Developmental trajectories of early higher-order thinking talk differ

for typically-developing children and children with unilateral brain injuries

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Lay Abstract

We examined talk produced by 1–5-year-old children going about their everyday routines. Some children had experienced early brain injuries, and we compared their talk to the talk of typically-developing children, to better understand how brain injury may affect language and thinking abilities. Children with brain injuries generally expressed less complex thinking in their talk (e.g., they compared how things look, as opposed to how things work), which may impact their school readiness and success.

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 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 typically-developing children. In contrast, children with smaller and earlier-occurring periventricular lesions produce HOTT at comparable rates to typically-developing 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.

Keywords: higher-order thinking, reasoning development, naturalistic observation, cognition, functional plasticity, early unilateral brain injury

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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 3-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; Lewis & Smith, 1993; Frausel, et al., 2020). 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 children in the acquisition of these skills (Richland & Burchinal, 2013), as well as differences among children with specific language impairments (Leroy, Parisse, & Maillart, 2012). Here, we focus on higher-order thinking talk in a group of children with pre- or perinatal unilateral brain injury. 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; Demir, Levine, & Goldin-Meadow, 2010; Demir, et al., 2015; Gutiérrez-Hernández, Harmony, &

Carlier, 2018; Moreno-Aguirre, et al., 2010; Reilly, Bates, & Marchman, 1998; Reilly, Wasserman, & Appelbaum, 2013).

In this paper, we ask whether the early developmental trajectories of higher-order thinking talk differ between typically-developing children (TD) and children with early brain injury (BI). Additionally, we examine other factors, such as family income (an index of environmental variation) and characteristics of the brain injury (an index of biological variation), that have the potential to affect the developmental trajectories of HOTT (see 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 early injury.

Defining Higher-Order Thinking Talk

We conceptualize higher-order thinking talk (HOTT) as the cognitive ability to linguistically construct explicit reference to inference, comparison, abstraction, and hierarchy (Frausel, et al., 2020). Although pre-linguistic 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 (see 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 typically-developing 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, 2003). 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. 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 typically-developing 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 typically-developing 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 typically-developing 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 pre- or peri-natal 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 (e.g., Bates & Roe, 2001; Bates, et al., 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 level of functioning as the language abilities of TD children (e.g., Bates & Dick, 2002; Feldman, 2005). However, children with BI often display delays in their language development (Thal, et al., 1991; Bates, et al., 1997; Feldman, et al., 1992), as well as more marked differences for particularly challenging language skills, such as narrative and complex sentence structure (Demir, Levine, & Goldin-Meadow, 2010; Demir, et al., 2015; Reilly et al., 1998; Reilly et al., 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 carryover 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 appearance or object-level similarities (see 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, Morrison, & Holyoak, 2006) and when they have strong prior knowledge in the domain (Goswami & Brown, 1990; Brown, 1989; Gentner & Rattermann, 1991). Importantly, the spontaneous HOTT identified in typically-developing 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 differing developmental trajectories for surface and structure HOTT will be similar for the 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, Zur, & Holyoak, 2007). One possibility is that brain injury leads to delays in the emergence and growth of *both* surface and structure HOTT. This pattern would indicate that higher-order thinking talk is broadly impacted by early brain injury. Alternatively, early brain injury could differentially impact the development of surface vs. structure HOTT. In particular, early brain injury 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, Bates, & Marchman, 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 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 two types of lesions, periventricular lesions (PV) or cerebrovascular infarcts (CI). These lesions differ in size, occur at different times in gestational development, and affect different types of matter. Periventricular lesions are smaller, usually occur in the early to mid-third trimester of pregnancy, and primarily affect white matter. Cerebrovascular infarcts are larger, occur later in the third trimester, and primarily affect gray matter (Staudt, et al., 2004; Demir, et al., 2015; Rowe, et al., 2009). 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. Since CI lesions tend to occur later and to be larger, this 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 brain injury (PV 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 hours 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 brain injury using spontaneous data from naturalistic settings, and explores whether and how these trajectories differ from those of typically-developing children.

Methods

Participants

Participants were 64 typically-developing 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, and 61% ($n = 27$) had CI. We focus on lesion type here, which captures variance in size and timing of injury, rather than on lesion laterality and size per se, due to the relatively small sample size (see Supplementary Materials, Table S1, for details).

Procedure

Families were visited in their homes every 4 months between 14 and 58 months, or, for the children with brain injury, beginning at the age point at which they entered the study. This varied for the children with brain injury because of the rarity of this condition and the limited population of children with early brain injury, which necessitated relaxing the time of entry into the study. The BI children's average age of 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 in Supplementary Materials). At each visit,

researchers recorded 90 minutes 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 ABC's) or prayer (e.g.,

reciting the Lord's Prayer), was not transcribed. 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 higher-order thinking talk (HOTT),

talk that indexes two or more representations and constructs a relational bridge or link

between them (see 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.

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 structural understanding of the referents indexed (Richland & Simms, 2015; Frausel, et al., 2020). 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 deeper understanding of the relations being linked (Richland & Simms, 2015; Frausel, et al., 2020). Structure HOTT is often decontextualized or abstract (e.g., "She's sad because she misses her momma"; see also Frausel, et al., 2021). See Table S3 in the supplementary materials 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 was conducted on 212 transcripts (18% of the 1,150 transcripts) coded by two or more people. 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* and *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) to TD children, again examining impacts of

other child-specific factors such as income. (3) We examine 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* and *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 minutes). 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, pg. 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:

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

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$_{ti}$ term represents the session length in hours, for child i at time t, and the λ_{ti}

term represents the latent event rate per hour. The η_{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. At level 1, children's HOTT

utterances are thus predicted by the intercept (π_0), interpreted as the child's HOTT

production at 36 months; the growth (π_1), or differences by linear age; and the

acceleration (π_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 (π_0), growth (π_1), and acceleration (π_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 (β_{00}), effects on the intercept for each child-specific characteristic (β_{0p}), the growth (β_{10}), effects on the growth for each child-specific characteristic (β_{1p}), the acceleration (β_{20}), effects on the acceleration for each child-specific characteristic (β_{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, first-born 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 reported in Table 2 (for surface HOTT) and Table 3 (for structure HOTT).

Controlling for family income and first-born status, we find significant interactions between brain-injury status and the intercept of both surface and structure HOTT (see β_{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 β_{21} in Table 2), and BI status and structure HOTT growth (see

β_{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 β_{02} in Tables 2 and 3), suggesting income-based disparities in use of

surface and structure HOTT. Finally, first-born status predicts the *structure*—but not the

surface—HOTT intercept (see β_{03} in Table 3), suggesting that children who are the first-

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 > 0.05) and their inclusion did not improve

model fit ($\chi^2(3) = 3.81$, $p = 0.28$ for surface; $\chi^2(3) = 3.25$, $p = 0.36$) 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,

parental education) was significant, and their inclusion did not improve model fit (all p's

> 0.05). Overall, the analyses show that children with BI use less, and grow more slowly,

in use of surface and structure HOTT than their TD peers, controlling for family income

and first-born 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 main effects of brain-injury 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 versus PV), again

using HOTT utterances of TD children as a baseline (see Figure 3). Figure 3a shows that,

for surface HOTT, children with PV follow a similar trajectory to TD children, whereas

children with CI show later emergence and shallower growth than both children with PV

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 Figure S1 in Supplementary Materials 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 (retaining

TD children as the reference category). As shown in Table 4, controlling for family

income and first-born status, children with CI differ from TD children in surface HOTT

intercept and acceleration (see β_{03} and β_{23} in left columns), and structure HOTT intercept

and growth (see β_{03} and β_{13} in right columns). Additionally, children with PV differ from

TD children in acceleration and growth of surface HOTT (see estimates for β_{14} and β_{24} in

left columns), and marginally differ from TD children in acceleration of structure HOTT

(see estimate for β_{14} in right columns).

However, children with PV do *not* differ in total amount of either surface or

structure HOTT (see estimates for β_{04}). This suggests that children with PV and TD

children use similar *amounts* of surface and structure HOTT, though the *pace* of HOTT

use in children with PV is reduced. In contrast, both the amount *and* pace of surface and

structure HOTT use is reduced in children with CI. (See Tables S4 and S5 in the

Supplementary materials 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 TD, CI, and PV 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.

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 in the Supplementary Materials for results by lesion size and laterality),

revealing that children with CI differ from children with PV in the amount of both

surface and structure HOTT produced (see estimate of β_{01} in both columns), though

neither growth nor acceleration is significantly different. This provides additional

evidence that children with PV use more surface and structure HOTT than children with

CI.

Discussion

Our study is the first to describe the emerging developmental trajectories of higher-order thinking talk in children with early brain injury. In this study, we compared TD and BI children's spontaneous talk at home during the early preschools 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 brain injury (PV vs CI) to explore how higher-order thinking, a cognitively complex task, is affected by the nature, timing, and size of the brain injury. We found no differences in *amount* of HOTT produced between children with smaller and earlier-occurring brain injuries that primarily affect white matter (PV lesions) and TD children regarding 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 brain injury particularly show negative effects for more complex structure HOTT use. This type of early brain injury 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 pre- or perinatal unilateral brain injury exhibit remarkable plasticity for early language functions, they have difficulty with more complex aspects of language particularly when lesions are larger (e.g., Demir, Levine, & Goldin-Meadow, 2010; Demir, et al., 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 higher-order thinking talk collected in the home of young, pre-school aged children. Our goal here was to describe the developmental trajectories of HOTT in a sample of typically-developing and brain-injured children. Because the results are correlational, they should be considered exploratory.

The sample of children with early brain injury was diverse. Although we examine differences between children with periventricular lesions and cerebrovascular infarcts, 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 than PL. However, we do not know which of the biological factors associated with these lesions contributes to this difference—their affect 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, et al., 2015; Stiles, et al., 2012; Vargha-Khadem, et al., 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 of time are difficult to maintain (Jewitt, 2012). As each of the visit recordings lasted about 90 minutes, we likely captured typical behaviors and talk that occur in the home.

Future Research

This paper focuses on the similarities and differences in higher-order thinking talk in a sample of typically-developing and brain injured children. An open question remains about whether the differences stem from BI children's challenges specific to language, to reasoning abilities, or to the ability to reason using language. Our prior research on these same typically-developing children show 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 BI children face producing higher-order thinking talk 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 typically-developing children and children with pre-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 firstborn-status plays a role in structure HOTT, possibly because first- 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 socio-economic environments that could potentially affect the development of these complex skills (e.g., sleep quality, stress, social relations, 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 are able to develop stronger higher-order thinking talk 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 that 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. 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 higher-order thinking talk is related to specific types of early brain injury. 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 typically-developing children and children with early brain injury.

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Table 1

TD and BI Sample Demographics

	TD sample ($n = 64$) *M* (*SD*)	BI sample ($n = 46$) *M* (*SD*)	*t*-test of difference
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

***$p < 0.001$

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

Table 2

Hierarchical Linear Models to Predict Child Surface HOTT Utterances

	Model 1 (unconditional)	Model 2 (adding BI status)	Model 3 (adding family income)	Model 4 (adding first-born status)
Fixed Effects	β (SE_{β}) $df = 106$	β (SE_{β}) $df = 106$	β (SE_{β}) $df = 106$	β (SE_{β}) $df = 106$
Intercept, β_{00}	1.64*** (0.09)	1.77*** (0.11)	1.87*** (0.09)	1.78*** (0.14)
Growth (Age), β_{10}	0.11*** (0.006)	0.12*** (0.008)	0.12*** (0.008)	0.12*** (0.01)
Acceleration (Age^2), β_{20}	-0.004*** (0.0003)	-0.004*** (0.0003)	-0.004*** (0.0004)	-0.005*** (0.0005)
BI, β_{01}		-0.29 (0.18)	-0.56** (0.19)	-0.57** (0.19)
BI x Age, β_{11}		-0.02^ (0.01)	-0.02 (0.01)	-0.02 (0.01)
BI x Age^2, β_{21}		0.002*** (0.0005)	0.002*** (0.0005)	0.002*** (0.0005)
Income, β_{02}			0.01*** (0.003)	0.01*** (0.0003)
Income x Age, β_{12}			-0.00009 (0.0002)	-0.00009 (0.0002)

Income x Age^2, β_{22}			0.00001	-0.00001
			(0.0001)	(0.00001)
First-Born, β_{03}				0.18
				(0.16)
First-Born x Age, β_{13}				-0.0009
				(0.01)
First-Born x Age^2, β_{23}				0.0004
				(0.0005)
Random Effects	*SD*	*SD*	*SD*	*SD*
	df = 93^a	*df* = 93^a	*df* = 93^a	*df* = 93^a
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^2), 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^

Note: HOTT = higher-order thinking talk. BI = brain-injured. TD (typically-developing) children are the reference category.

[a]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.

***$p < 0.001$, **$p < 0.01$, *$p < 0.05$, ^$p < 0.10$.

Table 3

Hierarchical Linear Models to Predict Child Structure HOTT Utterances

	Model 1 (unconditional)	Model 2 (adding BI status)	Model 3 (adding family income)	Model 4 (adding first-born status)
Fixed Effects	β (SE_β)	β (SE_β)	β (SE_β)	β (SE_β)
	$df = 106$	$df = 106$	$df = 106$	$df = 106$
Intercept, β_{00}	-0.20 (0.13)	-0.10 (0.16)	0.06 (0.15)	-0.30 (0.20)
Growth (Age), β_{10}	0.14*** (0.007)	0.15*** (0.009)	0.15*** (0.009)	0.16*** (0.01)
Acceleration (Age^2), β_{20}	-0.003*** (0.0003)	-0.004*** (0.0004)	-0.004*** (0.0005)	-0.004*** (0.0006)
BI, β_{01}		-0.23 (0.26)	-0.61* (0.26)	-0.62* (0.26)
BI x Age, β_{11}		-0.03* (0.01)	-0.03* (0.02)	-0.04* (0.01)
BI x Age^2, β_{21}		0.0004 (0.0007)	0.001 (0.0007)	0.001 (0.0008)
Income, β_{02}			0.02*** (0.005)	0.02*** (0.004)
Income x Age, β_{12}			-0.00008 (0.0003)	-0.00007 (0.0003)
Income x Age^2, β_{22}			-0.00002 (0.00001)	-0.00002 (0.00001)
First-Born, β_{03}				0.64** (0.23)
First-Born x Age, β_{13}				-0.02 (0.01)

First-Born x Age^2, β_{23}				0.0001
				(0.0008)
Random Effects	*SD*	*SD*	*SD*	*SD*
	df = 93[a]	*df* = 93[a]	*df* = 93[a]	*df* = 93[a]
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^2), 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 previous model	--	19.08***	14.32**	8.47*

Note: HOTT = higher-order thinking talk. BI = brain-injured. TD (typically-developing) children are the reference category.

[a]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.

***$p < 0.001$, **$p < 0.01$, *$p < 0.05$.

Table 4

Hierarchical Linear Models to Predict Child (a) Surface and (b) Structure HOTT

Utterances as a Function of BI Children's Lesion Type

	(a) Surface HOTT		(b) Structure HOTT	
	Model 1 (Income and First-Born)	Model 2 (Adding Lesion Type)	Model 1 (Income and First-Born)	Model 2 (Adding Lesion Type)
Fixed Effects	β (SE_β)	β (SE_β)	β (SE_β)	β (SE_β)
	$df = 105$	$df = 103$	df = 105	$df = 103$
Intercept, β_{00}	1.55*** (0.12)	1.79*** (0.13)	-0.55** (0.19)	-0.28 (0.20)
Growth (Age), β_{10}	0.11*** (0.01)	0.12*** (0.01)	0.15*** (0.01)	0.16*** (0.01)
Acceleration (Age^2), β_{20}	-0.004*** (0.0005)	-0.005*** (0.0005)	-0.004*** (0.0005)	-0.004*** (0.0006)
Income, β_{01}	0.008* (0.003)	0.01*** (0.003)	0.01** (0.004)	0.02** (0.005)
Income x Age, β_{11}	-0.0002 (0.0002)	-0.00007 (0.0002)	-0.0003 (0.0003)	-0.00006 (0.0003)
Income x Age^2, β_{21}	0.000001 (0.000009)	-0.00001 (0.00001)	-0.00001 (0.00001)	-0.00002 (0.00002)
First-Born, β_{02}	0.16 (0.17)	0.17 (0.16)	0.60* (0.24)	0.62** (0.23)
First-Born x Age, β_{12}	0.0002 (0.01)	0.0006 (0.01)	-0.02 (0.01)	-0.02 (0.01)
First-Born x Age^2, β_{22}	0.0004 (0.0006)	0.0004 (0.0006)	0.0002 (0.0007)	0.0002 (0.0007)
CI, β_{03}	--	-0.86***	--	-1.00**

	--	(0.23)		(0.31)
CI x Age, β_{13}	--	-0.01 (0.02)	--	-0.03* (0.02)
CI x Age^2, β_{23}	--	0.002* (0.0007)	--	0.001 (0.0009)
PV, β_{04}	--	-0.20 (0.28)	--	-0.14 (0.32)
PV x Age, β_{14}	--	-0.03* (0.01)	--	-0.04^ (0.02)
PV x Age^2, β_{24}	--	0.002*** (0.0006)	--	0.0009 (0.001)
Random Effects	*SD* $df = 92$[a]	*SD* $df = 90$[a]	*SD* $df = 94$[b]	*SD* $df = 90$[a]
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^2), 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: HOTT = higher-order thinking talk; BI = brain-injured; CI = cerebrovascular infarct; PV = periventricular lesion. TD (typically-developing) children are the reference category.

[a]The chi-square statistics reported are based on only 95 of 108 units that had sufficient data for computation. [b]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.

***$p < 0.001$, **$p < 0.01$, *$p < 0.05$, ^$p < 0.10$.

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
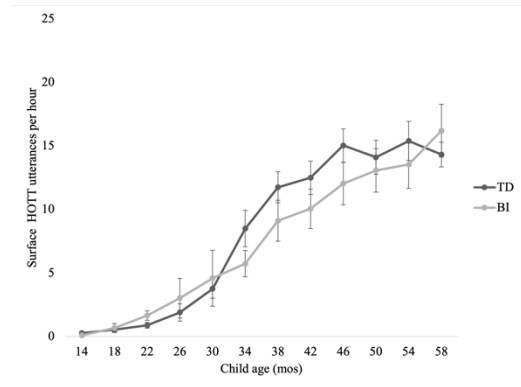
	(a) Surface HOTT	(b) Structure HOTT
	β (SE_β)	β (SE_β)
	$df = 42$	$df = 42$
Intercept, β_{00}	1.85***	0.20
	(0.24)	(0.28)
Growth (Age), β_{10}	0.09***	0.11***
	(0.01)	(0.02)
Acceleration. (Age^2), β_{20}	-0.002***	-0.003*
	(0.0004)	(0.0008)
CI, β_{01}	-0.65*	-0.88*
	(0.30)	(0.38)
CI x Age, β_{11}	0.02	0.01
	(0.02)	(0.02)
CI x Age^2, β_{21}	-0.0008	0.0002
	(0.0007)	(0.001)

Note: BI = brain-injured. CI = cerebrovascular infarct; children with PV (periventricular
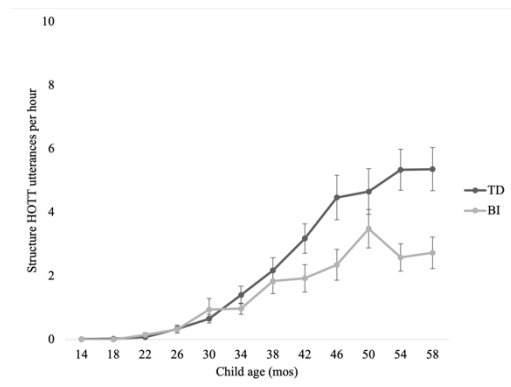
lesion) are the reference category.

Figure 1

Mean Number of Different Types of Utterances Produced by TD and BI Children
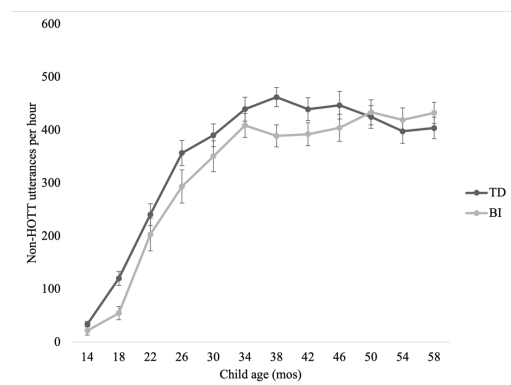
(a) Surface HOTT Utterances

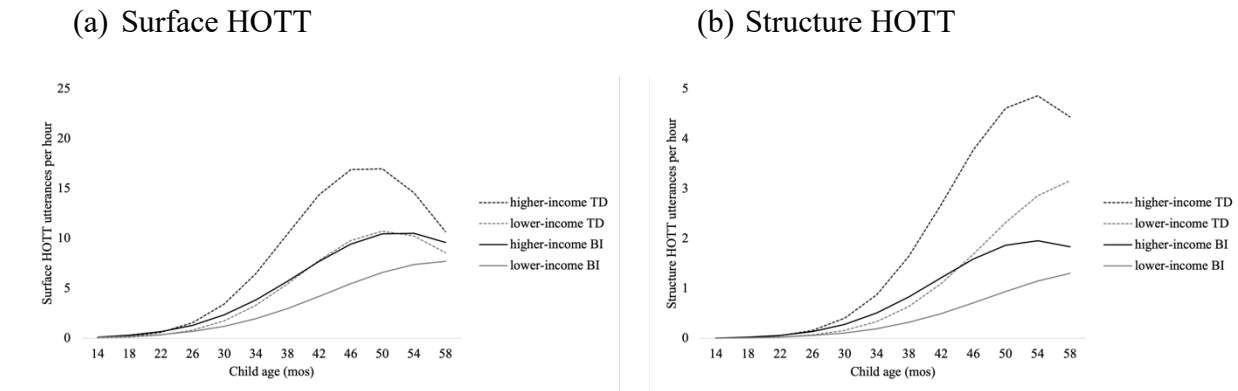(b) Structure HOTT Utterances

(c) Non-HOTT Utterances

Note: HOTT = higher-order thinking talk. TD = typically-developing; BI = brain-injured. Scales differ in each figure. Error bars ±1 SE.

Figure 2

Model Graphs of Trajectories of Surface and Structure HOTT as a Function of Family

Income and Brain-Injured Status

(a) Surface HOTT   (b) Structure HOTT

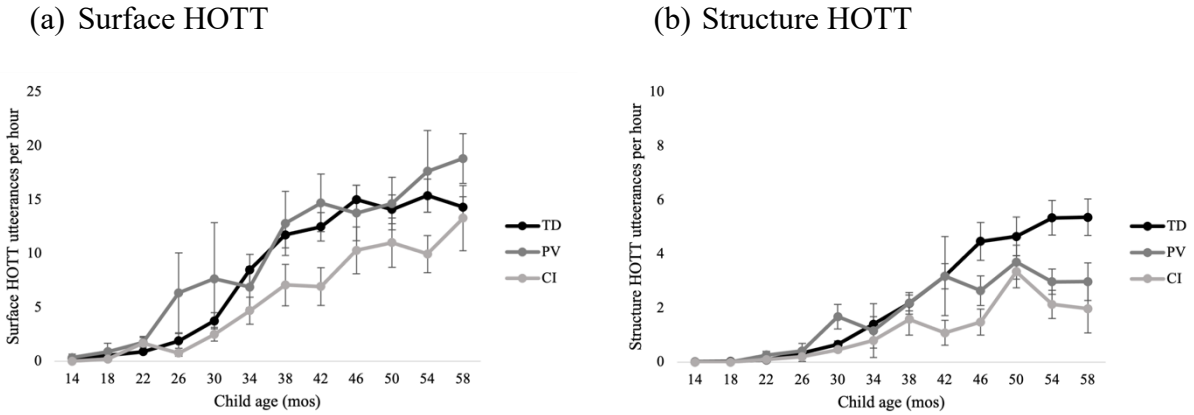*Note*: HOTT = higher-order thinking talk; TD = typically-developing; BI = brain-injured.

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.

Figure 3

Mean Number of Surface and Structure HOTT Utterances Produced by TD and BI

Children as a Function of Lesion Type

(a) Surface HOTT   (b) Structure HOTT

Note: HOTT = higher-order thinking talk; TD = typically-developing; BI = brain-injured;

PV = periventricular lesion; CI = cerebrovascular infarct. Scales differ for surface and

structure HOTT. Error bars ±1 SE.

Figure 4

Model Graphs of Trajectories of Surface and Structure HOTT as a Function of BI

Children's Lesion Type

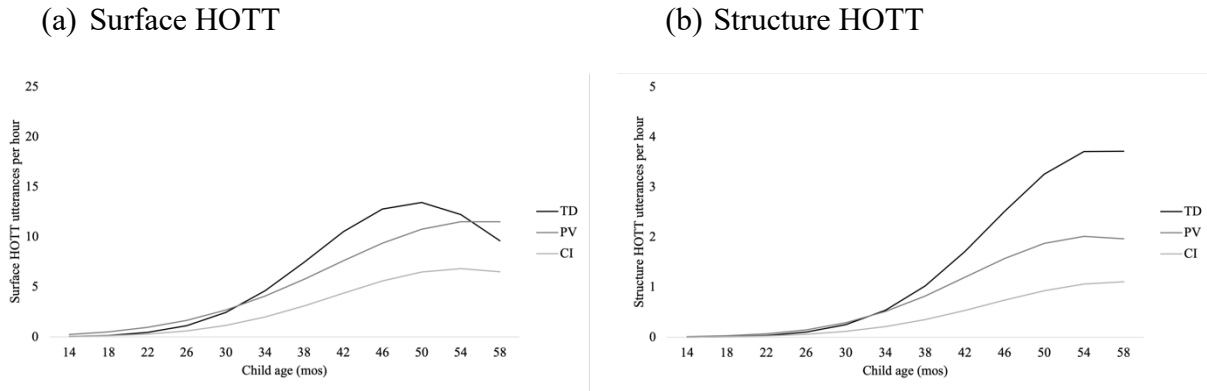(a) Surface HOTT   (b) Structure HOTT

Note: HOTT = higher-order thinking talk; TD = typically-developing; BI = brain-injured;

PV = periventricular lesion; CI = cerebrovascular infarct. Scales differ for surface and

structure HOTT.

Supplementary Materials

Table S1 summarizes the BI participant's lesion characteristics, Table S2 presents the number of TD and BI children with each session, and Table S3 provides examples of HOTT utterances produced by TD and BI children.

As with type (CI or PV), size (small, medium, or large) and laterality (left or right) were coded by two pediatric neurologists. Children with small and medium lesions were categorized into a single group, as previous analyses indicate that the two groups did not differ from each other on various language measures (Demir, Levine, & Goldin-Meadow, 2010; Demir, et al., 2015; Levine, et al., 2015; Rowe, et al., 2009).

The lesion characteristics are not independent. Lesion size and type are related ($\chi^2 = 6.20$, $p = 0.01$), such that PV tend to be small/medium lesions, whereas CI tend to vary in size more from small/medium to large lesions. Type was marginally but not strongly related to laterality ($\chi^2 = 2.56$, $p = .11$), such that CI lesions were more likely to be on the left hemisphere. However, lesion size was not related to laterality ($\chi^2 = .68$, $p = .41$).

Given the small number of children with BI, we analyzed each characteristic (type, size, laterality) independently, rather than including multiple characteristics in the same model. We report analyses in the main text by lesion type; analyses of size and laterality are presented in the following Supplementary Materials. A brief summary of the size and laterality analyses follows.

Unlike with type, we did not find strong evidence of heterogeneity within the BI sample as a function of lesion size and laterality. Children with small/medium and large lesions use less surface and structure HOTT than TD children (see Figure S1, top row, and Table S4). Additionally, children with small/medium lesions do not significantly

differ from children with large lesions in surface or structure HOTT use (see Table S6, two left columns). Likewise, both children with lesions to their left and right hemisphere use less surface and structure HOTT than TD children (see Figure S1, bottom row, and Table S5), and children with left and right lesions do not differ significantly from each other (see Table S6, two right columns).

However, it is relevant to note that when using TD children as the baseline, the interaction between the structure HOTT intercept and the dummy for small/medium lesions (β_{03} in right columns of Table S4), as well as the interaction between the structure HOTT intercept and the dummy for left hemisphere lesions (β_{03} in right columns of Table S5), do not reach statistical significance at $p < 0.10$. This potentially suggests the *amount* of structure HOTT utterances produced at 36 months by these two groups of children (small/medium lesions; left hemisphere lesions) is not reliably different from the amount of structure HOTT utterances produced by TD children. Regardless, the coefficient values (-0.48 for small/medium lesions and -0.49 for left hemisphere lesions) are relatively large, and these children *did* statistically differ from TD children in amount of surface HOTT utterances (see β_{03} in left columns of Tables S4 and S5). It would be surprising if these children differed in surface, but not structure HOTT. While difficult to interpret null findings, it is possible these null effects result from the small number of children in each size and laterality category; this study and our methods may be too underpowered to capture these differences.

Table S1

Lesion Characteristics of BI Children

		Lesion size			Total
		Small	Medium	Large	
Lesion type	Cerebrovascular infarct (CI)	5 (3 L, 2 R)	7 (5 L, 2 R)	15 (12 L, 3 R)	27 (20 L, 7 R)
	Periventricular lesion (PV)	9 (5 L, 4 R)	5 (2 L, 2 R, 1 unknown‡)	3 (1 L, 2 R)	17 (8 L, 8 R, 1 unknown‡)
	Total	14 (8 L, 6 R)	12 (7 L, 4 R, 1 unknown‡)	18 (13 L, 5 R)	44† (28 L, 15 R, 1 unknown‡)

Note: BI = brain-injured. L = left hemisphere, R = right hemisphere.

† Lesion characteristics were entirely unavailable for two children.

‡ Lesion laterality was unavailable for one child, whose size and type was known.

Table S2

Number of TD and BI Subjects with Each Session

	TD families with visit (% of TD sample)	BI families with visit (% of BI sample)	BI Children Recruited
Visit 1 (14 months)	64 (100%)	15 (33%)	15
Visit 2 (18 months)	63 (98%)	21 (46%)	6
Visit 3 (22 months)	62 (97%)	27 (59%)	7
Visit 4 (26 months)	61 (95%)	32 (70%)	5
Visit 5 (30 months)	61 (95%)	36 (78%)	3
Visit 6 (34 months)	62 (97%)	38 (83%)	3
Visit 7 (38 months)	61 (95%)	41 (87%)	5
Visit 8 (42 months)	60 (94%)	44 (93%)	0
Visit 9 (46 months)	59 (92%)	42 (87%)	0
Visit 10 (50 months)	59 (92%)	43 (89%)	0
Visit 11 (54 months	56 (88%)	41 (85%)	0
Visit 12 (58 months)	58 (91%)	44 (93%)	2

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

Table S3

Example HOTT Utterances Produced by TD and BI Children

	Surface		Structure	
	TD	BI	TD	BI
Inference	"Put the ear so the arms fit."	"Because I'm jumping."	"She's sad because she fell on the ground."	"Why is that a bad word?"
Comparison	"It tastes like yellow."	"They're both the same."	"I would rather be in school."	"What other fruit is red?"
Abstraction	"Cats say meow."	"M for Mom."	"Ladies go to work."	"Parrots stay in cages."
Hierarchy	"Spotty dog."	"A white Prius."	"What kind of cake you like?"	"I don't like that kind of preview."

Note: HOTT = higher-order thinking talk; TD = typically-developing; BI = brain-injured.

Table S4

Hierarchical Linear Models to Predict Child Surface and Structure HOTT Utterances as

a Function of BI Children's Lesion Size

	(a) Surface HOTT		(b) Structure HOTT	
	Model 1 (Income and First-Born)	Model 2 (Adding Lesion Size)	Model 1 (Income and First-Born)	Model 2 (Adding Lesion Size)
Fixed Effects	β (SE_β) $df = 105$	β (SE_β) $df = 103$	β (SE_β) $df = 105$	β (SE_β) $df = 103$
Intercept, β_{00}	1.55*** (0.12)	1.79*** (0.15)	-0.55** (0.19)	-0.28 (0.21)
Growth (Age), β_{10}	0.11*** (0.01)	0.12*** (0.01)	0.15*** (0.01)	0.16*** (0.01)
Acceleration (Age^2), β_{20}	-0.004*** (0.0005)	-0.005*** (0.0005)	-0.004*** (0.0005)	-0.004*** (0.0006)
Income, β_{01}	0.008* (0.003)	0.01*** (0.004)	0.01** (0.004)	0.02*** (0.004)
Income x Age, β_{11}	-0.0002 (0.0002)	-0.00008 (0.0002)	-0.0003 (0.0003)	-0.00007 (0.0003)
Income x Age^2, β_{21}	-0.000001 (0.000001)	-0.00001 (0.00001)	-0.00001 (0.00001)	-0.00002 (0.00002)
First-Born, β_{02}	0.16 (0.17)	0.15 (0.17)	0.60* (0.24)	0.59* (0.24)
First-Born x Age, β_{12}	0.002 (0.01)	0.0001 (0.01)	-0.02 (0.01)	-0.02 (0.01)
First-Born x Age^2, β_{22}	0.0004 (0.0006)	0.0004 (0.0006)	0.0002 (0.0007)	0.0002 (0.0007)
Small/Medium, β_{03}		-0.49* (0.23)		-0.48 (0.32)

Small/Medium x Age, β_{13}		-0.02 (0.02)		-0.04* (0.02)
Small/Medium x Age^2, β_{23}		0.002* (0.0007)		0.001* (0.0008)
Large, β_{04}		-0.72** (0.24)		-0.84* (0.34)
Large x Age, β_{14}		-0.02 (0.02)		-0.04^ (0.02)
Large x Age^2, β_{24}		0.002* (0.0008)		0.001 (0.001)
Random Effects	*SD* $df = 92$[a]	*SD* $df = 90$[a]	*SD* $df = 94$[b]	*SD* $df = 90$[a]
Intercept, r_0	0.88***	0.83***	1.12***	1.07***
Growth (Age), r_1	0.06***	0.06***	0.05***	0.05***
Acceleration (Age^2), r_2	0.003***	0.002***	0.002***	0.002***
Goodness of Fit	21,496.97 (15)	21,476.90 (21)	9,049.12 (15)	9,019.68 (21)
$\Delta\chi^2$ from model 1	--	20.07**	--	29.44***

Note: HOTT = higher-order thinking talk; BI = brain-injured; TD (typically-developing) children are the reference category.

[a]The chi-square statistics reported are based on only 95 of 108 units that had sufficient data for computation. [b]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.

***$p < 0.001$, **$p < 0.01$, *$p < 0.05$, ^$p < 0.10$.

Table S5

Hierarchical Linear Models to Predict Child Surface and Structure HOTT Utterances as a Function of BI Children's Lesion Laterality

	(a) Surface HOTT		(b) Structure HOTT	
	Model 1 (Income and First-Born)	Model 2 (Adding Laterality)	Model 1 (Income and First-Born)	Model 2 (Adding Laterality)
Fixed Effects	β (SE_β)	β (SE_β)	β (SE_β)	β (SE_β)
	$df = 104$	$df = 102$	df = 104	$df = 102$
Intercept, β_{00}	1.55*** (0.12)	1.78*** (0.14)	-0.55** (0.19)	-0.29 (0.21)
Growth (Age), β_{10}	0.11*** (0.01)	0.12*** (0.01)	0.15*** (0.01)	0.16*** (0.01)
Acceleration (Age^2), β_{20}	-0.004*** (0.0005)	-0.005*** (0.0005)	-0.004*** (0.0005)	-0.004*** (0.0006)
Income, β_{01}	0.008* (0.003)	0.01*** (0.003)	0.01** (0.004)	0.02*** (0.004)
Income x Age, β_{11}	-0.0002 (0.0002)	-0.00007 (0.0002)	-0.0003 (0.0003)	-0.00009 (0.0003)
Income x Age^2, β_{21}	-0.000002 (0.00001)	-0.00001 (0.00001)	-0.00001 (0.00001)	-0.00002 (0.00002)
First-Born, β_{02}	0.15 (0.17)	0.17 (0.17)	0.60* (0.24)	0.63** (0.23)
First-Born x Age, β_{12}	-0.00002 (0.01)	0.0004 (0.01)	-0.02 (0.01)	-0.02 (0.01)
First-Born x Age^2, β_{22}	0.0004 (0.0006)	0.0003 (0.0006)	0.0002 (0.0007)	0.0003 (0.0007)
Left, β_{03}		-0.52*		-0.49

		(0.25)		(0.30)
Left x Age, β_{13}		-0.02		-0.05**
		(0.02)		(0.02)
Left x Age^2, β_{23}		0.002*		0.001
		(0.0007)		(0.0009)
Right, β_{04}		-0.78**		-0.95*
		(0.26)		(0.38)
Right x Age, β_{14}		-0.03*		-0.03
		(0.02)		(0.02)
Right x Age^2, β_{24}		0.002***		0.001
		(0.0007)		(0.001)
Random Effects	*SD*	*SD*	*SD*	*SD*
	$df = 91$[a]	$df = 89$[a]	$df = 91$[a]	$df = 89$[a]
Intercept, r_0	0.89***	0.84***	1.13***	1.08***
Growth (Age), r_1	0.06***	0.06***	0.05***	0.04***
Acceleration (Age^2), r_2	0.003***	0.002***	0.002***	0.002***
Goodness of Fit	21,496.97 (15)	21,159.77 (21)	8,967.24 (15)	8,926.24 (21)
$\Delta\chi^2$ from model 1	--	20.69***	--	31.00***

Notes: HOTT = higher-order thinking talk; BI = brain-injured. TD (typically-developing) children are the reference category. Model 1 differs from Model 1 in Tables 4 and S4 because one BI child (whose laterality is unknown) is removed from analyses.

[a]The chi-square statistics reported are based on only 94 of 107 units that had sufficient data for computation.

***$p < 0.001$, **$p < 0.01$, *$p < 0.05$, ^$p < 0.10$.

Table S6

Fixed Effects from Hierarchical Linear Models within BI Sample to Predict Child (a)

Surface and (b) Structure HOTT Utterances as a Function of Lesion (1) Size and (2)

Laterality

	(1) Size			(2) Laterality	
	(a) Surface	(b) Structure		(a) Surface	(b) Structure
	β (SE_β)	β (SE_β)		β (SE_β)	β (SE_β)
	$df = 42$	$df = 42$		$df = 41$	$df = 41$
Intercept, β_{00}	1.65***	-0.01	Intercept, β_{00}	1.29***	-0.61^
	(0.20)	(0.26)		(0.22)	(0.34)
Growth (Age), β_{10}	0.10***	0.11***	Growth (Age), β_{10}	0.09***	0.12***
	(0.01)	(0.01)		(0.01)	(0.02)
Acceleration (Age^2), β_{20}	-0.003***	-0.003**	Acceleration (Age^2), β_{20}	-0.002**	-0.003*
	(0.0004)	(0.0006)		(0.0007)	(0.0009)
Large, β_{01}	-0.47	-0.74^	Left, β_{01}	0.22	0.40
	(0.30)	(0.38)		(0.30)	(0.42)
Large x Age, β_{11}	0.0008	0.006	Left x Age, β_{11}	0.01	-0.02
	(0.02)	(0.02)		(0.02)	(0.03)
Large x Age^2, β_{21}	0.0001	0.0002	Left x Age^2, β_{21}	-0.002	-0.0004
	(0.0007)	(0.001)		(0.0005)	(0.001)

Notes: HOTT = higher-order thinking talk. BI = brain-injured. In (1), children with

small/medium lesions are the reference category; in (2), children with right hemisphere

lesions are the reference category. ***$p < 0.001$, **$p < 0.01$, *$p < 0.05$.

Figure S1.

Mean Number of (a) Surface and (b) Structure HOTT Utterances Produced as a Function

of BI Children's Lesion Size (row 1) and Laterality (row 2).

(a) Surface HOTT   (b) Structure HOTT
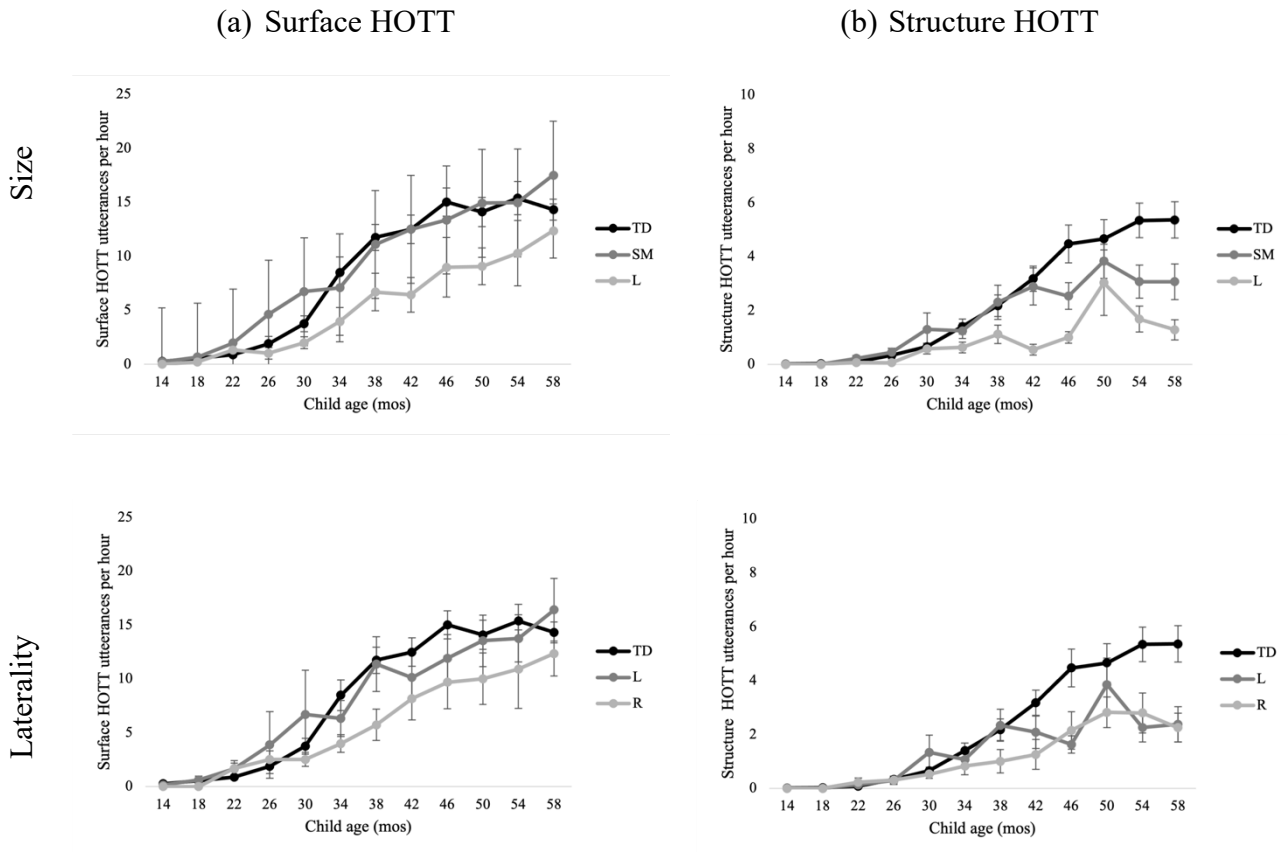
Notes: HOTT = higher-order thinking talk; BI = brain-injured; TD = typically-

developing. In row 1, SM = small or medium lesions, L = large lesions; in row 2, L = left

hemisphere, R = right hemisphere. The scales differ for surface and structure HOTT.

Error bars ±1 SE.