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Hearing me hearing you: Reciprocal effects between child and parent language in autism and typical development

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Abstract

Language development in typically developing children (TD) has traditionally been investigated in relation to environmental factors, while language in children with autism spectrum disorder (ASD) has primarily been related to child-based factors. We employ a longitudinal corpus of 32 preschoolers with ASD and 35 linguistically matched TD peers recorded over 6 visits (ranging between 2 and 5 years of age) to investigate the relative importance of child-based and environmental factors in language development for both populations. We also investigate the reciprocal interaction between children's response to parents' input, and parents' response to children's production. We report six major findings. (1) Children's production of word types, tokens, and MLU increased across visits, and were predicted by their Expressive Language (EL) (positively) and diagnosis (negatively) from Visit 1. (2) Parents' production also increased across visits, and was predicted by their child's nonverbal cognition (positively) and diagnosis (negatively) from Visit 1. (3) At all visits and across groups, children and parents matched each other in lexical and syntactic production; (4) Parents who produced longer MLUs during a given visit had children who produced more word types and tokens, and had longer MLUs, at the subsequent visit. (5) When both child EL at Visit 1 and parent MLU were included in the model, both contributed significantly to future child language; however, EL accounted for a greater proportion of the variance. (6) Finally, children's speech significantly predicted parent speech at the next visit. Taken together, these results draw more attention to the importance of child-based factors in the early language development of TD children, and to the importance of parental language factors in the early language development of children with ASD.

Keywords

language development; autism spectrum disorder; child-based factors; environmental factors; child directed speech

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1. Introduction

Successful language acquisition in childhood is a function of environmental factors and child-based factors, including biological components (Gleitman, 1984; L. R. Naigles & Bavin, 2015). Although one of the most enduring goals of research in this field is to discern the relative contributions of each (Gleitman & Wanner, 1982; Slobin, 1985), the focus of research has been largely tied to the population under study. Variability in language development in typically developing (TD) populations has been primarily investigated from the 'environmental factors' perspective, consistently finding that children who receive greater amounts of input, more responsive social interactions with caregivers, and more diverse input, demonstrate earlier and/or more complex language use (Gathercole & Hoff, 2007; Rowe, 2012; Rowe & Goldin-Meadow, 2009; Tamis-LeMonda, Kuchirko, & Song, 2014). Studies of child-based variability in TD children have been limited, with twin studies being the major exceptions (Colledge et al., 2002; Reznick, Corley, Robinson, & Matheny Jr, 1997). On the other hand, research on the variability of language development in children with autism spectrum disorders (ASD) has emphasized child-based factors such as verbal and nonverbal intelligence and autism severity, which account for significant variance in these children's language outcomes (Anderson et al., 2007; Bang & Nadig, 2015; Bopp, Mirenda, & Zumbo, 2009; Ellis Weismer & Kover, 2015; Howlin, Savage, Moss, Tempier, & Rutter, 2014; Kenworthy et al., 2012; Magiati, Tay, & Howlin, 2014; Munson et al., 2008; Paul, Chawarska, Cicchetti, & Volkmar, 2008; Szatmari et al., 2009; Venker et al., 2015; Wodka, Mathy, & Kalb, 2013), while investigations of the structure and content of parental input are only just beginning (Bang & Nadig, 2015; Nadig & Bang, 2017; Venker et al., 2015).

In the current paper, we combine these approaches in a longitudinal comparison of the language trajectories of initially language-matched TD children and children with ASD. We assess the influence of the children's early cognitive abilities, their autism symptomatology, *and* their parents' language production, on these children's language-learning trajectories. Moreover, we investigate an aspect of language-learning that has been often overlooked: the influence of children's linguistic productions, cognitive ability, and (when relevant) autism severity on their parents' speech; that is, how children affect their own language-learning environment (Sameroff, 2010; Warlaumont, Richards, Gilkerson, & Oller, 2014). Note that while we are aware of the importance of timing and responsiveness in parent-child interactions (Warlaumont et al., 2014; Weed, Fusaroli, Tranbjerg, Fein, & Naigles, 2017), in the current work we focus on verbal aspects only (i.e., utterance length, unique words and total words).

1.1. Variability in typical language development

How variable is language development in TD children? Broadly speaking, the overall developmental trajectory of TD children seems consistent across many investigations, and these children tend to reach their language-related milestones within the span of a few months. For example, they demonstrate sensitive auditory processing of speech at or before birth and linguistic processing by 6 months of age; they produce their first words around 12 months and show dramatic increases in vocabulary use at 18–24 months; at the same age,

they begin to produce word or morpheme combinations, and increase steadily in mean length of utterances (MLU) produced from two to four years, adding morphemes referring to tense and aspect, plurality and modality, elaborating relative clauses and sentence complements (see Hoff, 2013, and references therein).

However, each of these broad general stages manifests large individual differences in age of attainment, as well as efficiency and frequency of use (Fernald, Marchman, & Weisleder, 2013; Hoff-Ginsberg, 1985). These individual differences in child behavior are often correlated with consistent variability in numerous aspects of the children's environments (Hoff, 2006; Huttenlocher, Vasilyeva, Waterfall, Vevea, & Hedges, 2007), leading many researchers to attribute the former to the latter. For example, children who hear more word tokens subsequently produce more words themselves (Hart & Risley, 1995; Huttenlocher, Haight, Bryk, Seltzer, & Lyons, 1991; Mahr & Edwards; L. R. Naigles & Hoff-Ginsberg, 1998); moreover, children who hear a more *diverse* lexicon, including more word types, more rare words, and higher type-token ratios, also subsequently produce a greater diversity of word types and score higher on standardized tests of vocabulary (Barnes, Gutfreund, Satterly, & Wells, 1983; Hoff, 2003; Huttenlocher, Waterfall, Vasilyeva, Vevea, & Hedges, 2010; Jones & Rowland, 2017; Pan, Rowe, Singer, & Snow, 2005; Rowe, 2012; Rowe & Goldin-Meadow, 2009; Song, Spier, & Tamis-Lemonda, 2014)). Finally, the syntactic complexity and diversity of parental input has also been found to positively impact children's lexical and syntactic development (Gleitman, Newport, & Gleitman, 1984; Hoff, 2003; Hoff & Naigles, 2002; Hoff-Ginsberg, 1985; Huttenlocher et al., 2010; L. R. Naigles & Hoff-Ginsberg, 1998; Rowe & Goldin-Meadow, 2009; Szagun & Stumper, 2012).

Far less research has investigated the role of more biologically-based factors on variation in TD children's language development. The most prominent examples are those comparing monozygotic (MZ) and dizygotic (DZ) twins, where language outcomes that are more similar in the MZ than DZ twins are attributed to genetic influences. Indeed, twin studies have revealed that aspects of two-year-olds' word comprehension and production (Price et al., 2000; Reznick et al., 1997) have genetic bases that seem to be independent of nonverbal cognition. Composite measures of four-year-olds' language also demonstrate moderate genetic influences (Kovas et al., 2005); by this age, though, the degree to which these are independent of the (also) genetically-based influences of nonverbal cognition is unclear (Colledge et al., 2002). Intriguingly, a recent study has suggested that correlations between self-reported variation in parents' linguistic production and parent-reported variation in their children's language are not necessarily due to just environmental influence on the child development but also to shared genes, that is, to shared innate predispositions (P. S. Dale, Tosto, Hayiou-Thomas, & Plomin, 2015). The contribution of genetic factors to TD twins' early language variability seems clear, although the association of biological factors to specific aspects of TD language is— understandably, given the complexity of the gene-brain-behavior mapping—much less well-elaborated than the associations between environmental factors and children's language (but see Canfield, Edelson, & Saudino, 2017).

In sum, while TD children progress in broad strokes through the same macroscopic stages at a roughly comparable rate, variability among these children exists, and evidence suggests

that both environmental (linguistic input) and child-based factors can explain this variance. Further, there is some indication that these factors may interact with one another.

1.2. Explaining variability in the language of children with ASD

While research on TD children's language has focused primarily on environmental factors, research on children with ASD has focused on the more child-based aspects of the autism spectrum disorder itself on language development.

Researchers have long struggled to capture the distinctive quality and diversity of language in people with ASD (Kanner, 1943; Lord, Risi, & Pickles, 2004), and have distinguished anywhere from two to seven distinct language phenotypes. For instance, Tek and colleagues defined two groups of participants with ASD along a median split of their verbal skills, and found that the higher performing children were not significantly different from the TD participants (Tek, Mesite, Fein, & Naigles, 2014). Anderson and colleagues defined four groups based on their performance at age 9: children fluently using complex sentences; those using some complex sentences, but not fluently; children only using words in isolation; and those minimally verbal; with similar representation of children with ASD in these groups (Anderson et al., 2007). Wittke and colleagues divided their 5-year-old participants into three groups according to the sheer amount of their verbal productions and the number of grammatical errors they committed, yielding subgroups of language impaired, grammatically impaired, and normal language children (Wittke, Mastergeorge, Ozonoff, Rogers, & Naigles, 2017). Taking a different approach, Pickles and colleagues used latent growth curve analysis on parental reports to infer from the data seven latent classes of linguistic development in participants with ASD between the age of two and six: from nearly typical to 'catch-up' to different kinds of delay. Development between the ages of six and nineteen years showed remarkably similar patterns across the classes (Pickles, Anderson, & Lord, 2014). While subgrouping can be important for decisions about interventions, it is not yet clear to what extent the subgroups thus far proposed have independent validity; moreover, it is becoming increasingly recognized that ASD symptomatology—and language impairments—exist on spectra or continua rather than clustering into subgroups (e.g., Archibald & Noonan, 2015; Constantino, 2011; Tomblin, 2015). In our study, we attempt to more fully assess the heterogeneity of the participants by modelling the children's actual verbal and non-verbal cognitive skills as continuous variables, rather than splitting them into groups.

Studies investigating the early predictors of later language variability in these individuals have typically used standardized tests of language and communication, rather than the more specific language samples and/or linguistic assessments often used by research in TD samples. For example, variability in expressive communication was predicted positively by children's earlier scores on the receptive communication subscale of the Vineland Adaptive Behavior Scales (VABS; Sparrow, Balla, & Cicchetti, 2005) and negatively by their stereotyped behaviors on an earlier autism diagnostic assessment (Paul et al., 2008; Szatmari et al., 2009). The dominant and independent influences of (higher) nonverbal cognition and (milder) autistic behaviors on subsequent language performance, as measured by standardized tests of structural language, have been replicated by a number of different

research groups over the past decade (Bopp et al., 2009; Ellis Weismer & Kover, 2015; Thurm, Manwaring, Swineford, & Farmer, 2015; Wodka et al., 2013; Yoder, Watson, & Lambert, 2015).

There has been much less research investigating how children with ASD might make use of the linguistic input they are provided with; however, the extant data are mostly consistent with findings from TD children. Venker et al (2015) showed that telegraphic speech (omission of e.g. determiners to produce utterances like “Mommy go” or “cup full”) in parents of toddlers with ASD was a slight negative predictor of lexical diversity in these children a year later, indicating that richer syntactic input supports language-learning in children with ASD as well in TD children. As Nadig and Bang (2017) review, parents who produce more word tokens (Warren et al., 2010), noun types (Swensen, 2007), and utterances with longer MLUs (Bang & Nadig, 2015) have children with ASD who subsequently produce more words. Parents who produce more diverse yes/no and wh-questions have children with ASD who subsequently produce and understand more complex grammar themselves (Goodwin, Fein, & Naigles, 2015; Swensen, Kelley, Fein, & Naigles, 2007). Finally, in a meta-analysis, Sandbank and Yoder (2016) report that four studies show a strong correlation between the length of parents’ utterances and positive language outcomes in children with autism (Burgess, Audet, & Harjusola-Webb, 2013; Grelle, 2013; Konstantareas, Mandel, & Homatidis, 1988; Nadig & Bang, 2017).

These positive findings are very encouraging; however, many of these studies have had limited sample sizes, and have only rarely included both TD children and children with ASD in the same model to test whether the effects of parental input hold similarly for both groups (Bang & Nadig, 2015). Moreover, there have been concerns about whether the parental inputs provided to children with ASD and those provided to TD children are comparable. These concerns arose because children with ASD are characterized by social disengagement (DSM-V, APA 2015), which affects the duration and kind of interactions they engage in with their caregivers, and so may also influence the content and structure of that input. In other words, the children’s abilities and challenges have the potential to reciprocally affect their parents’ input. Studies comparing parents of age-matched children with ASD and TD children show significant differences in the amount and complexity of parental linguistic production, probably due to the children’s language delays. However, when the children are linguistically well matched no differences in parental linguistic production have been found (Bang & Nadig, 2015). This suggests that the effects of linguistic ability and social reciprocity might be disentangled.

1.3. Reciprocal processes in language acquisition

Because adults are the experts in language use, we tend to think of the parent-child relationship as unidirectional; that is, parents speak and children learn about the local language from that speech (Gathercole & Hoff, 2007). However, a number of proposals about child development as a whole have emphasized the interactional nature of these processes, claiming that cross-generational influence is bidirectional, with children influencing parents as well as parents influencing children (Chapman, 2000; Sameroff, 2010). Direct evidence for this proposal is somewhat mixed, though. For example, early

research on child-directed speech seemed to indicate stability over time, suggesting that parents do not adapt to their children's language growth (Cohen & Beckwith, 1976; Moss, 1967; Nelson & Bonvillian, 1973; Smolak & Weinraub, 1983). However, researchers have often reported that the input to older children is more complex than that to younger children; for example, parental MLUs increase across the span from child ages 1 to 5 years (Newport, Gleitman, & Gleitman, 1977; Rowe, 2012; Saint-Georges et al., 2013; Schwab & Lew-Williams, 2016), although the sheer quantity of speech does not seem to change (Gilkerson, Richards, & Topping, 2017; Rowe, 2012). Moreover, when controlling for child age but not language level, researchers report that parents of TD children produce more complex speech than parents of children with ASD, and parents of higher functioning children with ASD produce more complex speech than parents of lower-functioning children (Nadig & Bang, 2017). When child language level is controlled, though, TD/ASD group differences in parental word tokens, types, MLU, and many aspects of question use are not significant (Bang & Nadig, 2015; Goodwin et al., 2015; Slaughter, Peterson, & Mackintosh, 2007; Swensen, 2007; Swensen et al., 2007; Wolchik & Harris, 1982), suggesting that parents may respond more to the language level of their child, rather than directly to ASD severity.

Often these comparisons, however, have been confined to single points in developmental time; thus, what is still underinvestigated is how child-based factors such as initial language level, nonverbal cognition, and autism severity might influence parental input over time. Moreover, another way to investigate how parental language might be influenced by child factors is to see whether children's measures at a given time (N) might predict parents' language at a later time (N+1), controlling for parents' language at time (N). A small number of studies have done so, with (again) mixed results: For example, Huttenlocher et al. (2010) found significant relationships between two-year-old TD children's lexical diversity and their caregivers' lexical diversity four months later; however, for measures of grammatical complexity and diversity, only caregivers' speech positively predicted their children's subsequent speech (and not vice versa). Venker et al. (2015) similarly reported that caregivers' speech positively predicted the child's future linguistic production, but not vice versa. In contrast, Song et al. (2014) found a significant relationship between TD children's nonverbal cognitive levels and their parents' lexical diversity in speech one year later. Furthermore, in an intriguing fine-grained analysis of daylong recordings in age-matched children with ASD and TD children (age: 8–48 months), Warlaumont et al. (2014) investigated the temporal contingencies of parent and child vocalizations within the same conversations. They found that child vocalizations that were speech-like were more likely to elicit immediately contingent responses from parents. This relationship was observed for both TD children and children with ASD, although it was stronger for TD children, and for older children. Because prompt parent responses increased the likelihood of the next child vocalization to be speech-like, the authors suggest that parental adaptation to the child might underlie differential developmental trajectories, a claim supported by the results of a computer simulation of longitudinal effects of this feedback loop. The results were then replicated on a second sample (Harbison et al., 2018). In the current study, we extend these findings by investigating reciprocal effects of child language on parent language using the lexical and grammatical measures of word types and tokens, and MLU, and compare these

in dyads involving TD children vs. children with ASD. Future work will further focus on the issue of temporal responsiveness (Weed et al., 2017).

1.4. Studying language development across time

One of the challenges in studying language development is that it is a moving target. Children mature at different rates, and their linguistic ability develops in fits and starts. The language profiles of some children with ASD may look radically different at different points in development, while others may show very little change (Ellis Weismer & Kover, 2015; Frith & Happé, 1994; Pickles et al., 2014). More recent research has shown that the period between 2 and 5 years of age presents the highest variability in language development, while children above age 6 have quite stable developmental trajectories (Pickles et al., 2014). A few studies also find relations between cognitive abilities and severity of clinical features at age 2 and language development (e.g. Ellis Weismer & Kover, 2015; Paul et al., 2008). It is therefore necessary to move beyond static, matched-group comparisons, and adopt analytical methods that account for individual variation in starting conditions and development among participants, as well as the relation between the two (Hayiou-Thomas et al., 2006; Jarrold & Brock, 2004). Multi-level modelling techniques offer tools to do this, in a fashion that is robust to missing and unbalanced data (Gelman & Hill, 2007; McElreath, 2015). Although these techniques have not yet been widely applied in the field of child language development, with the notable exception of Rowe et al (2012), they are becoming common in other fields such as education (Kiwanuka et al., 2017; Oldfield, Humphrey, & Hebron, 2017) and cognitive science (Mirman, 2016).

1.5. The current study

The first two major goals of the current study were to investigate and compare the roles of child-based factors such as nonverbal IQ and autistic symptomatology, and concurrent environmental factors such as parent word use and MLU, on the language growth of TD children and children with ASD who were not different in language level at study onset. The third major goal was to investigate the question of reciprocal influence; that is, how the children's language use, clinical and cognitive characteristics might influence parental language concurrently and longitudinally. The literature reviewed above demonstrates that parents adapt to their children's language. Therefore, to adequately investigate the role of cognitive and clinical features of children with ASD and TD children in the parent-child reciprocal influence, language abilities should be controlled for. We accordingly chose to rely on actual language production in a naturalistic context: a longitudinal corpus of free-play interactions between children with ASD and their parents, and an initially language-matched group of TD children between the ages of 2 and 5. Language production in both children and parents was characterized in terms of number of word tokens, word types, and MLU. Note that a subset of the children (17 ASD out of 32 and 18 TD out of 35) were included in a previous study (Tek et al., 2014). In addition to employing a larger sample of children and in accordance with the points highlighted in the previous paragraphs, the current analysis differs from the previous study in modelling the full range of linguistic, cognitive and clinical features instead of focusing on low vs. high verbal skill. This allows us to better account for individual differences in language development.

In particular, we addressed six research questions.

1. *Longitudinal trajectories of language development – child-based factors:* We first characterized the children's developmental trajectories on our three language measures across a 2-year span, addressing the question: To what extent is this development related to child-based factors of initial language level, nonverbal cognition, and autism symptomatology?

2. *Longitudinal trajectories of parental language production – child-based factors:* We also characterized the linguistic profiles of the parents over the same time interval and asked: To what extent are the changes observed in the parents' language use influenced by the same child factors?

3. *Parent-child matching in concurrent linguistic production:* We then analyzed *matching* within the parent-child dyads. Matching is defined as the tendency of parent-child dyads to present similar levels of linguistic performance, that is, if during a visit the parent displays higher than average linguistic performance, we can expect a similar higher than average performance in the child. We asked: To what degree do parents' and children's productions match each other within the context of a single conversation, and how is this matching influenced by the same child factors?

4. *Predicting child linguistic development from environmental factors:* We next investigated the role of cross-lagged environmental factors in children's language development, asking to what degree do current measures of parental speech predict the measures of children's speech during the next visit 4 months later?

5. *Assessing the relative roles of child-based and environmental factors in child linguistic development:* >We compared the relative variance of child-based and environmental factors, asking: when both child-based and environmental factors are included in the same model, how much variance in child language outcomes is accounted for by each?

6. *Predicting parental linguistic production from child linguistic production:* Finally, to address the question of reciprocal influence, we asked to what degree do the measures of child speech predict parents' speech at a later point?

2. Methods

2.1. Participants

As part of an ongoing longitudinal study investigating language acquisition in young children with ASD (see L. R. Naigles & Fein, 2017, for an overview), we recruited 32 children diagnosed with ASD (mean age at recruitment = 32.76 months, 95% CI: 30.65 34.4) and 35 TD children (mean age at recruitment = 20.27 months, 95% CI: 19.78 20.93). All children were monolingual English learners. There were four girls in the ASD group and six in the TD group. Children in the ASD group had been previously diagnosed with Autistic Disorder or Pervasive Developmental Disorder-Not Otherwise Specified (PDD- NOS) by physicians or psychologists, and their diagnosis was confirmed with the Autism Diagnostic

Observation Schedule – Module 1 (ADOS, Lord, 2008) before the start of the study. Expressive language at visit 1, measured by the raw scores of the Expressive Language Scale of Mullen Scales of Early Learning (MSEL, see Table 1), was used to balance the composition of TD and ASD groups. Descriptive statistics for the sample can be found in Table 1. All children with ASD were enrolled in interventions that provided at least 20 hours/week of ABA (Applied Behavior Analysis; Lovaas, 1987).

The ASD group was recruited through treatment facilities and schools in the Northeastern U.S. The children in the TD group were recruited from a database of children in the UConn Child Language Lab. Children in the TD group were administered the ADOS – Module 1, and none had elevated total scores (see Table 1). The TD and ASD groups were composed to have similar levels of expressive language at visit 1, which was measured by the raw scores of the Expressive Language Scale of Mullen Scales of Early Learning (MSEL) and then corroborated by assessing actual level of linguistic production during the visit (see Table 1). The MSEL gives raw scores, standard T scores (average standard score is 50 with a standard deviation of 10), and age equivalents for each domain of the test. However, we did not use T scores of the MSEL Expressive Language Scale to compose the groups, as they were not matched on chronological age (Mervis & Klein-Tasman, 2004). We are aware of the limitations of using p-values to assess matching between groups: p-values are designed to control for the long-term rate of Type I error (incorrectly concluding that there is difference when there is no difference) and therefore – while commonly used – do not adequately assess the validity of the null hypothesis of no difference (which can only be rejected and never supported) (Kover & Atwood, 2013). While other approaches relying on effect sizes and confidence intervals have been suggested, we opted for a Bayesian analysis of the group difference in MSEL EL to directly estimate the evidence in favor of the null hypothesis (Ly, Verhagen, & Wagenmakers, 2016). Bayes factors (BF) express the relative evidence for the null hypothesis compared to that for the alternative hypothesis, given the observed data. If one hypothesis is more likely than the other given the data, there is evidence for that hypothesis over the other. We used a relatively agnostic prior for the difference: a normal distribution centered at 0, with a standard deviation of 10. The probable difference between the groups (posterior estimate) is an approximately normal distribution, centered at −2.26, with a standard deviation of 1.51, 95% CI −5.16, 0.69. The Bayes Factor indicates that after considering the data, there is 2.3 times more evidence for the null hypothesis (difference approximately equal to 0) than for the alternative hypothesis. In sum, while we cannot reject the null hypothesis ($p>0.05$), a BF analysis indicates some evidence for the null, and measures of actual linguistic production all indicate a pragmatically adequate matching. Further, by including MSEL EL in the actual statistical models, effects are calculated while accounting for this variable at the same time, thus providing a further statistical control of the individual and group variability in verbal IQ.

The ASD group included five children whose data at one visit were missing (one at visit 3, two at visit 4, two at visit 5 and one at visit 6, due to equipment failures during recording or digitizing). The TD group included one child whose data at one visit were missing (at visit 6, due to equipment failures during recording or digitizing). This generated a total of 395 visits (186 for the ASD and 209 for the TD group).

2.2. Procedure

The participants' data were collected across six home visits, each of which was separated by 4 months. At visit 1, children were administered the standardized measures, which included the ADOS – Module 1, and the MSEL. At all visits, children engaged in a 30-min semi-structured parent–child play session. The first 15 min of the session followed the structure of the Screening Tool for Autism in 2-year-olds (STAT; Stone, Coonrod, & Ousley, 2000), which consists of 12 play-based activities that involve the child in pretend play with dolls, interactive play with a ball or truck, imitative action play, and requests and joint attention (e.g., pointing, reaching, etc.). To ensure that the parents followed this structure, the experimenter handed the parents note cards, which stated what they should be doing with their children. During the second (free play) part of the session, the parent and the child were instructed to play "however they usually play at home." The visits were recorded and later transcribed at word-level, then analyzed via CLAN (MacWhinney & Wagner, 2010) at the level of grammatical morphemes. Transcriptions were performed by undergraduate research assistants at UConn, who were blind to diagnostic category (however, this information could be often inferred from the dynamics in the video-recorded interactions). Each interaction was transcribed by two different students; discrepancies on the level of words or grammatical morphemes were resolved via explicit discussion between the two students while viewing the video, with the last author acting as final arbiter.

2.3. Tests and measures

2.3.1. Standardized Test Measures—Standardized test measures were collected to confirm the children's placement into diagnostic groups, and to provide general characteristics of their intellectual level at visit 1.

The Autism Diagnostic Observation Schedule (ADOS, Lord, 2008) is a structured and play-based assessment for the diagnosis of ASD. It consists of a series of activities designed to interest young children and encourage them to communicate, and systematic probes are used to sample children's behavior in social interaction, communication, stereotypical behavior and repetitive interests. Scores greater than 7 place children on the autism spectrum, and scores greater than 12 indicate autistic disorder. Module 1 was administered at visit 1 because pre-visit conversation with parents indicated that none of the children were producing phrase speech; moreover, using the same module for all children facilitated comparisons across children.

The Mullen Scales of Early Learning (MSEL; Mullen, 1995) is a measure of intellectual development, which measures visual reception (nonverbal cognition), expressive language, receptive language, and motor development for children from birth to 5 years, 8 months. We focus on the expressive language (EL) and visual reception (VR) measures, employing raw scores, as age was not matched across participants. Although one common practice is to average Fine Motor and Visual Reception scores to estimate nonverbal IQ (Bishop, Guthrie, Coffing, & Lord, 2011), many children with ASD have fine as well as gross motor impairments; this is especially true in very young children (Ming, Brimacombe, & Wagner, 2007). For these children, VR by itself is a more accurate estimate of nonverbal reasoning ability. It should be noted that the ADOS, EL and VR are correlated. VR and EL are

positively related: $\beta = 0.88$, SE = 0.13, $p < 0.0001$, $R^2 = 0.39$. VR and ADOS are negatively related: $\beta = -0.93$, SE = 0.19, $p < 0.0001$, $R^2 = 0.44$. EL and ADOS are negatively related: $\beta = -1.26$, SE = 0.25, $p < 0.0001$, $R^2 = 0.46$. As a consequence, when two or more of these measures are jointly used, their statistical estimates and the choice of one measure over the other should be interpreted with caution.

2.3.2. Measures of Language Production—Measures of parents' and children's language were extracted using custom Python scripts from transcripts of spontaneous speech produced during parent–child play sessions, and included amount of speech (overall number of words uttered, or word tokens), vocabulary diversity (number of unique words used, or word types) and sentence complexity (Mean Length of Utterance, or MLU). MLU is commonly employed as a measure of linguistic complexity as it involves both free (words) and bound (inflections) morphemes in each utterance (Brown, 1973; Tager-Flusberg et al., 2009; Tek et al., 2014). MLU is calculated by dividing the total number of morphemes by the number of utterances in each speech sample. Note that we include all child-directed parental speech, including what would often be categorized as first initiation and as responsive interaction. Our naturalistic interactions present sequences quite extended in time with complex interdependencies, and we believe assessments of child-parent reciprocity should include all utterances. Further analyses of the interactions amongst speech input and joint attention episodes are only just beginning (for an example, see Abdel-Aziz, Kover, Wagner, & Naigles, in press).

2.4. Analyses

Six analyses were applied to each of the extracted linguistic features (word tokens, word types and MLU), following on the six research questions delineated in the introduction (paragraph 1.5 – The current study). These analyses modelled:

1. The longitudinal development of the children's expressive language as a function of time and child-based factors at first visit.
2. The longitudinal development of the linguistic environment (parental production) as a function of time and child-based factors at first visit
3. The level of within-conversation linguistic matching in parent-child dyads
4. The predictive power of parental production (environmental factors) on children's future linguistic performance.
5. The relative predictive power of child based factors (initial verbal and nonverbal cognition, as well as diagnosis), environmental factors (parental linguistic production), on the children's subsequent verbal performance.
6. The predictive power of children's production on parents' future linguistic performance.

2.4.1. Modelling clinical and cognitive factors as predictors of children's longitudinal linguistic trajectories

In order to assess the factors involved in the children's linguistic developmental trajectories we employed mixed effects growth curves. Growth curve models are a specific case of multi-level modelling, which accounts for intercepts and slopes at both individual and group levels. Multi-level models are particularly useful in handling missing data and the comparison of multiple predictors (Gelman & Hill, 2007; McElreath, 2015; Mirman, 2016). Mixed effects growth curve models specifically include non-linear components of changes in time of the dependent variable. It has been argued that linguistic development does not follow a consistent linear trajectory, with overall increases over time either punctuated with bursts of increased development or generally accelerating (cf. the debate between Ganger & Brent, 2004; and Goldfield & Reznick, 1990). For this reason, we tested linear and quadratic growth components in the present study. A quadratic component enables the model to describe general trends of acceleration and deceleration in the developmental trajectories, beyond simpler linear increase or decrease. In particular, positive linear and quadratic components indicate an increase over time that accelerates, that is, after a certain point at each visit the increase is higher than in the previous visit. Analogously, a positive linear and a negative quadratic component indicate an increase over time that slows down; that is, after a certain point at each visit the increase is lower than in the previous visit. Higher polynomial components were not considered, given the limited longitudinal density of the corpus (6 visits).

Each growth curve model included Visit (linear and quadratic), Diagnosis, Mullen Expressive Language Score at Visit 1 (EL), and Mullen Visual Reception Score at Visit 1 (VR) as fixed factors; individual participants' intercept and slope over time were included as random effects. We explicitly modelled random effects as potentially correlated, e.g. allowing for higher random intercepts, being systematically related to steeper linear and shallower quadratic components of the slope.

1. $LinguisticFeature = \beta_{0i} + \beta_{1i}Visit + \beta_{2i}Visit^2 + \beta_3 Diagnosis + \beta_4 EL + \beta_5 VR + \varepsilon$
   The subscript i indicates which components of the models include a random component by child.

Additionally, we were interested in how early individual differences (EL and VR) might interact in the linguistic development trajectories (2-way interactions) as well as whether diagnosis would affect these interactions (3-way interactions). Assessing all of these factors simultaneously would require a large model:

1. $LinguisticFeature = \beta_{0i} + \beta_{1i}Visit + \beta_{2i}Visit^2 + \beta_3 Diagnosis + \beta_4 EL + \beta_5 VR + \beta_6 Visit\ Diagnosis + \beta_7 Visit\ EL + \beta_8 Visit\ VR + \beta_9 Visit^2\ Diagnosis + \beta_{10} Visit^2\ EL + \beta_{11} Visit^2\ VR + \beta_{12} Visit\ EL\ Diagnosis + \beta_{13} Visit\ VR\ Diagnosis + \beta_{14} Visit^2\ EL\ Diagnosis + \beta_{15} Visit^2\ VR\ Diagnosis + \varepsilon$

Applying this model to this dataset was not advisable. First, the number of predictors and interactions would have resulted in a low statistical power for the analysis given the size of our dataset. Second, EL and VR are highly co-linear and if both were included by default, their shared variance would be excluded from the analysis. We therefore opted for a stepwise

model selection process, gradually decreasing the number of predictors in the model. We compared the relative plausibility of the models given the data, as measured by the Bayesian Information Criterion, a statistical estimate of out-of-sample error, that is, of the generalizability of the model to new data (BIC, Schwarz, 1978). This method was chosen to avoid the increased chance of false positives that would result from using significance testing or increases in explained variance for model comparison (Hastie, Tibshirani, & Friedman, 2009). Amongst information criteria, BIC was selected as it has been shown to generate the least prediction error in the presence of medium size datasets (>100 and <500 data points), with co-linear predictors and high unsystematic individual variability (that is, high variability due to random effects, as one would expect when dealing with children and in particular children with ASD) (Brewer, Butler, & Cooksley, 2016). In the interest of brevity, we report the selected models only, but the analysis script provided can be used to generate BIC scores for all submodels. Note that when performing model selection based on information criteria, the relevance of the predictors is only determined by whether they decrease the estimated out-of-sample-error and not by their p-value. P-values in the models defined via information-criteria-based model selection are only reported to comply with statistical reporting guidelines.

We chose to examine the impact of ADOS scores in separate models including the ASD group only. While ADOS scores were also obtained for TD participants, the distribution in the full sample was bimodal, and variability in ADOS scores on TD children is difficult to interpret. The full model explored was:

1. $$LinguisticFeature = \beta_{0i} + \beta_{1i} Visit + \beta_{2i} Visit^2 + \beta_3 ADOS + \beta_4 EL + \beta_5 VR + \beta_6 Visit\ ADOS + \beta_7 Visit\ EL + \beta_8 Visit\ VR + \beta_9 Visit^2\ ADOS + \beta_{10} Visit^2\ EL + \beta_{11} Visit^2\ VR + \beta_{12} Visit\ EL\ ADOS + \beta_{13} Visit\ VR\ ADOS + \beta_{14} Visit^2\ EL\ ADOS + \beta_{15} Visit^2\ VR\ ADOS + \varepsilon$$

2.4.2. Modelling clinical and cognitive factors as predictors of parents' longitudinal linguistic trajectories

We employed models analogous to those described above, with parental linguistic performance as the outcome and no other change.

2.4.3. Within-visit linguistic matching: modelling the relation between children's and parent's linguistic production within the same interaction

In order to assess linguistic matching within each visit, we created mixed effects model for each feature in the parent's linguistic productions: we included Child Production (for the same linguistic feature), Visit, Diagnosis, EL, and VR as fixed effects; individual participants' intercept and slope over time were included as correlated random effects.

1. $$ParentalLinguisticFeature = \beta_{0i} + \beta_1 ChildLinguisticFeature + \beta_{2i} Visit + \beta_3 Diagnosis + \beta_4 EL + \beta_5 VR + \varepsilon$$

Because we were interested in potential interactions between child production, individual differences, and diagnosis, we applied a model selection procedure analogous to the previous analyses.

2.4.4. Predicting future linguistic performance in the child from parental production, cognitive and clinical features

To predict language development in future visits, we created mixed effects models for each linguistic feature in children's production with the child's given linguistic feature at visit n as outcome (dependent variable); the child's linguistic feature at the previous visit (n-1), parental linguistic features (types, tokens, and MLU) at the previous visit, Diagnosis, EL, and VR as fixed effects; and individuals' intercepts and slopes over time as correlated random effects.

1. $$ChildLinguisticFeature = \beta_{0i} + \beta_1 ChildPreviousProduction + \beta_2 ParentalPreviousTotalWords + \beta_3 ParentalPreviousUniqueWords + \beta_4 ParentalPreviousMLU + \beta_5 Diagnosis + \beta_6 EL + \beta_7 VR + \varepsilon$$

As we were interested in potential interactions between parental production, individual differences, and diagnosis, we applied a model selection procedure analogous to the previous analyses. Because the previous analyses showed that Diagnosis, EL and their interaction were significant predictors of child development, we kept these as default components of all models tested.

2.4.5. Comparatively assessing the role of the child's cognitive and clinical features and of parents' production in predicting the child's future performance

To assess the relative importance of child-based and environmental factors in language development, we took the selected models from the previous analysis and compared the relative variance explained. In a first analysis, we standardized all relevant variables involved, in order to be able to directly compare the standardized coefficients (that is, the number of standard deviations in the outcome varying in relation to one standard deviation of change in the predictor). Second, we performed exploratory analysis of the standardized effects by group, to assess whether we would find different roles for the different factors in the two groups.

2.4.6. Predicting future linguistic performance in the parent from the child's production, cognitive and clinical features

We employed analogous models to those described above, with parental linguistic performance as the outcome and no other change.

2.4.7 Additional information on the implementation of the analyses

Unless otherwise specified, all predictors were centered but not scaled, to facilitate interpretation. Where relevant to the interpretation of interactions, we performed post hoc analyses by applying the same statistical model selected for the full analysis but separated by group. This of course involved excluding diagnosis and its interactions as a predictor. Note that this procedure is not aimed at assessing statistical significance within the post hoc models, but only to interpret the interactions highlighted by the full statistical models.

For each model, we also report measures of explained variance: conditional R^2 (R^2) indicating the percentage of variance in the outcome measure that is explained by the full model (fixed and random factors); marginal R^2 (R^2m) indicating the percentage of variance

in the outcome measure that is explained by the fixed factors alone (Johnson, 2014). We report both R^2 and R^2m to provide information not only on how much variance is explained, but also on how much of this variance pertains to systematic individual differences. Note that R^2 measures can theoretically cover the full range from 0 (no variance explained) to 1 (all variance explained), and correspond to squared Pearson's coefficients, or r. While reference scales of effect sizes have been suggested – e.g. r = 0.1 and R^2 = 0.01 as small; r = 0.3 and R^2 = 0.09 as medium; and r = 0.5 and R^2 = 0.25 as big – these scales apply poorly to multiple regression models and effect sizes are best interpreted contextually.

All models were implemented using lme4 1.1–15 (Bates, Maechler, Bolker, & Walker, 2014), MuMIn 1.40.4 (Bartoń, 2013), and ggplot2 2.2.1 (Wickham, 2009) on R 3.4.4 (RCoreTeam, 2018).

All data employed in the analyses (that is, indexes of child and parental linguistic performance at each visit, as well as relevant demographic, clinical and cognitive descriptors of the children), the pre-processing and analysis scripts are available as Open Science Framework: https://osf.io/u42dq

3. Results

3.1. Longitudinal Trajectories: Children

Table 2 presents the three measures of linguistic performance side by side (one per column) for ease of comparison, with rows reporting the single predictors in the models. If a predictor was not included in any of the models for the three linguistic measures (i.e., it did not decrease our BIC estimates of out-of-sample error), it was not included in the table. All linguistic features investigated in the children's production displayed very similar trajectories, showing a significant increase over time, which slowed down in later visits (i.e., see the significant β's for Visit and $Visit^2$ in Table 2, and Figure 1). Expressive language (EL) was a strong predictor of language development: the higher the EL, the higher the children's linguistic performance on all three measures. Moreover, higher EL at visit 1 also predicted steeper language increases over time for MLU (Visit : EL interaction in Table 2), and smaller eventual slow-downs in development ($Visit^2$: EL interaction in Table 2). Finally, diagnosis played a role in all features of language development after controlling for individual cognitive differences, with children with ASD producing fewer word tokens, and utterances with shorter MLUs than TD children (Diagnosis row in Table 2). Moreover, their rate of linguistic development was less steep (Visit : Diagnosis row). We also observed interactions between clinical and cognitive features, where Diagnosis modulated the effects of initial EL on average (2-way interaction) and over time (3-way interaction); see Table 2, bottom two rows. Post-hoc inference indicated that EL had a stronger relation with language development in the ASD group than in the TD group for word tokens (ASD: β = 14.33, SE = 5.07; vs. TD: β = 12.47, SE = 8.32), word types (ASD: β = 4.15, SE = 1.13; vs. TD: β = 1.68, SE = 1.77), and MLU (ASD: β = 0.05, SE = 0.02; vs. TD: β = 0.03, SE = 0.02).

Visual Reception (VR) did not play a significant role and so was not included in the models. The models explained a large portion of the variance in the children's linguistic performance (marginal R^2: word tokens: 0.59; word types: 0.68; MLU: 0.67), though the children's

individual variability is not exhausted by the factors analyzed (R^2: word tokens: 0.82, word types: 0.88; MLU: 0.79). The findings for each measure are depicted in Figure 1.

In the follow-up model, we assessed the role of the severity of clinical features (ADOS scores) within the ASD group alone; see Table 3. "Not Included" indicates predictors that did not improve the plausibility of the models and were therefore not added to them. ADOS score was never included, in other words, knowing the ADOS score of a child did not provide additional information about the child's linguistic development, beyond the information provided by visit and verbal IQ scores. All other parameters are reported as control parameters and show analogous, albeit weaker, patterns to the previous analyses.

3.2. Longitudinal Trajectories: Parents

Parental linguistic production, as shown in Table 4, increased over time (i.e., there were significant effects of Visit) for word types and MLU, with only MLU showing a quadratic tapering off over time. Parental word types were not related to the children's clinical or cognitive features; however, children's VR score was positively related to both parental word tokens and MLU, though no interaction with development over time was observed (and thus VR : Visit is not included in the table). Parents of children with ASD showed lower MLU overall; however, including ADOS scores did not increase the model's ability to explain parental linguistic performance (see Table 5). At the same time, we note that the explanatory power of our models for parental linguistic performance (R^2m between 7% and 27% of the variance in the data) is far below that for children's linguistic performance (R^2m > 57%). These results are illustrated in Figure 2.

3.3. Within-visit linguistic matching

Overall, parents and children matched each other's concurrent linguistic production. We observed a main matching effect of child production for all features (see Table 6, Child Production Row), with no included interactions of children's production with any other potential predictor. The matching was stable over time as well, with no significant interaction with Visit. These results are illustrated in Figure 3.

3.4. Predicting child's performance from earlier parental production—Detailed results about the models predicting child's performance from earlier parental production are reported in Table 7. We find that all indexes of child linguistic performance at visit N are significantly predicted by parent MLU at visit N-1. Parent tokens and types did not contribute significantly and so were not included in the final model. We find again that Diagnosis, EL scores, and their interaction affect children's performance on the three measures, even when controlling for the child's baseline (i.e., performance at visit N-1), thus suggesting that they regulate the rate of language acquisition. However, their interaction with parental production did not improve any of the models, suggesting that the impact of parental production is not related to either Diagnosis or EL.

3.5. Assessing the relative roles of child-based and environmental factors

In order to be able to compare the relations between child-based and environmental factors on child language development, we standardized all outcomes and numeric predictors. This

standardize all regression coefficients on the same scale (see Table 8). After controlling for child baseline for each measure, both child-based and environmental factors seem to have an impact on subsequent child language, with child-based factors, and diagnosis in particular, having a stronger role. Indeed, while parental input is comparable in effect to MSEL-EL, diagnosis and its interaction with MSEL-EL have decidedly greater effect sizes.

A post-hoc inference (see Table 8) - employing the same statistical model selected for the full analysis, but separated by group - suggested that in the TD group child-based (namely MSEL-EL) and environmental factors seemed to similarly relate to language development, while in the ASD group child-based factors were a stronger predictor than environmental ones.

3.6. Predicting parental performance from earlier child production

As summarized in Table 9, we found that knowing the child's linguistic production did not help in predicting the overall number of word tokens used by the parents. However, it did help in predicting both vocabulary diversity and sentence complexity. That is, the number of parental word types was positively related to the child's previous sentence complexity (MLU). Parental sentence complexity (MLU) was related in more nuanced ways to all three indexes of child linguistic production, with higher child word tokens and MLU at visit N predicting smaller parent MLUs at visit N+1, while greater child word types at visit N predicted larger parent MLUs at visit N+1. For the moment, these findings provide evidence of effects of child speech on subsequent parent speech; however, we note that interpretations of the directionality of these effects need to be extremely cautious, due to high co-linearity between the features.

It is also important to note that the variance in parental production explained by these models (R^2m of 61%, 20% and 49%) is generally lower than that explained in child production by models of parent production (R^2m of 63%, 73% and 64%), especially with respect to lexical and syntactic complexity (that is, word types and MLU). This suggests that the level of cross-lagged or progressive adaptation is higher for children than it is for adults.

4. Discussion

Recent research has begun to extend the literature investigating the roles of child-based and environmental factors on the language development of TD children, to children with ASD (Nadig & Bang, 2017). In this study, we continued this extension by (a) using a large longitudinal corpus that included six data collections across 2.5 years, (b) targeting parent and child language measures (word types, word tokens, MLU) drawn from naturalistic conversations, (c) comparing development during early childhood among children with ASD and TD peers who were initially matched on overall language, and (d) employing multi-level modelling that accounts for individual variation in starting conditions and development among participants, as well as the relation between the two. Crucially, we also wished to take seriously the potential role of children in influencing their own linguistic environment: that is, the reciprocal interaction between children's response to parents' input, and parents' response to children's production.

We report six major findings.

1. *Longitudinal trajectories of language development – child-based factors:* Children's production of all three measures increased significantly across visits, and was significantly predicted by their expressive language (EL) scores (positively) and diagnosis (negatively) from Visit 1 (Table 2, Figure 1).
2. *Longitudinal trajectories of parental language production – child-based factors:* Parents' productions also increased across visits, and were predicted by their child's nonverbal cognition scores (positively) and diagnosis (negatively) from Visit 1 (Table 4, Figure 2).
3. *Parent-child matching in concurrent linguistic production:* At any given visit and across groups, children and parents reliably matched each other in lexical and syntactic production (Table 5, Figure 3).
4. *Predicting child linguistic development from environmental factors:* Parents who produced longer MLUs during a given Visit (N) had children who produced more word types and tokens, and had longer MLUs, at the subsequent Visit (N +1), a relation that was not modulated by diagnosis (Table 7).
5. *Assessing the relative roles of child-based and environmental factors in child linguistic development:* When both child EL at Visit 1 and parent MLU were included in the model, both contributed significantly to future child language across both children with ASD and TD children; however, within the ASD group, EL accounted for a greater proportion of the variance than the environmental factors (Table 8).
6. *Predicting parental linguistic production from child linguistic production:* Finally, children's speech at Visit N also significantly predicted parent speech at Visit N+1, demonstrating reciprocal parent-child effects across the two groups (Table 9).

In what follows, we discuss these findings with respect to our three major goals: an assessment of how our findings replicate and extend previous studies on the role of 1) child-based and 2) environmental factors in TD children and children with ASD's linguistic development, as well as 3) an assessment of the reciprocal adaptation of children and parents in their language production.

4.1. Child-based factors influencing children's linguistic development

Our first major goal was to evaluate the impact of child-based factors such as initial language and cognitive levels and diagnosis on the patterns of language development for both groups of children (see Table 2). Consistent with the extant literature, we showed linear and quadratic patterns of development for all of our three linguistic measures (word tokens, word types, and MLU), with the slopes of the children with ASD being shallower than that of the TD children (Anderson et al., 2007; Paul et al., 2008; Szatmari et al., 2009). Diverging from earlier findings, though, symptom severity in the children with ASD was not predictive of their trajectory of language development (see Table 3); in contrast, previous researchers (Ellis Weismer & Kover, 2015; Paul et al., 2008; Szatmari et al., 2009; Thurm et al., 2015)

did find ASD severity to predict language growth during the preschool and school years. These differing findings may be attributable to the different outcome measures: our measures were drawn from speech produced during naturalistic parent-child interactions in the home whereas those of the previous studies relied exclusively on standardized tests. It is likely that children's performance on these tests is generally influenced by ASD severity (i.e., across contents) because of attentional and motivational demands; see Naigles and Chin (2015) for more discussion.

Additionally, by looking at child-based factors in all the children together, and using diagnosis as only one of several distinguishing factors, we were able to illuminate common factors that cut across group divisions. While non-verbal cognition did not add to the predictive power of the model in either group (in line with Bang & Nadig, 2015), initial expressive language scores were predictive of language development in both groups. Thus, early child-based measures, like the Mullen Expressive Language score, may be indexing a general orientation toward verbal language that is also to some degree independent of diagnosis. This corroborates previous findings with TD children (Fernald & Marchman, 2012; Hoff, 2003; Ramírez-Esparza, García-Sierra, & Kuhl, 2014; Rowe, 2008) as well as children with ASD (Bang & Nadig, 2015; Pickles et al., 2014; Venker et al., 2015). Importantly, the three-way interaction between Diagnosis, EL, and Visit also suggests that not only do children who are less advanced in verbal language from the beginning progress more slowly on speech production measures than those who are more advanced, but that this is even more true for children with ASD. This pattern of initial language level weighing more heavily in the ASD than TD group is new, as Bang and Nadig (2015) did not compare diagnostic groups on initial language effects and most other studies investigating predictors of language development in ASD have not included a TD comparison group. One conjecture is that this pattern derives from the differential distribution of the EL scores in our two groups. As depicted in Figure 4, variability in the ASD group extended to both the lower and higher ends of the scale at Visit 1, and measures with greater variability will generally demonstrate stronger statistical effects. Our findings further indicate that studies investigating the predictors of language outcomes in children with ASD should include early language measures, as previous studies reporting NVIQ as a predictor of later language (Bopp et al., 2009; Ellis Weismer & Kover, 2015; Wodka et al., 2013) did not include an early language measure as well. It is important to include both early language and early nonverbal cognition measures as predictors, so that their relative weighting can be ascertained.

4.2 Environmental factors influencing children's linguistic development

The second major goal of the present study was to evaluate the impact of environmental (parent-based) factors on both TD children and children with ASD. Previous studies have indicated that quantity and quality of linguistic input might positively affect child language development in both groups (Bang & Nadig, 2015; Venker et al., 2015). In our sample, syntactic complexity of parental speech (MLU) was a significant predictor of the number of words, lexical diversity, and syntactic complexity of children's productions at the subsequent visit, even when controlling for the child's diagnosis and language performance in the previous visit and at the beginning of the investigation. Thus, our findings are consistent

with those from previous studies that have argued for the importance of quality of parental input above and beyond quantity, focusing on word types, syntactic complexity and communication quality (Bang & Nadig, 2015; Demir, Rowe, Heller, Goldin-Meadow, & Levine, 2015; Hirsh-Pasek et al., 2015; Hoff & Naigles, 2002; Huttenlocher et al., 2010; Jones & Rowland, 2017). In particular, our results emphasize the importance of parental syntactic complexity (MLU) above (and controlling for) other measures of parental production (including lexical diversity) and suggest that syntactic complexity is equally beneficial across the six visits (i.e., interactions with Visit did not add significantly to the models). Like Venker et al. (2015), we find that providing children with ASD with more complex utterances (full NPs including determiners in their case, longer sentences including more morphological structures in ours) is beneficial to their subsequent lexical and/or grammatical development, with this complexity likely processed as data about their target language (Hoff & Naigles, 2002). We should emphasize that children with ASD seem to learn just as much as TD children from parental input (no interaction between diagnosis and parental MLU in predicting the child's subsequent linguistic performance), corroborating the findings of Bang & Nadig (2015) and Goodwin et al., (2015) with a larger sample. Because previous studies have investigated children of similar age (Venker et al., 2015) and older (Bang & Nadig, 2015) with similar results, the effect of complexity of parental input seems remarkably stable. Does this consistency of input usage across groups indicate that the TD children and children with ASD are using similar language learning mechanisms? While our production data cannot speak directly to this question, data gathered from these same children during comprehension tasks suggests that many language learning mechanisms are similar across the two groups, as discussed in detail by Naigles and Fein (2017).

Effects of parental input in our study were smaller than the effects of child-based factors; this relative weighting was also observed by Bang and Nadig (2015) and Venker et al., (2015). That is, children's initial language levels were stronger predictors of their subsequent language than was their parents' MLU. We conjecture that this relative weighting might go some way towards explaining why the children with ASD nonetheless showed shallower trajectories of language development than the TD children, even though they were evidently using their input to the same extent. More specifically, it is likely that the children with ASD with the lowest EL scores at Visit 1 were most implicated in the shallower slopes. However, we must also stress that children's expressive language at visit 1 should not be taken as a 'pure' index of biologically-based language ability. The children at visit 1 averaged 21 months (TD) to 33 months (ASD) of age, and so had already experienced 1–3 years of parental input and interactions, which likely influenced their language levels at visit 1. Indeed, Hoff (2003) has already shown the importance of maternal MLU on TD linguistic development at 21 months. Further investigations might distinguish the child-based and environmental effects more clearly by assessing children when they are younger (i.e., pre-diagnosis). For example, their performance on statistical/distributional learning tasks during their first year of life might be a less input-influenced indicator of their language learning abilities. Comparing the roles of therapists' speech and parents' speech on the subsequent language of children with ASD could also help disentangle the genetic and environmental effects.

Interestingly, child-based measures did not interact significantly with parental input in our models; that is, while child-based factors may set the stage for children's language learning, they do not seem to directly modulate the benefit that either TD or ASD groups can gain from higher levels of syntactic complexity in parental productions. This might seem contrary to the intuitive expectation that children with very low EL would require simpler parental input to achieve any learning, and hence would show smaller effect sizes for parental MLU. However, two phenomena could explain this counter-intuitive finding. As parents adapt to their children (see next section for more discussion), children with lower verbal skills receive lower complexity input, and both low verbal skills and less complex parental input are associated with lower learning, thus confounding the interaction. Additionally, early diagnosed children with ASD are often exposed to other adults for significant amounts of time, but we did not investigate the contribution of the language of these other adults in the current study. It is quite possible that the language of other significant adults in the family, or the child's therapists, would also have a significant effect on the child's language development, above and beyond explicit teaching. This relationship might be even more complex, since some young children with ASD are exposed for many hours per week to several different therapists or teachers, sequentially or simultaneously. This additional input from therapists may also serve to facilitate the child's usage of their input from their parents.

Finally, we acknowledge that responsive social interactions that are initiated and/or sustained by the caregiver, and include joint attention and engagement as well as routines, are also important components of children's language-learning environments. Experience with these interactions has been shown to positively predict larger vocabularies in both TD children and children with ASD (Adamson, Bakeman, Deckner, & Romski, 2009; Haebig, McDuffie, & Weismer, 2013; Hirsh-Pasek et al., 2015; McDuffie & Yoder, 2010; Tamis-LeMonda et al., 2014; Tamis LeMonda, Bornstein, & Baumwell, 2001). In the current study we have only focused on structural aspects of parental speech; however, it will be important for future investigations to include both structural and social/responsive measures of children's environments so as to provide a more complete model of their influences on later language (see Abdel-Aziz et al., in press; Park et al., 2012 for preliminary findings with this dataset).

4.3. Reciprocal influence in shaping the linguistic environment

Very little research has been done to date on the way children shape the linguistic environment in which they are raised, which in turn plays an important role in their linguistic development (Van Dijk et al., 2013; Yurovsky, 2017). For instance, it has been shown that within naturalistic conversations parents and TD children re-use each other's syntax (R. Dale & Spivey, 2006; Fernández & Grimm, 2014), and that the quality of pre-verbal vocalizations in infants might affect the speed of response in their parents (Warlaumont et al., 2014). However, no systematic investigations of the reciprocal adaptation in syntactic and lexical production over multiple visits in longitudinal corpora have been published, as previous studies mostly assessed children's influence on parent speech across two time points (Huttenlocher et al., 2010; Song et al., 2014; Venker et al., 2015). The paucity of studies may be in part because reciprocity, while constitutive of much human interaction, is difficult to quantify, particularly when dealing with a behavior that is developing at a rapid rate in one of the parties, and relatively stable in the other (R. Dale,

Fusaroli, Duran, & Richardson, 2013; Fusaroli, Konvalinka, & Wallot, 2014; Fusaroli, Raczaszek-Leonardi, & Tylén, 2014; Fusaroli & Tylén, 2016). To complicate matters, several factors can be in play simultaneously. For instance, Dale et al. (2015) point out that the shared genetic material of parents and children itself can be an important factor in the language environment surrounding the child. While there is substantial evidence showing that quantity of words can have a positive effect on child language development, those children who have a genetic predisposition to produce less language themselves and therefore might benefit most from increased input from their parents may be more likely to have parents who provide less input, since that very tendency to verbalize less may be inherited from the parents.

In order to provide a first assessment of these issues, we analyzed the impact of child production and child-based factors on parental production within the same visit (Table 6) and across the six visits (Table 9). Within the context of each filmed visit, children's and parents' productions matched each other on all of the three measures, that is, child production of types predicted parent production of types, etc. This was found for both TD and ASD child/parent dyads, and was stable from one visit to the next. While this is not a direct measure of reciprocal influence, it does suggest that parents and children are "on the same wavelength" in terms of their linguistic production. The fact that our parent-child dyads matched each other so well could be due to many factors, including genetic and cultural—some families will simply talk more or differently than others—but also to the parents' sensitivity to their children's speech attempts. It is important to point out that the kind of matching we assessed here was on the level of *numbers* of word tokens and types, and *overall* MLU, not on the more specific level of actually using the *same* words or structures. This latter kind of matching is termed linguistic alignment (Fusaroli & Tylén, 2016), and analyses thereof are currently underway with this dataset (Fusaroli, Weed, & Naigles, 2016).

To better assess parental adaptation to their children, we then investigated whether child production and child-based factors were able to predict parental production in the next visit, while controlling for parental baseline at the current visit. This analysis allowed us to side-step some of the issues in the previous analysis. Shared variance in the child-parent dyad is controlled for by focusing on changes in parental production from baseline, and having both parental and child production at baseline as predictors. Children's adaptation is controlled for by predicting future (and not current) parental production. We found that parental production was weakly (albeit significantly) related to earlier child-based factors and child production. Parents of children with ASD maintain a steady flow of linguistic input to their children, just as parents of TD children do; however, they may modulate the complexity of their input, based on the level of social and communicative skill displayed by their child (see also Yurovsky, Doyle, & Frank, 2016). This is reinforced by the analysis of conversational matching of language production (see Table 6): although parents of children with ASD produced the same number of tokens and types as parents of TD children, they had a significantly lower MLU overall, and the level of MLU was positively associated with children's non-verbal cognition (MSEL VR). While child-based factors were not nearly as predictive of parental production as they were of children's production, these findings do suggest that parental production is related to measurable child-based factors. The

relationship of child nonverbal cognition to parental production is harder to interpret, especially given the lack of importance of this measure for the child's own language development. One possibility is that while non-verbal cognition does not impact actual language development in the child, it does impact parents' perception of the child as a conversational partner, and they may adapt to this perception to some degree. We speculate that non-verbal cognition might act as an index of how well children seem to understand the world and how much they interact with it. Thus, children with higher VR might display higher engagement and afford more targeted and engaged speech from their parents (for a similar perspective on the role of gesture in eliciting more complex parental input, see Rowe & Goldin-Meadow, 2009; Schwab & Lew-Williams, 2016). However, we should also note that we did not find any interaction between non-verbal cognition and time, nor did we find any impact of non-verbal cognition on the number of types produced by parents, suggesting that whatever effect non-verbal cognition has on parental production, it may be limited.

Crucially, we also found child MLU was weakly but positively predictive of parental word types at the subsequent visit. Thus, children who produced longer MLU at Visit N have parents who produced more types at Visit N+1. We suggest that this pattern of adaptation occurred because parents (implicitly) observed the increased sentence complexity of their children at Visit N, and so increased their own lexical complexity to 'up the ante' by (and probably sooner than) Visit N+1. For example, one very concrete interpretation of this effect is that parents who hear children newly using specific constructions (e.g., PPs) might increase their usage of diverse e.g., nouns or prepositions that might 'populate' those constructions. We also found that diagnosis predicted parental MLU; however, because diagnosis interacted with child word token production in its influence on parental syntactic complexity, only in the ASD group did more word tokens from the children lead to longer MLU in the parents at the next visit. Thus, parents of children with ASD may be paying more attention to their children's word tokens as they adjust their own speech, compared with parents of TD children.

These findings resonate with recent literature, although they do not match it completely. Other studies have suggested that parents' speech is sensitive to children's earlier behavior (Huttenlocher et al., 2010; Song et al., 2014); however, these studies reported parental sensitivity to child vocabulary or cognitive development, whereas our findings point to parent sensitivity to prior child vocabulary and syntactic complexity (i.e., MLU). Neither Huttenlocher et al. (2010) nor Song et al. (2014) considered child MLU as a predictor of later parent speech; clearly, more research and replication of the results is needed. We note, however, that the variance explained in subsequent child speech was higher (R^2 = .66-.78) than that explained in subsequent parent speech (R^2 = .52-.65), and suggest that this is because the parent is the expert and the child is the novice at the language learning activity. Thus, the child has more ground to cover at their stage in development and so can be more influenced by their environment.

4.4 Potential for intervention

The role of parental or adult input in children's language production and development should not be underestimated. TD children who are exposed to more complex language learn

more and produce more complex language (Huttenlocher, Vasilyeva, Cymerman, & Levine, 2002; Huttenlocher et al., 2010; Newport et al., 1977); moreover, our results suggest that, within the parameter range we observe, children with ASD learn as much from parental production as TD children do. This is also supported by a previous meta-analysis (Sandbank & Yoder, 2016), indicating a strong positive association between parental input complexity and child language outcome in ASD.

Given our additional albeit weaker effect of children's language on parent production, feedback loops between children and parents—or therapists—might yield positive or negative cascading effects on the learning process, depending on their direction. At the very least, these findings support further calls for early diagnosis of ASD, and early and intensive interventions, with parents being made aware that their own speech is also important for their child's language development.

Several language intervention programs for children with language disabilities recommend the use of simplified and shortened speech (see a discussion of this with references in Sandbank & Yoder, 2016). Moreover, in the movement toward individualizing treatment, therapists often try to match the complexity of the child's expressive language in order to start teaching at the child's current level (Stahmer, Schreibman, & Cunningham, 2011). Our results suggest that this might not be fully adequate. Exposing the child to a balance of simple syntax which is matched to the child's output to ensure compliance with current activities, and more syntactically complex natural language in order to provide a more complete model of the target language, might be more beneficial for some children. Finally, scrutiny of Figures 1 and 4 suggests that at least some of the children with ASD might also have Developmental Language Disorder (Kjelgaard & Tager-Flusberg, 2001); for these children, if the above strategies prove less effective then they might be supplemented with other communication systems, such as gesture, PECS, or other augmented communication systems.

5. Conclusions

We set out to investigate the roles of child-based and environmental factors, as well as parent-child adaptation, in child language acquisition in a large longitudinal corpus of naturalistic parent-child interactions involving language matched children with ASD and TD children. In line with previous research we show that while autism has profound effects on language development, individual differences in early language ability have an additional effect for both TD children and children with ASD. To some extent, then, early language presentation, modulated here by ASD diagnosis, does set the stage for the language acquisition that ensues. However, these language trajectories are also influenced, significantly and positively, by the complexity of parental speech, and this finding is especially important for children with ASD. Finally, we provide new and important evidence for the existence of reciprocal influences of child on parent as well as parent on child, with parents weakly modulating their linguistic complexity to the child's IQ, autistic characteristics and actual linguistic production.

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Highlights

- Longitudinal corpus of naturalistic parent-child interactions
- 32 children with ASD and 35 language-matched controls.
- Language development in TD and ASD alike is shaped by child-based and environment al factors
- Parents adjust linguistic production to the child's level of production

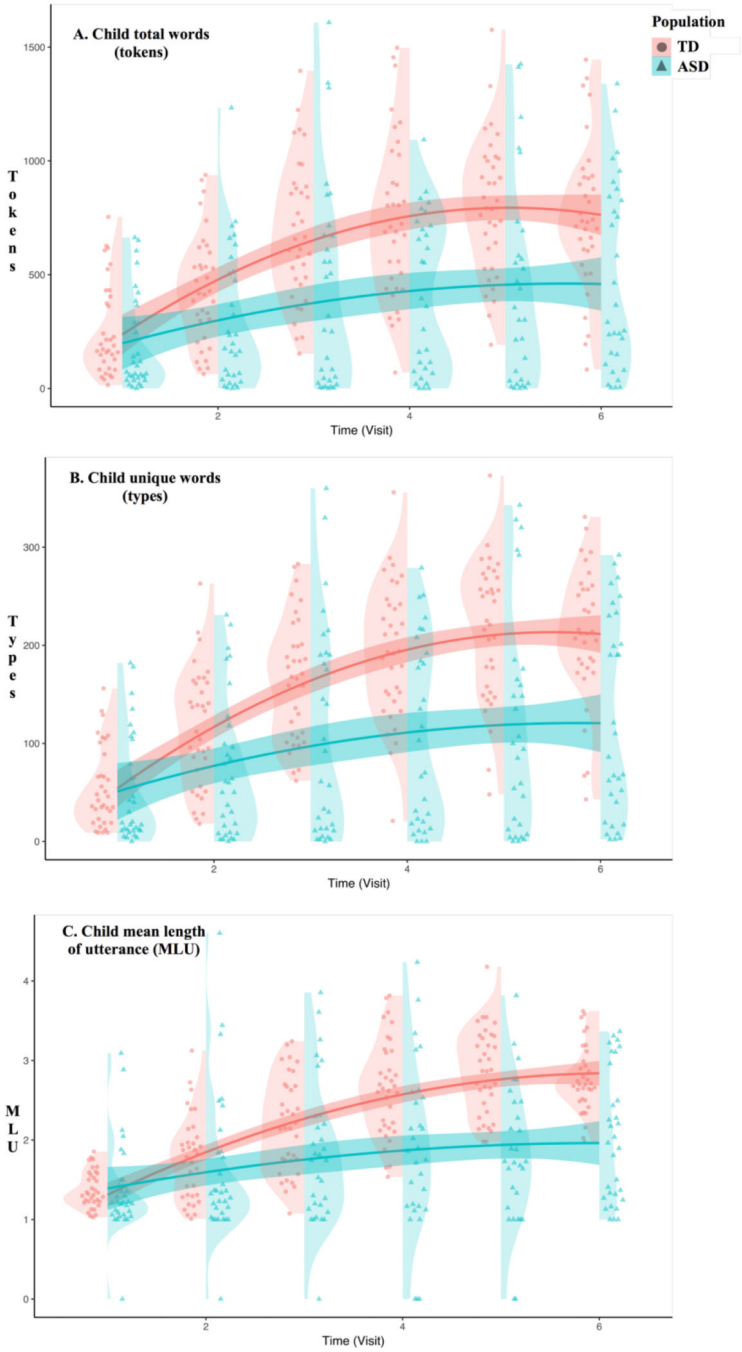

Figure 1 –. Longitudinal trajectories of linguistic performance in TD and ASD children.
Word tokens are in the top panel, word types in the central panel and MLU in the bottom panel. We split the data into children with ASD (blue triangles) and TD children (red circles) and included background half violin plots to better display the distribution of the data by group.

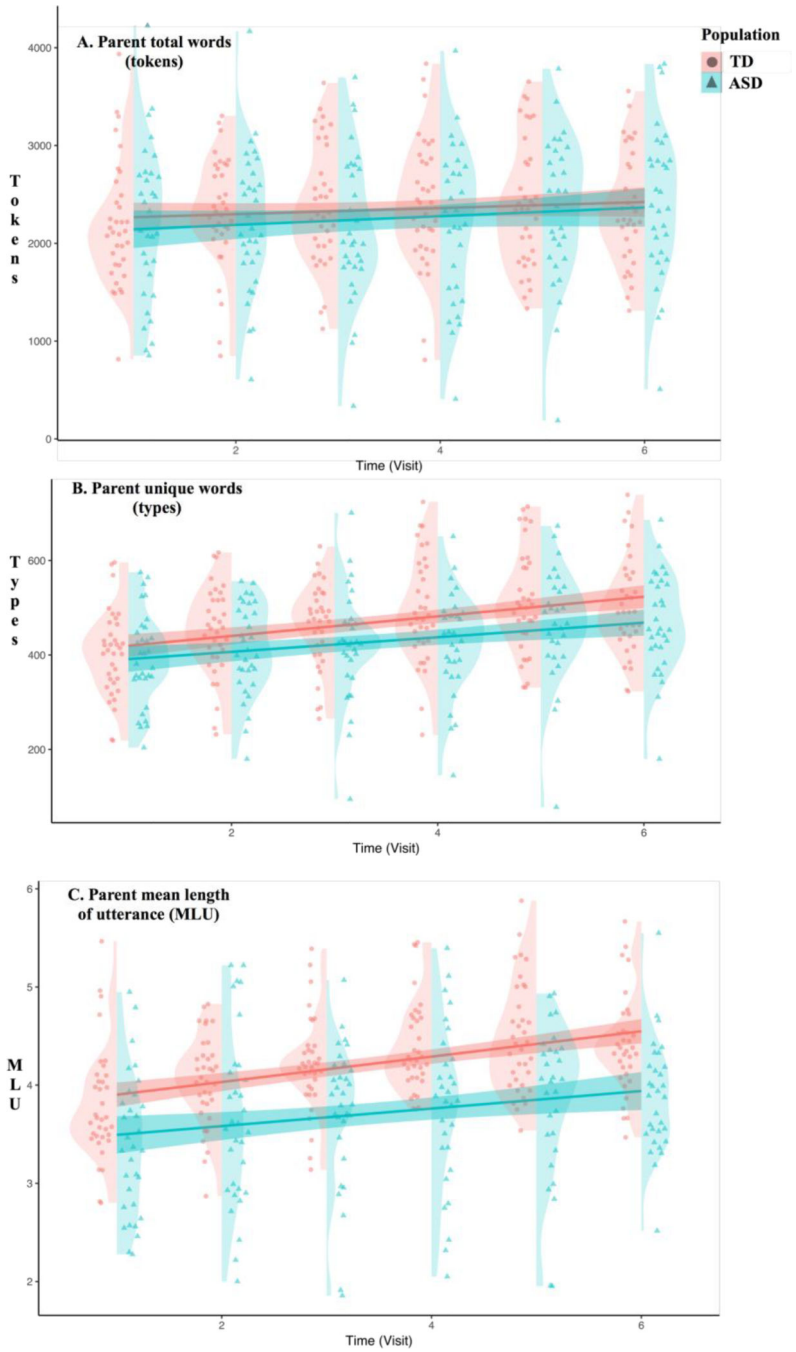

Figure 2 –. Longitudinal trajectories of linguistic production in parents of TD and ASD children. Word tokens are in the top panel, word types in the central one and MLU in the bottom one. The data is split into parents of children with ASD (blue triangles) and TD (red circles) children. The figures include background half violin plots to better display the distribution of the data by group.

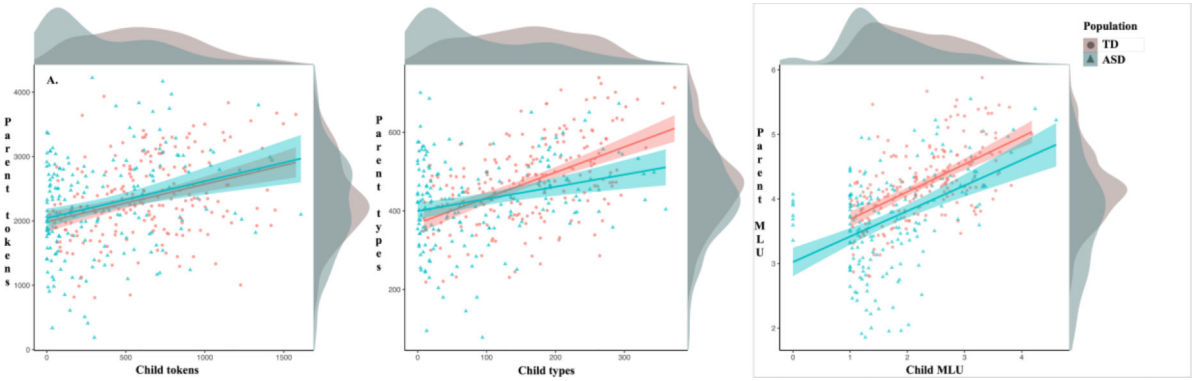

Figure 3 –. Linguistic matching in parent-child dyads.

Word tokens are in the left panel, word types in the central one and MLU in the right one. The data are split into parent-child dyads in the ASD (blue triangles) and TD (red circles) groups.

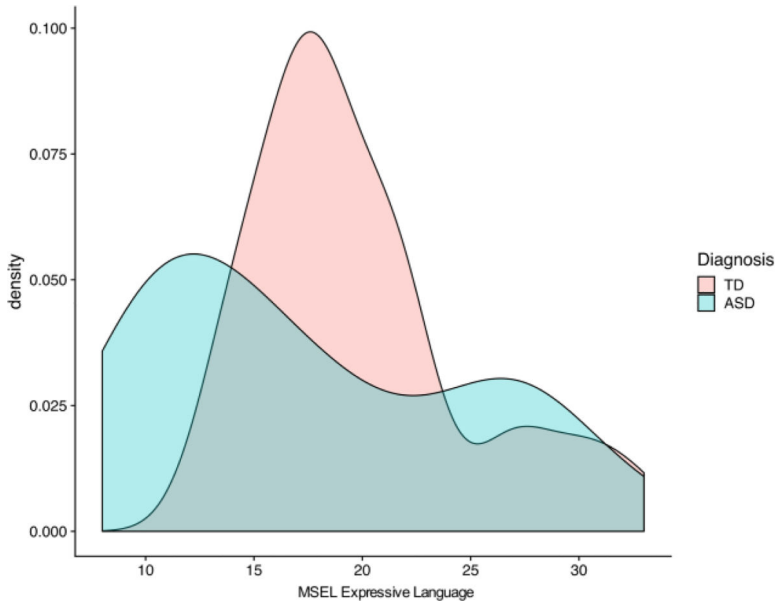

Figure 4 –.
Distribution of Expressive Language scores in the ASD and TD groups.

Table 1:

Descriptive statistics of the population.

Variable	TD Mean (95% CI)	ASD Mean (95% CI)	β (SE)	t/z-stats	p	R^2
Gender (Female vs. male participants)	29 boys, 6 girls	28 boys, 4 girls	0.37 (0.7)	z-stat: 0.53	0.6	0.01
Age (months)	20.27 (19.78 20.93)	32.98 (31.03 34.74)	12.79 (0.98)	t-stat: 13.09	<0.001	0.72
MSEL-EL	Raw scores: 19.89 (18.4 21.76) Age equivalent: 20.49 (18.80 22.94)	Raw scores: 17.56 (15.29 20.3) Age equivalent: 18.56 (15.67 21.85)	−2.32 (1.53)	t-stat: −1.52	0.13	0.03
MSEL-VR	Raw scores: 26 (24.83 27.09) Age equivalent: 23.19 (21.73 24.49)	Raw scores: 26.91 (14.94 28.79) Age equivalent: 25.24 (23.34 27.78)	0.91 (1.11)	t-stat: 0.82	0.4	0.01
ADOS Mod1 – total score	0.83 (0.43 1.34)	14.12 (12.84 15.47)	13.3 (0.7)	t-stat: 18.42	<0.001	0.84
Child word tokens	252.8 (194.1 331.05)	192.25 (126.83 280.61)	−60.55 (50.01)	t-stat: −1.21	0.23	0.02
Child word types	55.09 (42.53 70.65)	51.19 (33.72 72.08)	−3.9 (11.78)	t-stat: −0.33	0.74	0.02
Child MLU	1.38 (1.3 1.46)	1.36 (1.2 1.59)	−0.02 (0.1)	t-stat: −0.19	0.85	0
Father's education (years past 8th grade)	8.27 (7.39–9.21) range is 4–12	7.53 (6.59–8.5) range is 3–12	0.74 (0.67)	t-stat: 1.11	0.271	0.02
Mother's education (years past 8th grade)	7.91 (7.23–8.53) range is 4–12	7.89 (6.86–8.91) range is 3–12	0.02 (0.6)	t-stat: 0.031	0.975	0

N.B. Means and 95% CI are bootstrapped with 1000 iterations.

Table 2 –

Statistical models of longitudinal trajectories in children's linguistic performance, both TD and ASD groups combined. The models include 395 data points, 32 children with ASD and 35 TD children.

	Word tokens	**Word types**	**MLU**
R^2m, R^2	**R^2m = 0.59, R^2 = 0.82**	**R^2m = 0.68, R^2 = 0.88**	**R^2m = 0.67, R^2 = 0.79**
Predictor			
Intercept	β = 571.90, SE = 29.47, t-stat = 19.41, p < 0.0001	β = 153.44, SE = 6.90, t-stat = 22.24, p < 0.0001	β = 2.21, SE = 0.05, t-stat = 43.02, p < 0.0001
Visit	β = 272.65, SE = 29.56, t-stat = 9.22, p < 0.0001	β = 85.10, SE = 8.22, t-stat = 10.35, p < 0.0001	β = 0.59, SE = 0.06, t-stat = 9.19, p < 0.0001
$Visit^2$	β = −23.34, SE = 3.97, t-stat = −5.88, p < 0.0001	β = −7.48, SE = 1.12, t-stat = −6.71, p < 0.0001	β = −0.04, SE = 0.01, t-stat = −4.43, p < 0.0001
Diagnosis	β = −134.77, SE = 41.37, t-stat = −3.26, p = 0.0017	β = −44.51, SE = 9.99, t-stat = −4.46, p < 0.0001	β = −0.33, SE = 0.07, t-stat = −4.54, p = 0.0015
MSEL EL	β = 22.09, SE = 5.74, t-stat = 3.85, p = 0.0003	β = 4.51, SE = 1.30, t-stat = 3.47, p = 0.0009	β = 0.04, SE = 0.01, t-stat = 3.57, p = 0.0007
Visit : Diagnosis	β = −51.34, SE = 14.15, t-stat = −3.63, p = 0.0005	β = −47.35, SE = 12.05, t-stat = −3.93, p = 0.0002	β = −0.19, SE = 0.03, t-stat = −7.18, p < 0.0001
Visit : EL	β = 9.99, SE = 4.86, t-stat = 2.06, p = 0.0422	β = 1.78, SE = 1.02, t-stat = 1.74, p = 0.0851	β = 0.04, SE = 0.01, t-stat = 3.69, p = 0.0003
$Visit^2$: Diagnosis	Not included	β = 4.30, SE = 1.63, t-stat = 2.63, p = 0.0102	Not Included
$Visit^2$: EL	β = −2.16, SE = 0.63, t-stat = −3.41, p = 0.0010	β = −0.45, SE = 0.13, t-stat = −3.49, p = 0.0008	β = −0.01, SE = 0.00, t-stat = −4.97, p = 0.0001
Diagnosis : EL	β = 18.84, SE = 6.94, t-stat = 2.71, p = 0.0084	β = 5.89, SE = 1.56, t-stat = 3.77, p = 0.0003	β = 0.06, SE = 0.01, t-stat = 4.76, p < 0.0001
Diagnosis : EL : Visit	β = 8.65, SE = 2.37, t-stat = 3.65, p = 0.0005	β = 2.33, SE = 0.54, t-stat = 4.32, p = 0.0001	β = 0.02, SE = 0.00, t-stat = 3.52, p = 0.0007
Random Effects	Child intercept SD: 162.82 Visit slope SD: 140.34 $Visit^2$ slope SD: 18.77 Residual SD: 159.59	Child intercept SD: 37.83 Visit slope SD: 29.85 $Visit^2$ slope SD: 3.82 Residual SD: 32.25	Child intercept SD: 0.26 Visit slope SD: 0.24 $Visit^2$ slope SD: 0.04 Residual SD: 0.39

Table 3 –

Statistical Models of longitudinal trajectories in children with ASD linguistic performance (including ADOS scores). The models include 186 data points, and 32 children with ASD.

	Word tokens	**Word types**	**MLU**
R^2m, R^2	**R^2m = 0.5, R^2 = 0.81**	**R^2m = 0.72, R^2 = 0.91**	**R^2m = 0.59, R^2 = 0.75**
Predictor			
Intercept	β = 372.71, SE = 26.30, t-stat = 14.17, p < 0.0001	β = 109.01, SE = 6.83, t-stat = 15.95, p < 0.0001	β = 1.84, SE = 0.06, t-stat = 31.70, p < 0.0001
ADOS	Not Included	Not Included	Not Included
Visit	β = 44.43, SE = 9.07, t-stat = 4.90, p < 0.0001	β = 37.82, SE = 8.29, t-stat = 4.56, p = 0.0001	β = 0.11, SE = 0.02, t-stat = 5.16, p < 0.0001
$Visit^2$	Not Included	β = −3.19, SE = 1.01, t-stat = −3.15, p = 0.0027	Not Included
MSEL EL	β = 30.66, SE = 2.72, t-stat = 11.26, p < 0.0001	β = 10.41, SE = 0.93, t-stat = 11.19, p < 0.0001	β = 0.08, SE = 0.01, t-stat = 10.83, p < 0.0001
Visit : EL	Not Included	β = 4.15, SE = 1.13, t-stat = 3.68, p = 0.0007	Not Included
$Visit^2$: EL	Not Included	β = −0.46, SE = 0.14, t-stat = −3.35, p = 0.0015	Not Included
Random effects	Child intercept SD: 165.62 Visit slope SD: 166.72 $Visit^2$ slope SD: 17.61 Residual SD: 145.52	Child intercept SD: 36.12 Visit slope SD: 31.18 $Visit^2$ slope SD: 2.98 Residual SD: 28.69	Child intercept SD: 0.28 Visit slope SD: 0.39 $Visit^2$ slope SD: 0.05 Residual SD: 0.42

Table 4 –

Statistical models of longitudinal trajectories in parental linguistic performance. The models include 395 data points, 32 parents of children with ASD and 35 parents of TD children.

	Word tokens	**Word types**	**MLU**
R^2m, R^2	**R^2m = 0.11, R^2 = 0.82**	**R^2m = 0.07, R^2 = 0.77**	**R^2m = 0.27, R^2 = 0.70**
Predictor			
Intercept	β = 2284.70, SE = 67.43, t-stat = 33.88, p < 0.0001	β = 445.60, SE = 10.64, t-stat = 41.87, p < 0.0001	β = 3.27, SE = 0.31, t-stat = 10.52, p < 0.0001
Visit	Not Included	β = 17.17, SE = 2.01, t-stat = 8.55, p < 0.0001	β = 0.31, SE = 0.07, t-stat = 4.60, p < 0.0001
$Visit^2$	Not Included	Not Included	β = −0.03, SE = 0.01, t-stat = −3.17, p = 0.0023
Diagnosis	Not Included	Not Included	β = −0.54, SE = 0.10, t-stat = −5.21, p < 0.0001
Mullen Visual Reception (VR)	β = 51.13, SE = 15.06, t-stat = 3.40, p = 0.0012	Not Included	β = 0.04, SE = 0.01, t-stat = 3.15, p = 0.0025
Random Effects	Child intercept SD: 547.10 Visit slope SD: 321.87 $Visit^2$ slope SD: 38.16 Residual SD: 293.95	Child intercept SD: 87.90 Visit slope SD: 45.59 $Visit^2$ slope SD: 5.33 Residual SD: 52.44	Child intercept SD: 0.41 Visit slope SD: 0.30 $Visit^2$ slope SD: 0.03 Residual SD: 0.38

Table 5 –

Statistical models of longitudinal trajectories in parental linguistic performance, focusing on the ASD population and including ADOS scores. The models include 186 data points, and 32 parents of children with ASD.

	Word tokens	**Word types**	**MLU**
R^2m, R^2	**R^2m = 0.17, R^2 = 0.89**	**R^2m = 0.06, R^2 = 0.72**	**R^2m = 0.03, R^2 = 0.72**
Predictor			
Intercept	β = 2248.71, SE = 111.26, t-stat = 20.21, p < 0.0001	β = 429.39, SE = 14.95, t-stat = 28.72, p < 0.0001	β = 3.64, SE = 0.09, t-stat = 40.04, p < 0.0001
ADOS	Not Included	Not Included	Not Included
Visit	β = −33.17, SE = 115.88, t-stat = −0.29, p = 0.7769	β = 15.08, SE = 3.02, t-stat = 5.00, p < 0.0001	β = 0.08, SE = 0.03, t-stat = 2.92, p = 0.0065
$Visit^2$	β = 11.43, SE = 14.94, t-stat = 0.76, p = 0.4527	Not Included	Not Included
MSEL VR	β = 64.76, SE = 18.80, t-stat = 3.45, p = 0.0017	Not Included	Not Included
Random effects	Child intercept SD: 601.16 Visit slope SD: 439.02 Visit2 slope SD: 50.21 Residual SD: 262.11	Child intercept SD: 84.86 Visit slope SD: 56.25 $Visit^2$ slope SD: 6.37 Residual SD: 57.76	Child intercept SD: 0.57 Visit slope SD: 0.47 $Visit^2$ slope SD: 0.06 Residual SD: 0.40

Table 6 –

Statistical models of concurrent linguistic matching in parent-child dyads. The models include 395 data points, 32 children with ASD and 35 TD children.

	Parental word tokens	**Parental word types**	**Parental MLU**
R^2m, R^2	**R^2m = 0.14, R^2 = 0.79**	**R^2m = 0.16, R^2 = 0.75**	**R^2m = 0.39, R^2 = 0.72**
Predictor			
Intercept	β = 2296.43, SE = 67.05, t-stat = 34.25, p < 0.0001	β = 451.00, SE = 10.27, t-stat = 43.92, p < 0.0001	β = 4.14, SE = 0.07, t-stat = 60.49, p < 0.0001
Child Production	β = 0.32, SE = 0.08, t-stat = 4.15, p < 0.0001	β = 0.35, SE = 0.06, t-stat = 5.67, p < 0.0001	β = 0.36, SE = 0.04, t-stat = 9.49, p < 0.0001
Diagnosis	Not Included	Not Included	β = −0.40, SE = 0.10, t-stat = −3.92, p = 0.0002
Visit	Not Included	β = 9.81, SE = 2.41, t-stat = 4.06, p = 0.0001	Not Included
MSEL VR	β = 39.83, SE = 15.17, t-stat = 2.63, p = 0.0106	Not Included	β = 0.02, SE = 0.01, t-stat = 1.67, p = 0.0991
Child Production : MSEL VR	Not Included	Not Included	β = 0.03, SE = 0.01, t-stat = 3.63, p = 0.0003
Random effects	Child intercept SD: 534.31 Visit slope SD: 85.61 Residual SD: 312.18	Child intercept SD: 81.11 Visit slope SD: 9.32 Residual SD: 53.33	Child intercept SD: 0.55 Visit slope SD: 0.11 Residual SD: 0.37

Table 7 –

Statistical models predicting later child performance from parental production. The models include 335 data points, 32 children with ASD and 35 TD children.

	Child word tokens	**Child word types**	**Child MLU**
R2m, R2	**R^2m = 0.63, R^2 = 0.70**	**R^2m = 0.73, R^2 = 0.78**	**R^2m = 0.64, R^2 = 0.66**
Predictor			
Intercept	β = 598.42, SE = 22.72, t-stat = 26.33, p < 0.0001	β = 155.51, SE = 4.87, t-stat = 31.95, p < 0.0001	β = 2.24, SE = 0.04, t-stat = 51.70, p < 0.0001
Diagnosis	β = −93.28, SE = 34.19, t-stat = −2.73, p = 0.0089	β = −24.67, SE = 7.30, t-stat = −3.38, p = 0.0013	β = −0.20, SE = 0.07, t-stat = −3.06, p = 0.0029
Mullen expressive language (EL)	β = 10.70, SE = 4.55, t-stat = 2.35, p = 0.023	β = 1.72, SE = 0.97, t-stat = 1.77, p = 0.0814	β = 0.03, SE = 0.01, t-stat = 3.09, p = 0.0026
Diagnosis : EL	β = 12.80, SE = 5.50, t-stat = 2.33, p = 0.0247	β = 3.42, SE = 1.18, t-stat = 2.89, p = 0.0056	β = 0.02, SE = 0.01, t-stat = 2.05, p = 0.0431
Child performance in the previous visit (same index as the outcome)	β = 0.41, SE = 0.05, t-stat = 8.91, p < 0.0001	β = 3.51, SE = 1.18, t-stat = 2.97, p = 0.0045	β = 0.49, SE = 0.05, t-stat = 9.92, p < 0.0001
Parental word tokens in the previous visit	Not Included	Not Included	Not Included
Parental word types in the previous visit	Not Included	Not Included	Not Included
Parental MLU in the previous visit	β = 88.37, SE = 21.59, t-stat = 4.09, p = 0.0001	β = 17.60, SE = 4.74, t-stat = 3.72, p = 0.0002	β = 0.09, SE = 0.04, t-stat = 1.85, p = 0.0809
Random Effects	Child intercept SD: 95.16 Residual SD: 204.95	Child intercept SD: 20.65 Residual SD: 43.34	Child intercept SD: 0.21 Residual SD: 0.37

Table 8 –

Standardized statistical models predicting later child performance from parental production. The full models include 335 data points, 32 children with ASD and 35 TD children.

Predictor	Child word tokens	Child word types	Child MLU
Intercept	β = 0.13, SE = 0.06	β = 0.14, SE = 0.05	β = 0.12, SE = 0.05
	ASD: β = −0.14, SE = 0.07 TD: β = 0.12, SE = 0.06	ASD: β = −0.13, SE = 0.05 TD: β = 0.14, SE = 0.05	ASD: β = −0.15, SE = 0.06 TD: β = 0.11, SE = 0.05
Child language feature at previous visit	β = 0.40, SE = 0.04	β = 0.50, SE = 0.04	β = 0.48, SE = 0.05
	ASD: β = 0.21, SE = 0.07 TD: β = 0.48, SE = 0.06	ASD: β = 0.41, SE = 0.06 TD: β = 0.53, SE = 0.05	ASD: β = 0.28, SE = 0.08 TD: β = 0.56, SE = 0.06
Mullen expressive language (EL)	β = 0.17, SE = 0.07	β = 0.11, SE = 0.06	β = 0.19, SE = 0.06
	ASD: β = 0.48, SE = 0.07 TD: β = 0.15, SE = 0.07	ASD: β = 0.39, SE = 0.06 TD: β = 0.1, SE = 0.07	ASD: β = 0.49, SE = 0.07 TD: β = 0.16, SE = 0.06
Diagnosis	β = −0.24, SE = 0.09	β = −0.26, SE = 0.08	β = −0.23, SE = 0.08
Diagnosis : EL	β = 0.21, SE = 0.09	β = 0.23, SE = 0.08	β = 0.16, SE = 0.08
Parental MLU	β = 0.16, SE = 0.04	β = 0.12, SE = 0.04	β = 0.07, SE = 0.04
	ASD: β = 0.2, SE = 0.05 TD: β = 0.11, SE = 0.07	ASD: β = 0.15, SE = 0.04 TD: β = 0.12, SE = 0.06	ASD: β = 0.08, SE = 0.07 TD: β = 0.07, SE = 0.05

N.B. All estimates in the table are standardized to be more easily comparable with each other. Standardization involved centering (subtracting the mean) and scaling (dividing by the standard deviation).

Table 9 –

Statistical models predicting parental performance from earlier child production. The models include 335 data points, 32 parents of children with ASD and 35 parents of TD children.

	Parental word tokens	**Parental word types**	**Parental MLU**
R2m, R2	**R^2m = 0.61, R^2 = 0.64**	**R^2m = 0.2, R^2 = 0.62**	**R^2m = 0.49, R^2 = 0.53**
Predictor			
Intercept	β = 2334.06, SE = 25.31, t-stat = 92.22, p < 0.0001	β = 462.65, SE = 7.91, t-stat = 58.45, p < 0.0001	β = 4.19, SE = 0.04, t-stat = 100.08, p < 0.0001
Diagnosis	Not Included	Not Included	β = −0.22, SE = 0.07, t-stat = −3.41, p = 0.003
Parental performance in the previous visit (same index as the outcome)	β = 0.74, SE = 0.04, t-stat = 20.92, p < 0.0001	β = 0.34, SE = 0.05, t-stat = 6.91, p < 0.0001	β = 0.47, SE = 0.04, t-stat = 10.52, p < 0.0001
Child word tokens	Not Included	Not Included	β = 0.00001, SE = 0.0001, t-stat = 0.01, p = 0.99
Child word types	Not Included	Not Included	Not Included
Child MLU	Not Included	β = 16.6, SE = 5.45, t-stat = 3.04, p = 0.0025	Not Included
Child word tokens: Diagnosis	Not Included	Not Included	β = 0.001, SE = 0.0002, t-stat = 3.94, p = 0.0002
Random effects	Child Intercept SD: 111.78 Residual SD: 389.39	Child Intercept SD: 59.81 Residual SD: 57.17	Child Intercept SD: 0.13 Residual SD: 0.46