence structure (Demir et al., 2010, 2015; Reilly et al., 1998; Reilly, Losh, Bellugi, & Wulfeck, 2004). It is unknown whether these same delays and deficits extend to the intersection of language and complex cognition as expressed through higher-order thinking talk. However, previous work (Frausel et al., 2020) suggests that complex language is *not* a prerequisite for complex reasoning, leaving open the possibility that deficits in BI children's language abilities might not necessarily carry over to deficits in HOTT.

### Complexity of Reasoning

Children begin to use language to express HOTT around the second year of life (Frausel et al., 2020), but their relational reasoning skills are still developing at this point. They tend to more easily, or preferentially, draw correspondences across representations that derive from the appearance or object-level similarities (Holyoak, Junn, & Billman, 1984; Richland, Zur, & Holyoak, 2007). However, children can reason more deeply when the relational correspondences are not competing with featural matches (Richland et al., 2006) and when they have strong prior knowledge in the domain (Brown, 1989; Gentner & Rattermann, 1991; Goswami & Brown, 1990). Importantly, the spontaneous HOTT identified in TD children reflects the same developmental trajectory found in experimental studies. At first, young children almost exclusively produced *surface* HOTT (as in the "tornado:dark cloud" example), and only with age did they increasingly produce *structure* HOTT (as in the "tornado:mean monster" example; Frausel et al., 2020).

We predict that the different developmental trajectories for surface and structure HOTT will be similar for children with BI. However, we also predicted that there may be delays in the emergence of HOTT given the cognitive and linguistic demands of linking multiple representations (Richland et al., 2007). One possibility is that BI leads to delays in the

emergence and growth of *both* surface and structure HOTT. This pattern would indicate that HOTT is broadly impacted by early BI. Alternatively, an early BI could differentially impact the development of surface versus structure HOTT. In particular, early BI may be more detrimental to structure than surface HOTT. This would parallel the pattern found for other aspects of language, where deficits are more marked for relatively complex than relatively simple aspects of language (Demir et al., 2015; Reilly et al., 1998; Rowe et al., 2009).

### Differences by Income and Lesion Characteristics

In addition to comparing TD and BI children's trajectories of surface and structure HOTT, we examine individual differences regarding other child-specific characteristics, including family income and type of lesion. Income-based disparities have been observed in many aspects of early language, such as vocabulary size, for both TD children (Hoff, 2003) and children with BI (Rowe et al., 2009). Previous research has also shown that TD children from higher-income families tend to use more spontaneous HOTT than children from lower-income families (Frausel et al., 2020), pointing to an environmental role in HOTT development. We examine the effects of income on TD and BI children's HOTT trajectories and test whether the effects of income are similar in both groups.

For the children with BI, we also examine how lesion type relates to HOTT. The children in our sample display one of the two types of lesions, periventricular (PV) lesions or cerebrovascular infarcts (CI). These lesions differ in size, occur at different times in gestational development, and affect different types of matter. PV lesions are smaller, usually occur in the early to mid-third trimester of pregnancy, and primarily affect white matter. CIs are larger, occur later in the third trimester, and primarily affect gray matter (Demir et al., 2015; Rowe et al., 2009; Staudt et al., 2004). In our analyses, we examine whether CI versus PV lesions differ in their relationship to HOTT development. Based on previous findings (Frausel et al., 2020), we predict that children with CI will be more delayed in HOTT, particularly structure HOTT, than children with PV lesions. Since CI lesions tend to occur later and to be larger, thus potentially allows for less neural reorganization to take place.

### The Current Study

We address three main research questions: (1) Does the growth of surface and structure HOTT differ for TD and BI children between 14 and 58 months of age? (2) Are there different developmental trajectories for surface or structural HOTT by type of BI (PV lesions vs. CI)? (3) Does family income play a similar role for BI and TD children?



To address these questions, we use a rich set of naturalistic, longitudinal data of children in their everyday home contexts to examine the nature and complexity of higher-order thinking embedded in children's spontaneous talk. The TD children (described in Frausel et al., 2020) serve as a comparison group for children with BI. All children were videotaped in their homes every 4 months between 14 and 58 months while engaging in unstructured, everyday activities, yielding over 1,500 hr of video. This period is a crucial time in development when children's language and abstract reasoning skills are emerging and growing in complexity. Our study thus assesses, for the first time, the HOTT trajectories of children with early BI using spontaneous data from naturalistic settings and explores whether and how these trajectories differ from those of TD children.

## METHODS

### Participants

Participants were 64 TD children and 46 children with brain injuries participating in a larger study of language and reasoning development (Goldin-Meadow et al., 2014). TD children were recruited via advertisements and direct mailings to targeted zip codes, and the sample was carefully constructed to mirror the demographic and socioeconomic diversity characteristic of the Chicago area (as reported in the 2000 U.S. Census). Children with BI were recruited by contacting pediatric neurologists and through parent support groups. Table 1 presents the demographics of the two samples. Although the samples are similar in parental education level and proportion of female and first/only-born children, the BI sample had significantly higher income than the TD sample (because of the difficulty in recruiting the BI sample, and the care with which the TD sample was recruited to reflect a wide range of SES backgrounds).

The brain lesions of the BI children were coded by two pediatric neurologists according to type (PV or CI), as well as laterality (left or right hemisphere) and size (small, medium, or large; size was coded based on the number of lobes and subcortical regions affected). Lesion characteristics were unavailable for two children; in the remaining 44, 39% ( $n = 17$ ) had PV lesions, and 61% ( $n = 27$ ) had CI. We focus on lesion type here, which captures variance in size and timing of the injury, rather than on lesion laterality and size per se, due to the relatively small sample size (see Table S1, for details).

### Procedure

Families were visited in their homes every 4 months between 14 and 58 months, or, for the children with BI, beginning

**Table 1**  
TD and BI Sample Demographics

	<i>TD sample</i> ( $n = 64$ ), M (SD)	<i>BI sample</i> ( $n = 46$ ), M (SD)	<i>t-test of</i> <i>difference</i>
Female	0.48 (0.50)	0.56 (0.50)	-0.83
First- or only-born	0.56 (0.50)	0.61 (0.49)	-0.48
Annual family income	\$61,000 (\$32,000)	\$83,000 (\$21,000)	-4.39***
Parent education (years)	15.66 (2.24)	15.87 (2.04)	-0.52

Note. TD = typically-developing. BI = brain-injured.

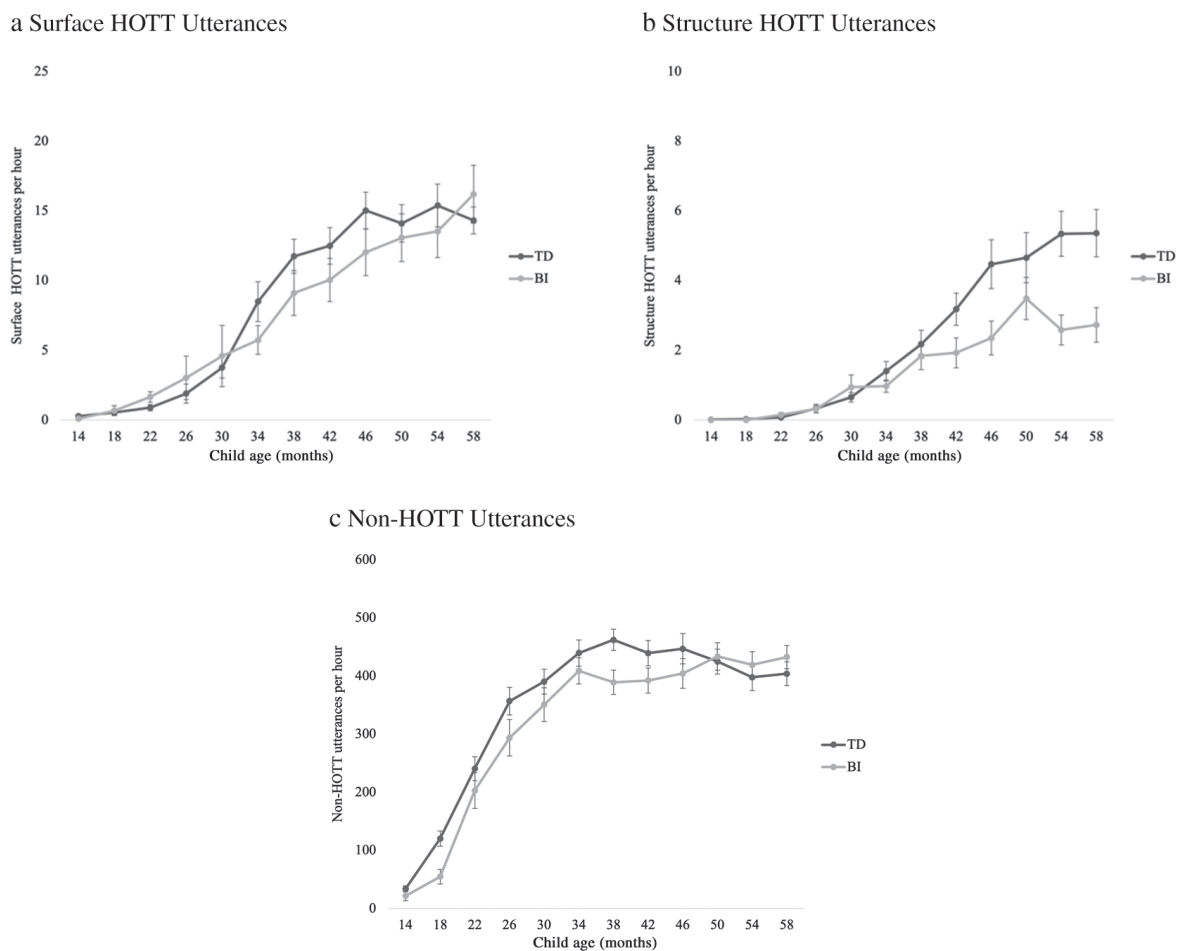
\*\*\* $p < .001$ .

at the age point at which they entered the study. This varied for the children with BI because of the rarity of this condition and the limited population of children with early BI, which necessitated relaxing the time of entry into the study. The BI children's average age of the first visit was 23.9 months ( $SD = 11.0$  months, range 14–58 months), and the BI sample had more missing visits ( $n = 128$ , 23.2%) than the TD sample ( $n = 42$ , 5.4%), due to differences in the ages at which they entered the study (see Table S2). At each visit, researchers recorded 90 min of spontaneous interactions between parents and their children as they went about their typical routines.

### Transcription and Coding of Spontaneous Talk

All parent- and child-produced speech was transcribed, including all dictionary words, onomatopoeic sounds (e.g., woof-woof), and evaluative sounds (e.g., uh-oh). Ritualized or memorized speech, such as song (e.g., singing the ABCs) or prayer (e.g., reciting the Lord's Prayer), was not transcribed. The speech was divided by trained and reliable coders into utterances (sequences of words preceded or followed by a pause, a change in intonational pattern, or a change in conversational turn; Rowe & Goldin-Meadow, 2009; Rowe, 2012).

Each utterance was coded for the presence of HOTT, talk that indexes two or more representations and constructs a relational bridge or link between them (Frausel et al., 2020). HOTT was coded when the speaker's utterance contained both representations and the link between them (e.g., "They're laughing because he fell down"); when speakers responded to HOTT-eliciting questions (e.g., a parent asks, "Why were they laughing?" and the child replies, "Because he fell down"); and when speakers ask HOTT-eliciting questions (e.g., if the child asks, "Why were they laughing?"). While HOTT was coded in both parents' and children's talk, this paper only focuses on children's talk.



**Fig 1.** Mean number of different types of utterances produced by TD and BI children. Scales differ in each figure. Error bars  $\pm 1$  SE. BI = brain-injured; HOTT = higher-order thinking talk; TD = typically-developing.

Utterances were coded as HOTT if they contained one or more of the following: *inferences* (including hypotheticals, conditionals, cause-and-effect, and speculation based on evidence), *abstractions* (including generics, word definitions, societal rules, and archetypes), *comparisons* (including similarities and differences), and *hierarchies* (including categorization, taxonomies, and identifying super- and subordinate category names). The full coding manual for HOTT is available from the first author.

Each HOTT utterance was also coded for conceptual complexity. Surface HOTT was defined as a single-level mapping where the relationship between the representations was not complex and not dependent on the structural understanding of the referents indexed (Frausel et al., 2020; Richland & Simms, 2015). This type of relational utterance was often evident in the immediate environment (i.e., not decontextualized) and often corresponded to more featural or perceptual alignments (e.g., “Those are both red”). Structure HOTT, in contrast, is defined as a complex mapping at a systemic level and requires a

deeper understanding of the relations being linked (Frausel et al., 2020; Richland & Simms, 2015). Structure HOTT is often decontextualized or abstract (e.g., “She’s sad because she misses her momma”; see also Frausel, Richland, Levine, & Goldin-Meadow, 2021). See Table S3 for examples of each HOTT type at both levels of complexity produced by TD and BI children.

Reliability analyses were performed for parent and child speech combined due to the interdependent nature of the coding. Reliability analyses for identification of utterances as surface or structure HOTT were conducted on 212 transcripts (18% of the 1,150 transcripts) coded by two or more people. The average percent agreement was 98.4% (average kappa = 0.83), indicating high agreement.

## RESULTS

The results are presented in three sections: (1) We compare the developmental trajectory of *surface* or *structure* HOTT in TD and BI children (collapsing across lesion type)

between 14 and 58 months while examining impacts of other child-specific factors such as income. (2) We compare the surface and structure HOTT use of children with the two types of lesions (CI vs. PV lesions) to TD children, again examining impacts of other child-specific factors such as income. (3) We examine the effects of lesion type *within* the BI sample.

### TD and BI Children's Use of Surface and Structure HOTT

In Figure 1, we report the mean number (a) *surface* and (b) *structure* HOTT utterances produced by children per hour over the study period, with (c) non-HOTT utterances as a baseline. This figure shows that, although TD and BI children produce similar numbers of non-HOTT utterances, TD children, on average, produce more *surface* and *structure* HOTT utterances per hour than BI children, with particularly strong disparities in *structure* HOTT use.

We used Hierarchical Linear Modeling (HLM; Raudenbush & Bryk, 2002) to characterize children's use of HOTT across development as a function of their *intercept*, *growth*, and *acceleration*. We used a two-level longitudinal HLM model (with age points at level 1 nested in individuals at level 2), with the number of child *surface* or *structure* HOTT utterances at a given age as a *Poisson* outcome (i.e., log link function), including session length in hours as an exposure variable (to capture variations in session length when visits were not exactly 90 min). The level 1 model accounts for variation within subjects over time, and the level 2 model accounts for variation between subjects. One of the benefits of HLM is its accommodation of missing data at level 1, and the ability to incorporate all participants who have been observed at least once (Raudenbush & Bryk, 2002, p. 199).

Level 1:

$$E(Y_{ti}|\pi_i) = \lambda_{ti} * SessionHours_{ti}$$

$$\log[\lambda_{ti}] = \eta_{ti}$$

$$\eta_{ti} = \pi_{0i} + \pi_{1i} * (age_{ti} - 36) + \pi_{2i} * (age_{ti} - 36)^2 + e_{ti}, e_{ti} \sim N(0, \sigma_t^2)$$

Level 2:

$$\pi_{0i} = \beta_{00} + \sum_{p=1}^n \beta_{0p} * C_{pi} + r_{0i}$$

$$\pi_{1i} = \beta_{10} + \sum_{p=1}^n \beta_{1p} * C_{pi} + r_{1i}$$

$$\pi_{2i} = \beta_{20} + \sum_{p=1}^n \beta_{2p} * C_{pi} + r_{2i}$$

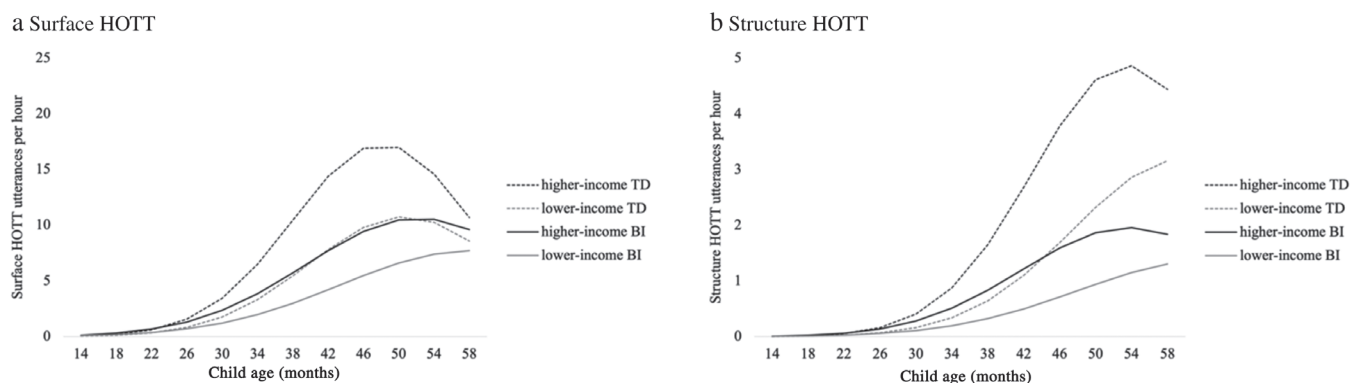
Mixed model:

$$\begin{aligned} \eta_{ti} = & \beta_{00} + \sum_{p=1}^n \beta_{0p} * C_{pi} + \beta_{10} * (age_{ti} - 36) \\ & + \sum_{p=1}^n \beta_{1p} * C_{pi} * (age_{ti} - 36) + \beta_{20} * (age_{ti} - 36)^2 \\ & + \sum_{p=1}^n \beta_{2p} * C_{pi} * (age_{ti} - 36)^2 + r_{0i} \\ & + r_{1i} * (age_{ti} - 36) + r_{2i} * (age_{ti} - 36)^2 \\ & + e_{ti}, e_{ti} \sim N(0, \sigma_t^2) \end{aligned}$$

In this model, the outcome  $Y_{ti}$  at level 1 is the number of child HOTT utterances (either surface or structure, depending on the analysis) for child  $i$  at time  $t$ . The *SessionHours<sub>ti</sub>* term represents the session length in hours, for child  $i$  at time  $t$ , and the  $\lambda_{ti}$  term represents the latent event rate per hour. The  $\eta_{ti}$  term is the link function, representing the natural logarithm of the HOTT utterance rate. The  $age_{ti}$  term represents the child's age in months, which we centered at 36 months (the middle of the study period). At level 1, children's HOTT utterances are thus predicted by the intercept ( $\pi_0$ ), interpreted as the child's HOTT production at 36 months; the growth ( $\pi_1$ ), or differences by linear age; and the acceleration ( $\pi_2$ ), or differences by quadratic age (a cubic term for age was tested but did not improve model fit). The residual  $e_{ti}$  is the portion of child  $i$ 's HOTT utterances at age point  $t$  not predicted by the intercept, growth, or acceleration. At level 2, the intercept ( $\pi_0$ ), growth ( $\pi_1$ ), and acceleration ( $\pi_2$ ) are predicted by each  $p$  in  $n$  different child-specific factors ( $C_i$ ) (e.g., brain-injury status, family income), and we also include random effects for the intercept ( $r_{0i}$ ), growth ( $r_{1i}$ ), and acceleration ( $r_{2i}$ ), allowing these to vary by individual  $i$ .

As the mixed model shows, child  $i$ 's HOTT utterances at age point  $t$  are predicted by the intercept ( $\beta_{00}$ ), effects on the intercept for each child-specific characteristic ( $\beta_{0p}$ ), the growth ( $\beta_{10}$ ), effects on the growth for each child-specific characteristic ( $\beta_{1p}$ ), the acceleration ( $\beta_{20}$ ), effects on the acceleration for each child-specific characteristic ( $\beta_{2p}$ ), and random effects ( $r_{0i}$ ,  $r_{1i}$ ,  $r_{2i}$ ). For the following analyses, we pay particular attention to the interactions between children's *brain-injured status* and the intercept, growth, and acceleration.

We modeled surface and structure HOTT trajectories separately. After running the unconditional growth models, we added child-specific factors to level 2 of the model in a stepwise manner. The level 2 variables that we tested include brain-injury status, family income, firstborn status, gender, and parent education. We assessed whether these variables explain variation between individuals in intercept, growth, and acceleration of surface and structure HOTT. The estimates of the fixed and random effects are



**Fig 2.** Model graphs of trajectories of surface and structure HOTT as a function of family income and brain-injured status. Higher income = 75th percentile, or \$100,000 per year; lower-income = 25th percentile, or \$42,500 per year. Scales differ for surface and structure HOTT. BI = brain-injured; HOTT = higher-order thinking talk; TD = typically-developing.

reported in Table 2 (for surface HOTT) and Table 3 (for structure HOTT).

Controlling for family income and firstborn status, we find significant interactions between brain-injury status and the intercept of both surface and structure HOTT (see  $\beta_{01}$  in both tables), suggesting that BI children produce fewer HOTT utterances than TD children. Moreover, the interactions between BI status and surface HOTT acceleration (see  $\beta_{21}$  in Table 2), and BI status and structure HOTT growth (see  $\beta_{11}$  in Table 3) were also significant, suggesting that not only the *frequency* of HOTT but also the *pace* with which HOTT utterances are produced is reduced in children with BI. Family income significantly predicts differences in both surface and structure HOTT intercept (see  $\beta_{02}$  in Tables 2 and 3), suggesting income-based disparities in the use of surface and structure HOTT. Finally, the firstborn status predicts the *structure*—but not the *surface*—HOTT intercept (see  $\beta_{03}$  in Table 3), suggesting that children who are the first-born or only-born child in their families use more structure HOTT than later-born children.

We tested interaction terms between BI status and income, but none of the interaction terms were significant (all  $p$ 's > .05) and their inclusion did not improve model fit ( $\chi^2(3) = 3.81, p = .28$  for surface;  $\chi^2(3) = 3.25, p = .36$ ) for structure, suggesting that family income plays a similar role for both TD children and children with BI. Neither of the other two child-specific characteristics that we tested (child gender and parental education) was significant, and their inclusion did not improve model fit (all  $p$ 's > .05). Overall, the analyses show that children with BI use less, and grow more slowly, in surface and structure HOTT use than their TD peers, controlling for family income and firstborn status.

In Figure 2, we illustrate these findings using model graphs (from Model 4 in Tables 2 and 3), showing the hypothetical trajectories of (a) surface and (b) structure HOTT, for later-born BI and TD children at the 25th (\$42,500) and

75th (\$100,000) income percentiles. Although we see the main effects of BI and income in both graphs, higher-income BI children have a similar HOTT trajectory to lower-income TD children (two middle lines in panels a and b), which suggests both a biological and an environmental component to children's HOTT.

### Comparing TD and BI Children's Surface and Structure HOTT by Lesion Type

Next, we examined differences between surface and structure HOTT utterances produced by children with BI as a function of their lesion type (CI vs. PV), again using HOTT utterances of TD children as a baseline (Figure 3). Figure 3a shows that, for surface HOTT, children with PV lesions follow a similar trajectory to TD children, whereas children with CI show later emergence and shallower growth than both children with PV lesions and TD children. Figure 3b shows a different pattern for structure HOTT, such that children with either CI or PV lesions show shallower trajectories than the TD children. (See Fig. S1 for figures by lesion size and laterality).

We tested the effects of lesion type again using HLM, but instead of including BI status as a dummy variable, we include dummy variables for both CI and PV lesions (retaining TD children as the reference category). The results are presented in Table 4.

Controlling for family income and firstborn status, children with CI differ from TD children in surface HOTT intercept and acceleration (see  $\beta_{03}$  and  $\beta_{23}$  in left columns), and structure HOTT intercept and growth (see  $\beta_{03}$  and  $\beta_{13}$  in right columns). Additionally, children with PV lesions differ from TD children in acceleration and growth of surface HOTT (see estimates for  $\beta_{14}$  and  $\beta_{24}$  in left columns) and marginally differ from TD children in the acceleration of structure HOTT (see estimate for  $\beta_{14}$  in right columns).

**Table 2**  
Hierarchical Linear Models to Predict Child Surface HOTT Utterances

	<i>Model 1</i> (unconditional)	<i>Model 2</i> (adding BI status)	<i>Model 3</i> (adding family income)	<i>Model 4</i> (adding firstborn status)
Fixed effects	$\beta$ ( $SE_{\beta}$ ) <i>df</i> = 106	$\beta$ ( $SE_{\beta}$ ) <i>df</i> = 106	$\beta$ ( $SE_{\beta}$ ) <i>df</i> = 106	$\beta$ ( $SE_{\beta}$ ) <i>df</i> = 106
Intercept, $\beta_{00}$	1.64*** (0.09)	1.77*** (0.11)	1.87*** (0.09)	1.78*** (0.14)
Growth (age), $\beta_{10}$	0.11*** (0.006)	0.12*** (0.008)	0.12*** (0.008)	0.12*** (0.01)
Acceleration (age <sup>2</sup> ), $\beta_{20}$	-0.004*** (0.0003)	-0.004*** (0.0003)	-0.004*** (0.0004)	-0.005*** (0.0005)
BI, $\beta_{01}$		-0.29 (0.18)	-0.56** (0.19)	-0.57** (0.19)
BI × Age, $\beta_{11}$		-0.02 <sup>^</sup> (0.01)	-0.02 (0.01)	-0.02 (0.01)
BI × Age <sup>2</sup> , $\beta_{21}$		0.002*** (0.0005)	0.002*** (0.0005)	0.002*** (0.0005)
Income, $\beta_{02}$			0.01*** (0.003)	0.01*** (0.0003)
Income × Age, $\beta_{12}$			-0.00009 (0.0002)	-0.00009 (0.0002)
Income × Age <sup>2</sup> , $\beta_{22}$			0.00001 (0.0001)	-0.00001 (0.00001)
Firstborn, $\beta_{03}$				0.18 (0.16)
Firstborn × Age, $\beta_{13}$				-0.0009 (0.01)
Firstborn × Age <sup>2</sup> , $\beta_{23}$				0.0004 (0.0005)
Random effects	<i>SD</i> <i>df</i> = 93 <sup>a</sup>	<i>SD</i> <i>df</i> = 93 <sup>a</sup>	<i>SD</i> <i>df</i> = 93 <sup>a</sup>	<i>SD</i> <i>df</i> = 93 <sup>a</sup>
Intercept, $r_0$	0.91***	0.89***	0.84***	0.83***
Growth (age), $r_1$	0.06***	0.06***	0.06***	0.06***
Acceleration (age <sup>2</sup> ), $r_2$	0.003***	0.002***	0.002***	0.002***
Goodness of fit	22,094.48 (9)	22,084.42 (12)	22,069.60 (15)	22,062.48 (18)
$\Delta\chi^2$ from previous model	—	10.06*	14.81**	7.13 <sup>^</sup>

Note. Typically-developing children are the reference category. BI = brain-injured; HOTT = higher-order thinking talk.

<sup>a</sup>The chi-square statistics reported are based on only 97 of 110 units that had sufficient data for computation. Fixed effects and variance components are based on all the data.

\*\*\**p* < .001, \*\**p* < .01, \**p* < .05, <sup>^</sup>*p* < .10.

**Table 3**  
Hierarchical Linear Models to Predict Child Structure HOTT Utterances

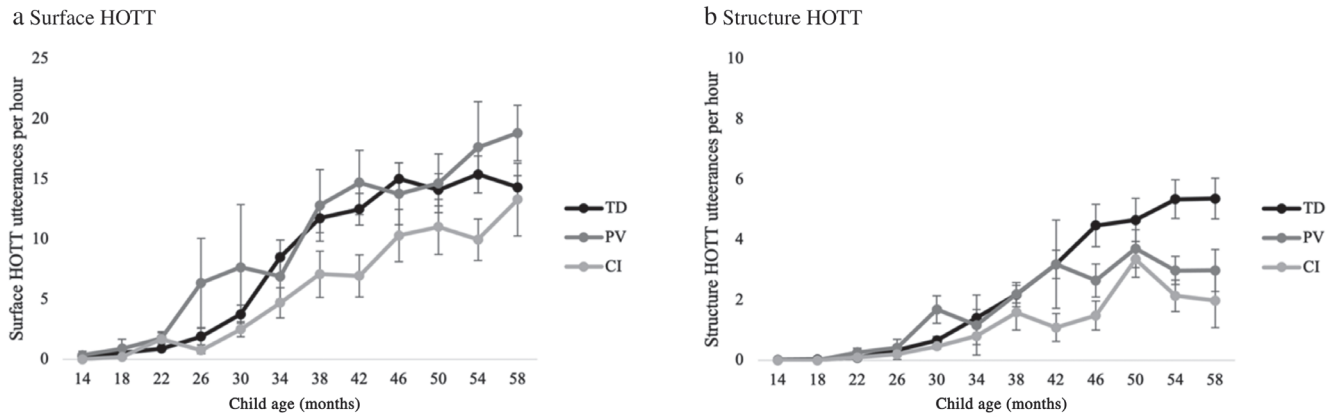
	<i>Model 1</i> (unconditional)	<i>Model 2</i> (adding BI status)	<i>Model 3</i> (adding family income)	<i>Model 4</i> (adding firstborn status)
Fixed effects	$\beta$ ( $SE_{\beta}$ ) <i>df</i> = 106	$\beta$ ( $SE_{\beta}$ ) <i>df</i> = 106	$\beta$ ( $SE_{\beta}$ ) <i>df</i> = 106	$\beta$ ( $SE_{\beta}$ ) <i>df</i> = 106
Intercept, $\beta_{00}$	-0.20 (0.13)	-0.10 (0.16)	0.06 (0.15)	-0.30 (0.20)
Growth (age), $\beta_{10}$	0.14*** (0.007)	0.15*** (0.009)	0.15*** (0.009)	0.16*** (0.01)
Acceleration (age <sup>2</sup> ), $\beta_{20}$	-0.003*** (0.0003)	-0.004*** (0.0004)	-0.004*** (0.0005)	-0.004*** (0.0006)
BI, $\beta_{01}$		-0.23 (0.26)	-0.61* (0.26)	-0.62* (0.26)
BI × Age, $\beta_{11}$		-0.03* (0.01)	-0.03* (0.02)	-0.04* (0.01)
BI × Age <sup>2</sup> , $\beta_{21}$		0.0004 (0.0007)	0.001 (0.0007)	0.001 (0.0008)
Income, $\beta_{02}$			0.02*** (0.005)	0.02*** (0.004)
Income × Age, $\beta_{12}$			-0.00008 (0.0003)	-0.00007 (0.0003)
Income × Age <sup>2</sup> , $\beta_{22}$			-0.00002 (0.00001)	-0.00002 (0.00001)
Firstborn, $\beta_{03}$				0.64** (0.23)
Firstborn × Age, $\beta_{13}$				-0.02 (0.01)
Firstborn × Age <sup>2</sup> , $\beta_{23}$				0.0001 (0.0008)
Random effects	<i>SD</i> <i>df</i> = 93 <sup>a</sup>	<i>SD</i> <i>df</i> = 93 <sup>a</sup>	<i>SD</i> <i>df</i> = 93 <sup>a</sup>	<i>SD</i> <i>df</i> = 93 <sup>a</sup>
Intercept, $r_0$	1.21***	1.21***	1.12***	1.07***
Growth (age), $r_1$	0.05***	0.05***	0.05***	0.04***
Acceleration (age <sup>2</sup> ), $r_2$	0.002***	0.002***	0.002***	0.002***
Goodness of fit	9,312.03 (9)	9,292.95 (12)	9,278.62 (15)	9,270.15 (18)
$\Delta\chi^2$ from the previous model	—	19.08***	14.32**	8.47*

Note. Typically developing children are the reference category. BI = brain-injured; HOTT = higher-order thinking talk.

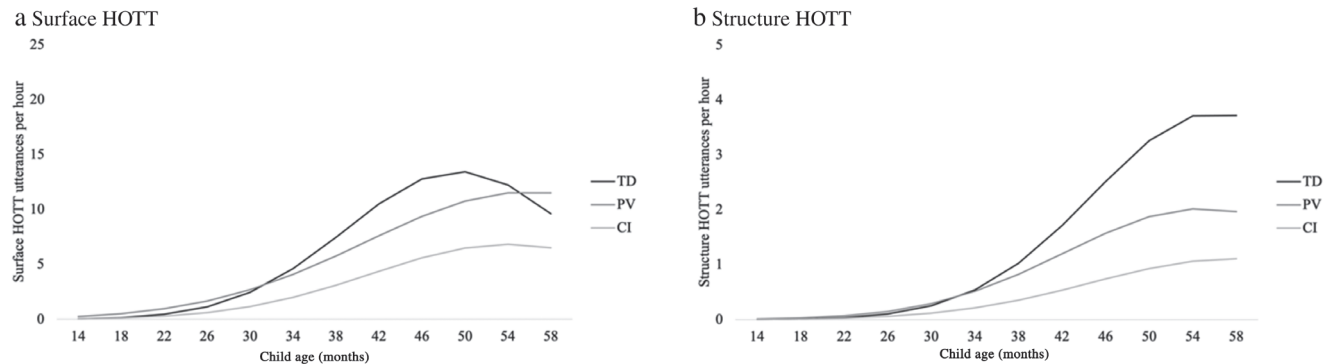
<sup>a</sup>The chi-square statistics reported are based on only 97 of 110 units that had sufficient data for computation. Fixed effects and variance components are based on all the data.

\*\*\**p* < .001, \*\**p* < .01, \**p* < .05.





**Fig 3.** Mean number of surface and structure HOTT utterances produced by TD and BI children as a function of lesion type. Scales differ for surface and structure HOTT. Error bars  $\pm 1$  SE. BI = brain-injured; CI = cerebrovascular infarct; HOTT = higher-order thinking talk; PV = periventricular lesion; TD = typically-developing.



**Fig 4.** Model graphs of trajectories of surface and structure HOTT as a function of BI children's lesion type. Scales differ for surface and structure HOTT. BI = brain-injured; CI = cerebrovascular infarct; HOTT = higher-order thinking talk; PV = periventricular lesion; TD = typically-developing.

However, children with PV lesions do *not* differ in the total amount of either surface or structure HOTT (see estimates for  $\beta_{04}$ ). This suggests that children with PV lesions and TD children use similar *amounts* of surface and structure HOTT, though the *pace* of HOTT use in children with PV lesions is reduced. In contrast, both the amount *and* pace of surface and structure HOTT use are reduced in children with CI (see Tables S4 and S5 for results by lesion size and laterality, which show that both children with small/medium and large, as well as left and right hemisphere lesions, produce less surface and structure HOTT than TD children).

In Figure 4, we illustrate these findings with model graphs (using Model 2 in Table 4), which show the hypothetical trajectories of (a) surface and (b) structure HOTT for later-born children with CI and PV lesions and TD children at the mean income of the combined sample (\$70,000). Children with PV lesions follow similar trajectories as TD children for surface and structure HOTT, though growth is

shallower. Children with CI follow shallower trajectories for both surface and structure HOTT, compared to both TD children and children with PV lesions.

### Surface and Structure HOTT Within BI Sample by Lesion Type

In our final set of analyses, we restrict our sample only to BI children and compare the trajectories of surface and structure HOTT between children with the two types of lesions. Table 5 presents the results of the fixed effects from the models (see Table S6 for results by lesion size and laterality).

These analyses reveal that children with CI differ from children with PV lesions in the amount of both surface and structure HOTT produced (see estimate of  $\beta_{01}$  in both columns), though neither growth nor acceleration is significantly different. This provides additional evidence that children with PV lesions use more surface and structure HOTT than children with CI.

## DISCUSSION

Our study is the first to describe the emerging developmental trajectories of HOTT in children with early BI. In this study, we compared TD and BI children's spontaneous talk at home during the early preschool years and asked three main research questions about the complexity of their spontaneous talk.

First, we examined whether the developmental trajectories of surface and structure HOTT differ for TD and BI children between the ages of 14 and 58 months. Although the TD and BI children produce similar amounts of non-HOTT utterances, our results demonstrate that TD children produce more surface and structure HOTT, and grow more quickly in their use of both types of HOTT, than children with BI, with particularly large gaps in structure HOTT.

Second, we examined the developmental trajectories of surface and structure HOTT by type of BI (PV lesions vs. CI) to explore how higher-order thinking, a cognitively complex task, is affected by the nature, timing, and size of the BI. We found no differences in the *amount* of HOTT produced between children with smaller and earlier occurring brain injuries that primarily affect white matter (PV lesions) and TD children regarding the use of HOTT. This highlights the remarkable plasticity of the infant brain for emerging higher-order thinking skills for this type of lesion.

However, children with larger and later-occurring brain injuries that primarily affect gray matter (CI) show later emergence and shallower growth of higher-order thinking talk. Children with this type of BI particularly show negative effects for more complex structure HOTT use. This type of early BI appears to make it more difficult for children to link ideas that are more disparate to one another—to make inferences, draw comparisons and analogies, think in terms of taxonomic relations, and abstract from specific instances—at least insofar as the children can express these ideas in language. These findings are in-line with previous research demonstrating that while children with prenatal or perinatal unilateral BI exhibit remarkable plasticity for early language functions, they have difficulty with more complex aspects of language particularly when lesions are larger (Demir et al., 2010, 2015).

Finally, we examined how environmental factors influence the developmental trajectories of higher-order thinking. We examined family income as the primary environmental factor, as it may directly impact or act as a proxy for other environmental differences in home environments. Results suggest that family income influences the use of surface and structure HOTT in both TD and BI children. Children growing up in higher-income environments produce more HOTT, and grow more quickly in HOTT production,

compared to children growing up in lower-income environments. As income did not interact with BI status, this suggests the effects of income are similar for both TD and BI children, which is supported by the model graphs in Figure 2.

### Limitations

This study is one of the first analyses of naturally occurring and spontaneous HOTT collected in the home of young, preschool-aged children. Our goal here was to describe the developmental trajectories of HOTT in a sample of TD and brain-injured children. Because the results are correlational, they should be considered exploratory.

The sample of children with early BI was diverse. Although we examine differences between children with PV lesions and CI, it is unclear whether these findings will generalize to other populations of children with similar brain injuries or to children with different kinds of brain injuries. Additionally, there may be other factors, both biological and cognitive, not explored in this paper that significantly impact the developmental trajectories of early higher-order thinking and language. In terms of biology, we find that CI is associated with lower surface and structure HOTT compared to PV lesions. However, we do not know which of the biological factors associated with these lesions contribute to this difference—their effect on predominantly gray versus white matter, their relative size, and/or the timing of the lesion. These are important questions for future research. It is also possible that the presence of recurrent seizures may affect HOTT, by transforming a focal lesion into a more global pathology that can interfere with functional plasticity (Levine, Beharelle Raja, Demir, & Small, 2015; Stiles, Reilly, Levine, Trauner, & Nass, 2012; Vargha-Khadem, Isaacs, Van Der Werf, Robb, & Wilson, 1992). In terms of cognitive factors, domain knowledge (Gentner & Rattermann, 1991) or executive function resources (Richland & Burchinal, 2013) may also play a role in HOTT. More research is necessary to examine these possibilities.

Finally, the data were collected in videotaped sessions in the home. We make the assumption that children's experiences captured during data collection are representative of children's typical experiences at home. Although all families were instructed to go about their typical routines, the presence of a video camera and experimenter in the home may have led to atypical behaviors. However, most research using this methodology has found that participants behave as they normally do, as scripted patterns of behavior for extended periods are difficult to maintain (Jewitt, 2012). As each of the visit recordings lasted about 90 min, we likely captured typical behaviors and talk that occur in the home.

**Table 4**

Hierarchical Linear Models to Predict Child (a) Surface and (b) Structure HOTT Utterances as a Function of BI Children’s Lesion Type

	<i>(a) Surface HOTT</i>		<i>(b) Structure HOTT</i>	
	<i>Model 1</i> <i>(income and firstborn)</i>	<i>Model 2</i> <i>(adding lesion type)</i>	<i>Model 1</i> <i>(income and firstborn)</i>	<i>Model 2</i> <i>(adding lesion type)</i>
Fixed effects	$\beta$ ( $SE_{\beta}$ ) <i>df</i> = 105	$\beta$ ( $SE_{\beta}$ ) <i>df</i> = 103	$\beta$ ( $SE_{\beta}$ ) <i>df</i> = 105	$\beta$ ( $SE_{\beta}$ ) <i>df</i> = 103
Intercept, $\beta_{00}$	1.55*** (0.12)	1.79*** (0.13)	-0.55** (0.19)	-0.28 (0.20)
Growth (age), $\beta_{10}$	0.11*** (0.01)	0.12*** (0.01)	0.15*** (0.01)	0.16*** (0.01)
Acceleration (age <sup>2</sup> ), $\beta_{20}$	-0.004*** (0.0005)	-0.005*** (0.0005)	-0.004*** (0.0005)	-0.004*** (0.0006)
Income, $\beta_{01}$	0.008* (0.003)	0.01*** (0.003)	0.01** (0.004)	0.02** (0.005)
Income $\times$ Age, $\beta_{11}$	-0.0002 (0.0002)	-0.00007 (0.0002)	-0.0003 (0.0003)	-0.00006 (0.0003)
Income $\times$ Age <sup>2</sup> , $\beta_{21}$	0.000001 (0.000009)	-0.00001 (0.00001)	-0.00001 (0.00001)	-0.00002 (0.00002)
Firstborn, $\beta_{02}$	0.16 (0.17)	0.17 (0.16)	0.60* (0.24)	0.62** (0.23)
Firstborn $\times$ Age, $\beta_{12}$	0.0002 (0.01)	0.0006 (0.01)	-0.02 (0.01)	-0.02 (0.01)
Firstborn $\times$ Age <sup>2</sup> , $\beta_{22}$	0.0004 (0.0006)	0.0004 (0.0006)	0.0002 (0.0007)	0.0002 (0.0007)
CI, $\beta_{03}$		-0.86*** (0.23)		-1.00** (0.31)
CI $\times$ Age, $\beta_{13}$		-0.01 (0.02)		-0.03* (0.02)
CI $\times$ Age <sup>2</sup> , $\beta_{23}$		0.002* (0.0007)		0.001 (0.0009)
PV, $\beta_{04}$		-0.20 (0.28)		-0.14 (0.32)
PV $\times$ Age, $\beta_{14}$		-0.03* (0.01)		-0.04^ (0.02)
PV $\times$ Age <sup>2</sup> , $\beta_{24}$		0.002*** (0.0006)		0.0009 (0.001)
Random effects	<i>SD</i> <i>df</i> = 92 <sup>a</sup>	<i>SD</i> <i>df</i> = 90 <sup>a</sup>	<i>SD</i> <i>df</i> = 94 <sup>b</sup>	<i>SD</i> <i>df</i> = 90 <sup>a</sup>
Intercept, $r_0$	0.88***	0.81***	1.11***	1.04***
Growth (age), $r_1$	0.06***	0.06***	0.05***	0.05***
Acceleration (age <sup>2</sup> ), $r_2$	0.003***	0.002***	0.002***	0.002***
Goodness of fit	21,496.97 (15)	21,467.31 (21)	9,049.12 (15)	9,014.09 (21)
$\Delta\chi^2$ from model 1		29.66***		35.04***

Note: Typically-developing children are the reference category. BI = brain-injured; CI = cerebrovascular infarct; HOTT = higher-order thinking talk; PV = periventricular lesion.

<sup>a</sup>The chi-square statistics reported are based on only 95 of 108 units that had sufficient data for computation.

<sup>b</sup>The chi-square statistics reported are based on only 97 of 108 units that had sufficient data for computation. Fixed effects and variance components are based on all the data.

\*\*\* $p < .001$ , \*\* $p < .01$ , \* $p < .05$ , ^ $p < .10$ .

**Table 5**

Fixed Effects from Hierarchical Linear Models Within BI Sample to Predict Child (a) Surface and (b) Structure HOTT Utterances as a Function of BI Children’s Lesion Type

	<i>(a) Surface HOTT,</i> $\beta$ ( $SE_{\beta}$ ), <i>df</i> = 42	<i>(b) Structure HOTT,</i> $\beta$ ( $SE_{\beta}$ ), <i>df</i> = 42
Intercept, $\beta_{00}$	1.85*** (0.24)	0.20 (0.28)
Growth (age), $\beta_{10}$	0.09*** (0.01)	0.11*** (0.02)
Acceleration (age <sup>2</sup> ), $\beta_{20}$	-0.002*** (0.0004)	-0.003* (0.0008)
CI, $\beta_{01}$	-0.65* (0.30)	-0.88* (0.38)
CI $\times$ Age, $\beta_{11}$	0.02 (0.02)	0.01 (0.02)
CI $\times$ Age <sup>2</sup> , $\beta_{21}$	-0.0008 (0.0007)	0.0002 (0.001)

Note: Children with periventricular lesion are the reference category. BI = brain-injured; CI = cerebrovascular infarct; HOTT = higher-order thinking talk.

**Future Research**

This paper focuses on the similarities and differences in HOTT in a sample of TD and brain-injured children. An

open question remains about whether the differences stem from BI children’s challenges specific to language, reasoning abilities, or to the ability to reason using language. Our prior research on these same TD children shows that utterances displaying higher-order thinking tend to be longer and more syntactically complex than non-HOTT utterances. However, the majority of even structure HOTT utterances are expressed in simple utterances (i.e., they contain 0 or 1 verbs; Frausel et al., 2020). As HOTT can be produced using simple language, we theorize that children with BI are likely most impacted in their ability to reason using language. To further examine this question, future research can examine the linguistic complexity of the HOTT utterances produced by BI children to shed additional light on the relationship between complex reasoning, complex language, and the ability to express reasoning through language in BI children. Regardless of the underlying mechanism, the difficulty that BI children face in producing HOTT is likely to negatively impact their school readiness and early academic achievement.

Our findings also suggest that the social environment has the potential to impact HOTT trajectories. Family income, either as an independent factor or a proxy for other environmental factors, plays a role in both *surface* and *structure* HOTT, and this relationship holds for both TD children and children with prenatal or perinatal lesions. It is not surprising that environmental factors influence the developmental trajectories of HOTT, but more work is needed to elucidate the specific aspects of the environment that are linked to and impact early language and reasoning development. This study also found that the firstborn status plays a role in structure HOTT, possibly because firstborn or only-born children are likelier to receive more individualized language and cognitive inputs from their caregivers. Yet there are many more potential environmental factors that exist in different kinds of socioeconomic environments that could potentially affect the development of these complex skills (e.g., sleep quality, stress, social relations, and access to preschool).

In our future research, we plan to examine the role of the environmental input through parent talk. Specially, we are exploring whether children who receive particular kinds of parent support can develop stronger HOTT skills earlier in development. The children in our study are often scaffolded and supported by questions, prompts, and statements from their caregivers. We plan to examine specifically how parents use HOTT in spontaneous speech, and how parent HOTT input (or parent linguistic input more broadly) is related to children's emerging higher-order thinking skills. We also plan to examine the *timing* of parent HOTT: that is, whether early parent HOTT input is more important than later parent HOTT, whether later HOTT input is more important than early HOTT input, or whether there are cumulative effects of parent HOTT input on children's emerging higher-order thinking abilities (Silvey, Demir-Lira, Raudenbush, & Goldin-Meadow, 2021). If parental support significantly impacts children's developmental trajectories, this would provide evidence that these skills may be malleable and thus responsive to interventions that enhance parent supports. This would open up the exciting possibility that early HOTT can be fostered with certain kinds of input, potentially reducing achievement gaps between lower- and higher-income children, and TD and BI children.

In sum, we have demonstrated that children's spontaneously produced HOTT is related to specific types of early BI. We have also shown that there are environmental influences of family income on the development of children's HOTT. Finally, we have demonstrated how naturalistic data gathered in the home can provide critical and nuanced insights into the developmental trajectories of children's early reasoning and language abilities, for both TD children and children with early BI.

*Acknowledgments*—Thank you to Kristi Schonwald for your administrative and technical assistance, and to Cassie Freeman, Natalie Dowling, Alyssa Guillu, and Maryam Mohammad-Norgan for your coding assistance. The authors have no conflicts of interest to disclose.

## SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

**Appendix S1.** Supporting information.

## REFERENCES

- Avecilla-Ramírez, G. N., Ruiz-Correa, S., Marroquin, J. L., Harmony, T., Alba, A., & Mendoza-Montoya, O. (2011). Electrophysiological auditory responses and language development in infants with periventricular leukomalacia. *Brain and Language, 119*(3), 175–183.
- Bates, E., & Dick, F. (2002). Language, gesture, and the developing brain. *Developmental Psychobiology, 40*(3), 293–310.
- Bates, E., Reilly, J., Wulfeck, B., Dronkers, N., Opie, M., Fenson, J., ... Herbst, K. (2001). Differential effects of unilateral lesions on language production in children and adults. *Brain and Language, 79*(2), 223–265.
- Bates, E., & Roe, K. (2001). Language development in children with unilateral brain injury. In C. A. Nelson, & M. Luciana (Eds.), *Handbook of developmental cognitive neuroscience*. (pp. 281–307). Cambridge, MA: MIT Press.
- Bates, E., Thal, D., Trauner, D., Fenson, J., Aram, D., Eisele, J., & Nass, R. (1997). From first words to grammar in children with focal brain injury. *Developmental Neuropsychology, 13*(3), 275–343.
- Brown, A. L. (1989). Analogical learning and transfer: What develops? In S. Vosniadou, & A. Ortony (Eds.), *Similarity and analogical reasoning*. (pp. 369–412). New York, NY: Cambridge University Press.
- Bruner, J. (1973) *The relevance of education*. New York, NY: W.W. Norton & Company.
- Demir, Ö. E., Levine, S. C., & Goldin-Meadow, S. (2010). Narrative skill in children with early unilateral brain injury: A possible limit to functional plasticity. *Developmental Science, 13*(4), 636–647.
- Demir, Ö. E., Rowe, M. L., Heller, G., Goldin-Meadow, S., & Levine, S. C. (2015). Vocabulary, syntax, and narrative development in typically-developing children and children with early unilateral brain injury: Early parental talk about the “there-and-then” matters. *Developmental Psychology, 51*(2), 161–175.
- Feldman, H., Goldin-Meadow, S., & Gleitman, L. (1978). Beyond Herodotus: The creation of language by linguistically deprived deaf children. In A. Lock (Ed.), *Action, symbol, and gesture: The emergence of language*. (pp. 351–414). New York, NY: Academic Press.
- Feldman, H. M. (2005). Language learning with an injured brain. *Language Learning and Development, 1*(3–4), 265–288.



- Feldman, H. M., Holland, A. L., Kemp, S. S., & Janosky, J. E. (1992). Language development after unilateral brain injury. *Brain and Language*, 42(1), 89–102.
- Frausel, R. R., Richland, L. E., Levine, S. C., & Goldin-Meadow, S. (2021). Personal narrative as a “breeding ground” for higher-order thinking talk in early parent–child interactions. *Developmental Psychology*, 57(4), 519–534.
- Frausel, R. R., Silvey, C., Freeman, C., Dowling, N., Richland, L. E., Levine, S. C., & Goldin-Meadow, S. (2020). The origins of higher-order thinking lie in children’s spontaneous talk across the pre-school years. *Cognition*, 200, 104274.
- Gentner, D. (1983). Structure-mapping: A theoretical framework for analogy. *Cognitive Science*, 7(2), 155–170.
- Gentner, D., & Goldin-Meadow, S. (2003). Whither Whorf. In D. Gentner, & S. Goldin-Meadow (Eds.), *Language in mind: Advances in the study of language and cognition*. (pp. 3–14). Cambridge, MA: MIT Press.
- Gentner, D., & Rattermann, M. J. (1991). Language and the career of similarity. In S. A. Gelman, & J. P. Byrnes (Eds.), *Perspective on language and thought: Interrelations in development*. (pp. 225–277). London, England: Cambridge University Press.
- Goldin-Meadow, S. (2005) *The resilience of language: What gesture creation in deaf children can tell us about how all children learn language*. New York, NY: Psychology Press.
- Goldin-Meadow, S., Levine, S. C., Hedges, L. V., Huttenlocher, J., Raudenbush, S. W., & Small, S. L. (2014). New evidence about language and cognitive development based on a longitudinal study: Hypotheses for intervention. *American Psychologist*, 69(6), 588–599.
- Goswami, U., & Brown, A. L. (1990). Higher-order structure and relational reasoning: Contrasting analogical and thematic relations. *Cognition*, 36(3), 207–226.
- Gutiérrez-Hernández, C. C., Harmony, T., & Carlier, M. E. M. (2018). Behavioral and electrophysiological study of attention process in preterm infants with cerebral white matter injury. *Psychology & Neuroscience*, 11(2), 132–145.
- Hoff, E. (2003). Causes and consequences of SES-related differences in parent-to-child speech. In M. H. Bornstein, & R. H. Bradley (Eds.), *Monographs in parenting series. Socioeconomic status, parenting, and child development*. (pp. 147–160). Mahwah, NJ: Lawrence Erlbaum Associates Publishers.
- Holyoak, K. J., Junn, E. N., & Billman, D. O. (1984). Development of analogical problem-solving skill. *Child Development*, 55, 2042–2055.
- Holyoak, K. J., Thagard, P., & Sutherland, S. (1995). Mental leaps: Analogy in creative thought. *Nature*, 373(6515), 572–572.
- Jewitt, C. (2012). *An introduction to using video for research*. National Centre for Research Methods Working Paper (unpublished). Retrieved from [http://eprints.ncrm.ac.uk/2259/4/NCRM\\_workingpaper\\_0312.pdf](http://eprints.ncrm.ac.uk/2259/4/NCRM_workingpaper_0312.pdf)
- Koenig, J. A. (2015). *Assessing 21st century skills: Summary of a workshop*. Retrieved from <https://www.learntechlib.org/p/159080/>
- Lawrence, J. F., & Snow, C. E. (2010). Oral discourse and reading. In P. Michael Kamill, D. Pearson, E. B. Moje & P. Afflerbach (Eds.), *Handbook of reading research*. (Vol. IV, pp. 320–339). Abingdon: Routledge.
- Leroy, S., Parrisé, C., & Maillart, C. (2012). Analogical reasoning in children with specific language impairment. *Clinical Linguistics & Phonetics*, 26(4), 380–396.
- Levine, S. C., Beharelle Raja, A., Demir, Ö. E., & Small, S. (2015). Perinatal focal brain injury: Scope and limits of plasticity for language functions. In G. S. Hickok, & S. L. Small (Eds.), *Neurobiology of language*. (pp. 969–983). San Diego, CA: Elsevier. <https://doi.org/10.1016/B978-0-12-407794-2.00077-8>
- Lewis, A., & Smith, D. (1993). Defining higher-order thinking. *Theory Into Practice*, 32(3), 131–137.
- Moreno-Aguirre, A. J., Santiago-Rodríguez, E., Harmony, T., Fernández-Bouzas, A., & Porrás-Kattz, E. (2010). Analysis of auditory function using brainstem auditory evoked potentials and auditory steady state responses in infants with perinatal brain injury. *International Journal of Audiology*, 49(2), 110–115.
- Özçalışkan, Ş., Goldin-Meadow, S., Gentner, D., & Mylander, C. (2009). Does language about similarity play a role in fostering similarity comparison in children? *Cognition*, 112(2), 217–228.
- Raudenbush, S. W., & Bryk, A. S. (2002) *Hierarchical linear models: Applications and data analysis methods*. New York, NY: Sage.
- Reilly, J. S., Bates, E. A., & Marchman, V. A. (1998). Narrative discourse in children with early focal brain injury. *Brain and Language*, 61(3), 335–375.
- Reilly, J. S., Losh, M., Bellugi, U., & Wulfeck, B. (2004). “Frog, where are you?” Narratives in children with specific language impairment, early focal brain injury, and Williams syndrome. *Brain and Language*, 88(2), 229–247.
- Reilly, J. S., Wasserman, S., & Appelbaum, M. (2013). Later language development in narratives in children with perinatal stroke. *Developmental Science*, 16(1), 67–83.
- Richland, L. E., & Burchinal, M. R. (2013). Early executive function predicts reasoning development. *Psychological Science*, 24(1), 87–92.
- Richland, L. E., Morrison, R. G., & Holyoak, K. J. (2006). Children’s development of analogical reasoning: Insights from scene analogy problems. *Journal of Experimental Child Psychology*, 94(3), 249–273.
- Richland, L. E., & Simms, N. (2015). Analogy, higher-order thinking, and education. *Wiley Interdisciplinary Reviews: Cognitive Science*, 6(2), 177–192.
- Richland, L. E., Zur, O., & Holyoak, K. J. (2007). Cognitive supports for analogies in the mathematics classroom. *Science*, 316(5828), 1128–1129.
- Rowe, M. L. (2012). A longitudinal investigation of the role of quantity and quality of child-directed speech in vocabulary development. *Child Development*, 83(5), 1762–1774.
- Rowe, M. L., & Goldin-Meadow, S. (2009). Differences in early gesture explain SES disparities in child vocabulary size at school entry. *Science*, 323(5916), 951–953.
- Rowe, M. L., Levine, S. C., Fisher, J. A., & Goldin-Meadow, S. (2009). Does linguistic input play the same role in language learning for children with and without early brain injury? *Developmental Psychology*, 45(1), 90–102.
- Schleppegrell, M. J. (2004) *The language of schooling: A functional linguistics perspective*. Mahwah, NJ: Erlbaum.
- Silvey, C., Demir-Lira, Ö. E., Goldin-Meadow, S., & Raudenbush Stephen, W. (2021). Effects of time-varying parent input on children’s language outcomes differ for vocabulary and syntax. *Psychological Science*, 32(4), 536–548. <http://dx.doi.org/10.1177/0956797620970559>

- Staudt, M., Gerloff, C., Grodd, W., Holthausen, H., Niemann, G., & Krägeloh-Mann, I. (2004). Reorganization in congenital hemiparesis acquired at different gestational ages. *Annals of Neurology*, *56*(6), 854–863.
- Stiles, J., Reilly, J., Paul, B., & Moses, P. (2005). Cognitive development following early brain injury: Evidence for neural adaptation. *Trends in Cognitive Sciences*, *9*(3), 136–143.
- Stiles, J., Reilly, J. S., Levine, S. C., Trauner, D. A., & Nass, R. (2012). *Neural plasticity and cognitive development: Insights from children with perinatal brain injury*. New York, NY: Oxford University Press.
- Thal, D. J., Marchman, V., Stiles, J., Aram, D., Trauner, D., Nass, R., & Bates, E. (1991). Early lexical development in children with focal brain injury. *Brain and Language*, *40*(4), 491–527.
- Vargha-Khadem, F., Isaacs, E., Van Der Werf, S., Robb, S., & Wilson, J. (1992). Development of intelligence and memory in children with hemiplegic cerebral palsy: The deleterious consequences of early seizures. *Brain*, *115*, 315–329.
- Wang, S. H., & Baillargeon, R. (2008). Can infants be “taught” to attend to a new physical variable in an event category? The case of height in covering events. *Cognitive Psychology*, *56*(4), 284–326.